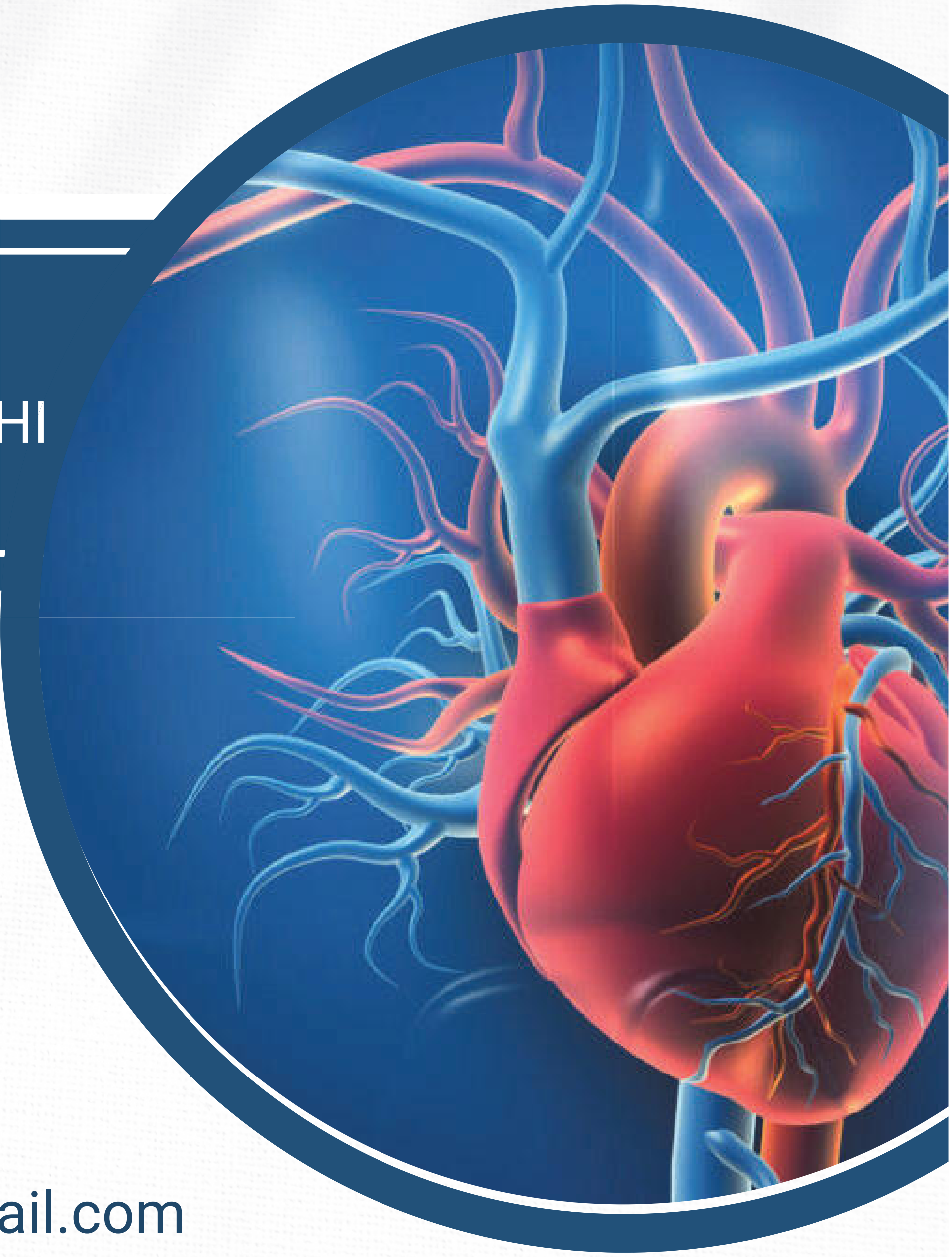
Alrajhi

PASSMEDICINE

CARDIOLOGY

Internal medicine

EDITED BY
DR. JAMIL ALRAJHI



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773-555-493
jameelalrajhi@gmail.com

PASSMEDICINE CARDIOLOGY PART 2

Question 1 of 328

A 31-year-old woman comes to the emergency department complaining of palpitations and dizziness. She has had palpitations before, but this is the first time she is feeling dizzy.

On examination, she is afebrile. Her heart rate is 140 bpm, and her blood pressure is 110/90 mmHg. She has a respiratory rate of 12 breaths per minute, and oxygen saturation is 96% on air. The rest of her examination is normal.

A 12-lead ECG shows polymorphic ventricular tachycardia, QTc 510ms. Her laboratory studies show:

Which of the following is the best next step in the management of this patient?

A. Intravenous amiodarone
B. Intravenous lidocaine
C. Intravenous magnesium sulphate
D. Immediate defibrillation
E. Synchronised DC cardioversion

C. Intravenous magnesium sulphate

IV magnesium sulfate is used to treat torsades de pointes Important

This patient is presenting with an acute episode of torsades de pointes. Her ECG shows polymorphic ventricular tachycardia with a prolonged QT interval, which is consistent with torsades de pointes. It is characterised by rapid, irregular QRS complexes, which appear to be twisting around the ECG baseline. Torsades de pointes is treated with IV magnesium sulphate, even when the serum magnesium levels are normal.

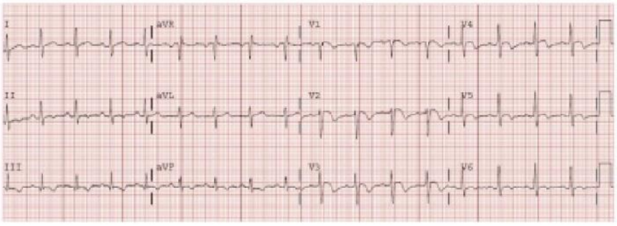
Immediate defibrillation is indicated in patients with pulseless torsades de pointes. This patient is conscious and has a pulse.

Synchronised DC cardioversion is indicated in patients with haemodynamically unstable torsades de pointes. This patient is haemodynamically stable.

IV amiodarone and IV lidocaine can be used to treat monomorphic ventricular tachycardia. Torsades de pointes is a polymorphic ventricular tachycardia.

Question 2 of 328

A 35-year-old man presents to his GP with a five day history of dyspnoea, cough, chest pain and palpitations. He describes the chest pain as being worse when he takes a deep breath in and the only relieving factor is ibuprofen. There has been no sputum production or haemoptysis. He has no significant past medical history of note. On examination his pulse is 102/min, blood pressure is 124/78 mmHg and oxygen saturations are 95% on room air. Auscultation of his chest is normal. An ECG is taken:



What is the most likely diagnosis?

A. Pulmonary embolism
B. Acute coronary syndrome
C. Pericarditis
D. Musculoskeletal chest pain
E. Viral pleurisy

A. Pulmonary embolism

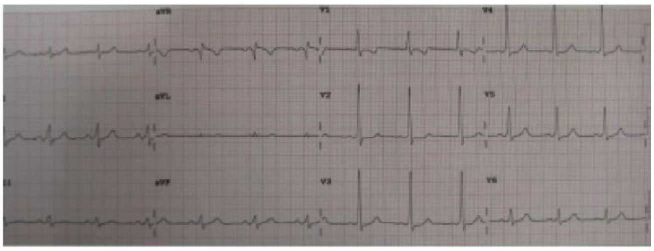
S1Q3T3 is a classic but uncommon ECG finding in PE

The unifying diagnosis here is pulmonary embolism. He has dyspnoea and pleuritic chest pain. The tachycardia and low oxygen saturations are also suggestive. Low oxygen saturations in someone who is otherwise fit and well are unusual and need explaining. Acute coronary syndrome, pericarditis, viral pleurisy and musculoskeletal chest pain would not normally drop your saturations unless complications develop.

The ECG however is the clincher - he has the classic S1Q3T3 changes.

Question 3 of 328

A 26-year-old female presents with a third episode of palpitations associated with shortness of breath and chest discomfort. She has no other past medical history, thyroid function tests unremarkable. She denies taking any recreational drugs and has no significant family history. Her ECG is as follows:



Which of the following drugs would be safe to administer immediately if she becomes tachycardic?

A. Adenosine
B. Digoxin
C. Diltiazem
D. Verapamil
E. Procainamide

E. Procainamide

The ECG demonstrates Wolff-Parkinson-White (WPW) syndrome, with a delta wave upstroke as a result of pre-excitation prior to the QRS complex. WPW is an atrioventricular re-entry tachycardia, activity, and subsequently stimulates a ventricular response.VVI pacemaker is incorrect.

A VVI pacemaker is a single lead pacemaker that senses and paces the ventricles only. The 'I' stands for 'inhibition' and indicates that if the pacemaker senses ventricular activity it inhibits its pacing output. VVI pacemakers, also known as ventricular demand pacemakers, are commonly used for ventricular bradycardia such as atrial fibrillation with a slow ventricular response.

AAI pacemaker is incorrect. An AAI pacemaker is a single lead pacemaker that senses and paces the atria only. The 'I' stands for 'inhibition' and indicates that if the pacemaker senses atrial activity it inhibits its pacing output. It is used only for patients with sinus node dysfunction.

Transcutaneous pacing would be used for patients who were unstable as a stop-gap measure whilst they awaited pacemaker insertion. This would not be appropriate in this case as we have a stable and well patient.

Ablation is used to prevent aberrant electrical signalling from causing arrhythmias. In the case of Mobitz type II, the issue is non-conduction rather than aberrant conduction, ablation is therefore not appropriate.

Question 7 of 328

A 28-year-old woman attended the emergency department and was subsequently referred to the cardiology team due to concerns about the risk of potential cardiac arrhythmia. The patient presented to the emergency department due to ongoing nausea and vomiting secondary to hyperemesis gravidarum. The patient stated she was 10 weeks pregnant with her first child and had been suffering from these symptoms for the past 6 weeks. Clinical records indicated the patient had also attended the emergency department 3 weeks previously with the same presenting complaint.

On the initial occasion the patient had presented to the emergency department with hyperemesis gravidarum, she had been rehydrated with intravenous fluids and prescribed vitamin B6 and ginger capsules four times daily on the advice of the obstetric team. The patient reported that this treatment had given her only a modest improvement in her symptoms and she had stopped taking these medications after a few days. Subsequently, she had attended an out-of-hours medical centre and been prescribed ondansetron (4 mg three times daily). The patient had found a more significant benefit from ondansetron and had been taking it regularly over the past 2 weeks. In addition to her symptoms of nausea and vomiting, the patient reported feeling tired, lethargic and dehydrated.

Haemoglobin	123 g / dL
Mean cell volume	84.9 fl
White cell count	11.7 x 10>3 / microlitre
Platelets	187 x 10>3 / microlitre
Urea	9.0 mmol / L
Creatinine	82 micromol / L
Sodium	139 mmol / L
Potassium	3.6 mmol / L

Presentation	Relative date ofECG	Corrected QT interval / ms
2nd ED attendance for hyperemesis	Today	490
1st ED attendance for hyperemesis	3 weekspreviously	430
Unrelated ED attendance	4 yearspreviously	415

However, she denied experiencing any other symptoms, including palpitations or pre-syncopal episodes.

The patient's past medical history included a period of intravenous drug use in her early twenties. Three years previously the patient had successfully managed to stop her use of illicit drugs after being enrolled in a methadone treatment program. The patient was now on a consistent methadone dose of 80 mg once daily. There was no other significant past medical history and the patient took no other regular medications.

During the patient's assessment after attending the emergency department, concern was raised over a prolonged corrected QT interval on an ECG performed due to borderline hypokalaemia. The patient's ECG was otherwise unremarkable and was compared to previous ECGs the patient had undergone on her previous visits to the emergency department. Please see below for blood results and data from the patient's current and previous ECG.

With regard to the patient's prolonged corrected QT interval, what is the most appropriate strategy for managing her medications?

A. Continue both methadone and ondansetron and admit for cardiac monitoring
B. Stop both methadone and ondansetron
C. Continue both methadone and ondansetron and arrange outpatient ambulatory ECG
D. Continue methadone, stop ondansetron
E. Continue ondansetron, stop methadone

D. Continue methadone, stop ondansetron

The upper limit of normal for corrected QT interval is considered to be 450 ms in men and 460 ms in women. QT prolongation is associated withtorsades de pointes (TdP) and sudden cardiac death. However, the presence of a prolonged QT interval does not inevitably lead to TdP and TdP can occur in the presence of a normal QT interval. Each 10 ms increase in QT interval increases the risk of TdP by around 5-7 % and once corrected QT interval exceeds 500 ms, the risk of TdP is markedly increased and intervention is necessary.

The patient is taking 2 medications that are known to prolong the QT interval and these can be seen to have had an additive effect in prolonging the patient's QT interval. Specifically, the corrected QT interval increased from 430 ms when the patient was taking methadone to 490 ms when the patient was taking methadone and ondansetron. The normal baseline ECG suggests that the patient does not have a congenital condition causing QT prolongation. At the time of presentation, the patient's borderline hypokalaemia is a factor increasing the risk of TdP in the setting of her drug-induced prolonged QT interval.

Few recommendations exist for managing the risk of drug-induced prolonged QT interval. Expert advice suggests that when a QT prolonging drug is associated with a corrected QT interval of 480-500 ms in a woman (470-500 ms in a man), or when an increase of 60 ms or greater occurs, dose reduction or discontinuation is advised. If the corrected QT interval exceeds 500 ms then the drug should be discontinued and expert advice sought.

The use of methadone alone does not appear to cause a prolonged QT interval in this patient, whereas her QT interval increased by 60 ms following the addition of ondansetron. Given the importance to the patient's long-term health of adequate opiate replacement therapy, the risks associated with opiate withdrawal to a foetus in early pregnancy and the availability of other anti-emetic medications the most sensible course of action out of the available options is to discontinue ondansetron and continue methadone treatment.

Advice from the cardiology team could be sought to determine if the patient should be admitted for cardiac monitoring. The only available answer to include admission as an option also requires the patient to continue with both medications that prolong the QT interval and so is incorrect. Continuing both medications and discharging the patient for an outpatient ambulatory ECG (presumably to assess for TdP) is not an advisable management plan.

Question 8 of 328

A 65-year-old man is admitted with central chest pain that has been ongoing for two hours. It radiates to the left arm and is associated with nausea and vomiting. He has a past medical history of hypertension and diabetes which is diet controlled. He takes amlodipine and atorvastatin.

On examination his blood pressure is 147/89 mmHg and his heart rate is 110 beats per minute. His saturations are 96% on room air and he is afebrile His cardiovascular and respiratory examinations are unremarkable.

An ECG shows T wave inversion in leads I, V4, V5 and V6. Blood tests are as follows:

Hb	131 g/l	Na	136 mmol/l
Platelets	430 * 10 /l	K	4 mmol/l
WBC	8 * 10 /l	Urea	4 mmol/l
Neuts	6 * 10 /l	Creatinine	84 µmol/l
Lymphs	1 * 10 /l	CRP	41 mg/l
Eosin	0.3 * 10 /l	Trop	130 ng/l

He is diagnosed with non-ST elevation myocardial infarction and commenced on aspirin, clopidogrel and low molecular weight heparin. His pain settles with morphine and glyceryl trinitrate spray within an hour. The following day he is commenced on ramipril and bisoprolol. 3 days later he undergoes an uncomplicated angiogram with stunting of the left circumflex artery.

On systems reviews prior to discharge he comments that he has noticed a rash on his legs and has not been passing much urine despite drinking plenty of water. On examination he has a bluish lacey discolouration over his legs. He has no palpable bladder.

Repeat bloods are as follows:

Hb	12.1 g/l	Na +	137 mmol/l
Platelets	560 * 10 /l	K	5 mmol/l
WBC	10 * 10 /l	Urea	12 mmol/l
Neuts	7 * 10 /l	Creatinine	184 µmol/l
Lymphs	1.2 * 10 /l	CRP	60 mg/l
Eosin	1.5 * 10 /l		

What is the most likely cause of his symptoms and deranged blood tests?

A. Cholesterol embolism
B. Contrast nephropathy
C. Endocarditis
D. Hypovolaemia during NSTEMI
E. Newly started ACE inhibitor

A. Cholesterol embolism

This gentleman as developed an acute kidney injury following angiography. The combination of a lacey rash (livedo reticularis) and raised eosinophils points to a likely diagnosis of cholesterol embolism. Contrast induced nephropathy is also possible following angiography but is not associated with rash or eosinophilia. Although renal function may deteriorate after starting an ACE inhibitor, it is unlikely to cause this degree of derangement so quickly. As the angiogram was uncomplicated, hypovolaemia is unlikely. Endocarditis would be unusual in the absence of fevers and other systemic signs.

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A 52-year-old man is asymptomatic but concerned about his risk of having a heart attack. His 58-year-old brother died of a myocardial infarction 2 months ago. He has a past medical history of hypertension, for which he takes perindopril and amlodipine. His sister takes tablets for diabetes and high blood pressure. He successfully quit smoking over 1 year ago and drinks approximately 2 units of alcohol per day.

On examination, his blood pressure is 130/70 mmHg and BMI is 32 kg/m2.
Investigations:

haemoglobin	16g/dL (13-18)
mean corpuscular volume	90 fL (80-96)
serum cholesterol	5.0 mmol/L (<5.2)
serum LDL cholesterol	3.1 mmol/L (<3.36)
serum HDL cholesterol	1.55 mmol/L (>1.55)
fasting serum triglycerides	1.6 mmol/L (0.451.69)
haemoglobin A1c	5.4% (4.06.0)
serum aspartate aminotransferase	19 U/L (131)
plasma thyroid-stimulating hormone	2.4 mU/L (0.45.0)
estimated glomerular filtration rate (MDRD)	85 mL/min (>60)

His QRISK2 score estimates a 13% 10 year risk of myocardial infarction or stroke.

In addition to discussing lifestyle modifications, what treatment should you offer this patient?

A. Aspirin
B. Atorvastatin
C. Indapamide
D. Metformin
E. Omega-3-polyunsaturated fatty acid supplements

B. Atorvastatin

The 2014 NICE guidelines: Cardiovascular disease: risk assessment and reduction, including lipid modification have lowered the UK threshold for offering a statin for primary prevention to a 10% or greater 10year risk of developing cardiovascular disease, according to the QRISK2 score. The previous recommendation advised offering a statin to patients with a 20% 10-year risk. A high-intensity low cost statin should be 1st line in this context, such as atorvastatin 20mg.

Prescribing aspirin for primary prevention is controversial, whether the benefits in primary prevention outweigh the bleeding risks is unclear, therefore most guidelines do not recommend aspirin in primary prevention. Unlike in secondary prevention where the benefits usually outweigh the risks.

The NICE guidelines advise NOT prescribing omega-3-fatty acid supplements for primary or secondary prevention of cardiovascular disease, as they have not shown to be efficacious in reducing cardiovascular risk in meta-analyses. However, guidance continues to encourage consuming regular portions of oily fish, the 2014 nice guidance specifically advises: 2 portions of fish per week, including a portion of oily fish.

Metformin would be inappropriate in this patient as he is not diabetic.

This patients hypertension is well controlled, therefore indapamide would be inappropriate

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A 60-year-old man presents to the emergency department with crushing central chest pain. The pain came on suddenly approximately one hour ago. A 12-lead ECG reveals >2mm of ST elevation in all anterior and lateral leads. The nearest centre offering primary angioplasty is 140 minutes away. There is no contraindication to thrombolysis.

In addition to standard acute coronary syndrome treatment (analgesia, aspirin, a heparin and a P2Y12 inhibitor), what is the best management for this patient?

- A. Immediate transfer for acute angioplasty
- B. Non-urgent angioplasty
- C. Thrombolysis
- D. Thrombolysis and immediate transfer for acute angioplasty
- E. Thrombolysis and non-urgent angioplasty

D. Thrombolysis and immediate transfer for acute angioplasty

The recommended door-to-balloon aka door-to-device time for primary percutaneous coronary intervention (PCI) is within 90 minutes. However, many centres do not have provision for primary PCI and are unable to transfer patients to a centre that does in a timely manner.

The NORDISTEMI TRIAL (NORwegian study on DIstrict treatment of STElevation Myocardial Infarction) demonstrated that in centres with a long transfer time (mean transfer time was 130 minutes in this trial) for PCI, patients with STEMI should initially receive thrombolysis, with subsequent immediate transfer for PCI. For patients with STEMI this approach reduced the rate of death, re-infarction, or stroke at 12 months compared to patients treated with a conservative, ischaemia-guided approach (e.g thrombolysis alone or thrombolysis with rescue angioplasty).

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A 73-year-old gentleman sees his GP with progressive shortness of breath. He can now walk 10 yards before stopping to catch his breath and he is unable to lie flat. He sleeps upright in his armchair and has stopped sleeping in his bed over the last three months.

He has known hypertension and has had two previous myocardial infarctions. On examination, his apex beat is displaced to the 7th intercostal space in the left mid-axillary line and there are crepitations in the two lung bases. His JVP is raised at 5cm and there is bilateral leg oedema.

Troponin	<20 ng/ml
B-natriuretic peptide	14,000 ng/L (normal range 0-200)
Chest X-ray	cardiomegaly and increased alveolar shadowing in the perihilar areas
ECG	QRS 110ms, large volume complexes in chest leads

Which intervention is proven to reduce mortality in this case?

- A. Cardiac Resynchronisation Therapy (CRT)
- B. Digoxin
- C. Furosemide
- D. Ramipril
- E. Coronary artery bypass graft

D. Ramipril

ACE inhibitors offer prognostic benefit in chronic heart failure

Of the above options, ramipril is proven to reduce mortality in congestive cardiac failure through adjusting remodelling of the heart.Digoxin and furosemide can help symptoms but no trial shows a benefit in mortality. CRT can be helpful in cases of ventricular dyssynchrony but the QRS is narrow and this gentleman would not benefit from it. Coronary artery bypass grafting may be useful if this gentleman had symptoms of angina but he does not

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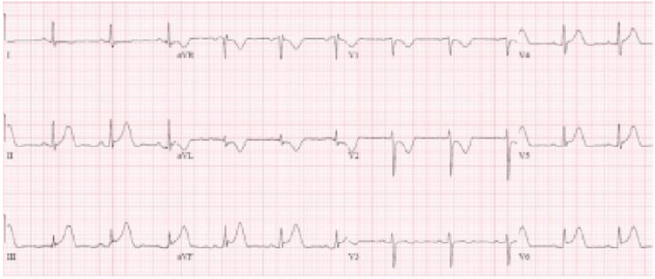
A 75-year-old male presents to the emergency department with central crushing chest pain which has been ongoing for 4 hours. It is associated with shortness of breath.

Blood results are as follows:

Hb	125 g/L	Male: (135-180)Female: (115 - 160)
Platelets	424 * 10 /L	(150 - 400)
WBC	14.6 * 10 /L	(4.0 - 11.0)
Na	135 mmol/L	(135 - 145)
K	4.4 mmol/L	(3.5 - 5.0)
Urea	7.8 mmol/L	(2.0 - 7.0)
Creatinine	134 µmol/L	(55 - 120)
Troponin	8422 ng/L	(< 15)

An ECG is performed:



What is the most likely diagnosis?

- A. Anterolateral STEMI
- B. Inferolateral STEMI
- C. Myocarditis

D. Pericarditis
E. Posterior STEMI

B. Inferolateral STEMI

The ECG demonstrates ST elevation in the inferior (II, III, and aVF) and lateral leads (V5-6) confirming a diagnosis of an inferolateral STEMI. ST depression in V1-2 is suggestive of an associated posterior infarction. This constellation of ECG abnormalities is typically produced by occlusion of the proximal circumflex artery.

Anterolateral STEMI would present with ST elevation in the anterior (V1-V4) and lateral leads.

Posterior STEMI presents with reciprocal changes in the anterior leads including horizontal ST depression and a dominant R wave in V2.

Both myocarditis and pericarditis present with global concave ST elevation. The troponin is usually normal in pericarditis and raised in myocarditis.

Question 13 of 328

A 66-year-old lady attended a cardiology clinic for follow up after a recent hospital admission with pulmonary oedema. She continued to experience significant shortness of breath at rest with exercise tolerance that was limited to a few steps around the house. She had a previous medical history of hypertension, diabetes and myocardial infarction 5 years previously treated with percutaneous coronary intervention. Her medications comprised of bisoprolol 10mg, ramipril 10mg, furosemide 80mg twice daily, spironolactone 25mg, simvastatin 40mg, metformin 1g twice daily and aspirin 75mg. On examination she appeared breathless, the jugular venous pressure was elevated at 7cm, there were bibasal fine crepitations and moderate pitting oedema to her knees. Her heart rate was 68 beats per minute and blood pressure was 95/65mmHg.

Investigations:

Haemoglobin	115 g/L
White cell count	5.6 x10^9/L
Platelet Count	268 x10^9/L
Serum sodium	132mmol/L
Serum potassium	4.3mmol/L
Serum urea	6.7mmol/L
Serum creatinine	68micromol/

Electrocardiogram: normal sinus rhythm. Rate 65 beats per minute. QRS duration 155ms. No acute ST changes.

Echocardiography: left ventricular ejection fraction of 30%. No significant valvular abnormalities.

Coronary angiography: Patent left anterior descending coronary artery stent. Minor diffuse coronary artery disease.

What is the most appropriate next management step?

A. Increase dose of furosemide
B. Cardiac resynchronisation therapy
C. Coronary artery bypass graft
D. Start amlodipine
E. Home oxygen

B. Cardiac resynchronisation therapy

This lady has ischaemic heart disease with New York Heart Association (NYHA) class 4 heart failure and is on maximal medical therapy. Her ECG shows a wide QRS complex which suggests ventricular dyssynchrony (i.e. The left and right ventricle are not beating at the same time). Resynchronisation of the ventricles with cardiac resynchronisation therapy with a paced device (CRT-P) involves simultaneous stimulation of both ventricles to reduce dyssynchrony and therefore increase cardiac output.

Nice supports the use of CRT-P in patients with:

- NYHA class three or four symptoms
- They are in normal sinus rhythm with either → QRS duration of 150 ms or longer estimated by standard electrocardiogram (ECG)
- → QRS duration of 120-149 ms estimated by ECG and mechanical dyssynchrony that is confirmed by echocardiography.
- They have a left ventricular ejection fraction of 35% or less.
- They are receiving optimal pharmacological therapy.

In this selected group of patients, it has been shown to improve symptoms by a reduction of about one NYHA classification, reduce hospitalisations and reduce mortality. These devices can be fitted with an implantable cardiac defibrillator (i.e. A CRT-D where the D stands for defibrillator as opposed to a CRT-P where P stands for paced and thus this device lacks the defibrillator component) for primary or secondary prophylaxis against ventricular dysrhythmias.

Increasing her dose of furosemide is unlikely to significantly improve her symptoms and is, therefore, an incorrect answer. Coronary artery bypass grafting has greater benefit than percutaneous coronary intervention at treating multivessel disease, however, this lady has disease of the left anterior descending artery which has been previously treated and only minor disease in her other coronary vessels; therefore this is an incorrectanswer. She is hypotensive and therefore starting amlodipine is inappropriate. Home oxygen can be used for the palliation of shortness of breath in heart failure and is not appropriate in this case.

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A 55-year-old with previous rheumatic heart disease aged 32 presents with an 18-month history of exertional dyspnoea. An initial echo demonstrated significant raised pulmonary arterial pressures of 77 mmHg, she undergoes a left and right heart catheter with results as follows:-

Oxygen saturations		
Right atrium	8 mmHg	71%
Right ventricle	39/8 mmHg	71%
Pulmonary artery	45/12 mmHg	71%
Capillary wedge	20 mmHg	93%
Left ventricle	165/11 mmHg	93%
Aorta	90/58 mmHg	

What is the most likely diagnosis?

A. Aortic stenosis
B. Mitral stenosis
C. Aortic stenosis and mitral stenosis

D. Aortic stenosis and pulmonary hypertension
E. Aortic stenosis, mitral stenosis, pulmonary hypertension

E. Aortic stenosis, mitral stenosis, pulmonary hypertension

The key to questions regarding saturations and cardiac catheters is to spot the 'step-up' in oxygen saturation and abnormalities in gradients across valves. There are no 'step-ups' in oxygen saturations, demonstrating no shunts. However, you will note that the pulmonary arterial pressure is greater than the normal one-fifth of systolic measurements; hence pulmonary hypertension is present. In addition, there is a greater than 25mmHg gradient across the aorta valve, demonstrating moderate aortic stenosis. Lastly, the capillary wedge pressure is equivalent to the left atrial pressure, which should also be the same as the left ventricular diastolic pressure. A normal mitral valve expects less than 5mmHg pressure difference. Using these inferences, the mitral valve gradient is calculated by the capillary wedge pressure of 20mmHg (same as the left atrial pressure) minus the diastolic left ventricular pressure of 11mmHg: the 9mmHg difference thus also demonstrates mitral stenosis.

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A 72-year-old female presents with left chest pain associated with sweating in her hands and nausea. Her past medical history includes T2 diabetes mellitus, hypertension and hypercholesterolaemia, with her regular medications including metformin 850mg BD, gliclazide 40mg OD, ramipril 10mg, amlodipine 5mg and atorvastatin 40mg ON. On examination, her heart sounds are normal with a pansystolic murmur. Her ECG demonstrates ST depression in V2 to V5. Her BMs have ranged between 6 and 14 mmol/L since her admission 4 hours ago.

How should you manage her diabetic medications?

A. No changes to medication
B. Increase metformin to 850mg TDS
C. Increase gliclazide to 40mg BD
D. Actrapid PRN 4 units when BM >11mmol/L
E. Stop all medications, start insulin sliding scale

E. Stop all medications, start insulin sliding scale

Patients with acute coronary syndrome demonstrate significantly lower mortality rates at one year when treated with tight glycaemic control than without (19% vs 26%). Metformin has the potential to induce lactic acidosis in the context of any kind of ischaemia. An insulin based regime is recommended by the European Society of Cardiology , on the basis of results from DIGAMI trial . PRN actrapid should never be prescribed routinely.

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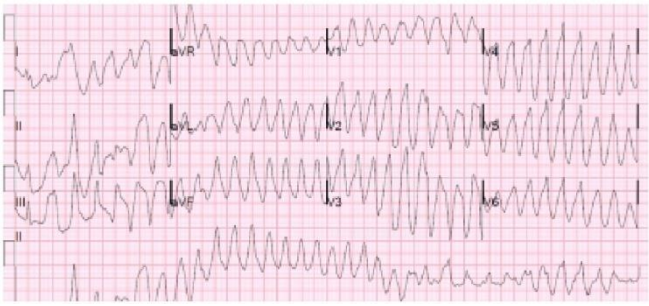
A 25-year-old man presents with a one-week history of feeling generally unwell, reporting myalgia, nausea and weakness.

His observations are:
Respiratory rate: 24/min

- Blood oxygen saturation: 97% on room air
- Heart rate: 109bpm
- Blood pressure: 91/56mmHg
- Temperature: 37.7 C
- GCS 15/15

The patient is given intravenous anti-emetics and a bolus of crystalloid fluid. Whilst the fluid is running, the patient begins to complain of palpitations, chest pain and shortness of breath. An ECG is performed and is shown below.



What electrolyte abnormality is most likely to have precipitated this rhythm?

A. Hypercalcaemia
B. Hyperkalaemia
C. Hypermagnesaemia
D. Hypokalaemia
E. Hyponatraemia

D. Hypokalaemia

The ECG shows polymorphic ventricular tachycardia. This is shown by the broad complexes, and the QRS complexes varying in amplitude, axis and duration. The variation in QRS complexes is due to having multiple ventricular foci.

Hypokalaemia is the correct answer. Symptoms of myalgia and weakness are in keeping with hypokalaemia. Hypokalaemia can cause prolonged QT which can precipitate polymorphic ventricular tachycardia, as seen in the ECG. Hypokalaemia is the only electrolyte abnormality listed above that can precipitate prolonged QT.

Hypercalcaemia is the incorrect answer. Hypocalcaemia can precipitate prolonged QT.

Hyperkalaemia is the incorrect answer. As above, hypokalaemia can precipitate prolonged QT.

Hypermagnesaemia is the incorrect answer. Hypomagnesaemia can precipitate prolonged QT.

Hyponatraemia is the incorrect answer. Sodium derangement is not associated with prolonged QT and polymorphic ventricular tachycardia.

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A 25-year-old woman has recently been diagnosed with Marfan's syndrome. She has no other known diagnosis. She undergoes an echocardiogram which demonstrates severe aortic regurgitation, significant aortic root dilatation and a left ventricular ejection fraction of 70%. She is asymptomatic and has no family history of aortic dissection.

What is the most appropriate management of her cardiac problem?

A. Follow-up in six months

B. ACE inhibitor
C. Exercise testing
D. Offer surgery if planning to become pregnant
E. Referral for aortic valve replacement

E. Referral for aortic valve replacement

The correct answer is referral for aortic valve replacement. Surgery for aortic regurgitation is indicated if there is significant enlargement of the ascending aorta, severe regurgitation with symptoms or if severe with an ejection fraction of less than 50%. ACE inhibitors can be useful when there is hypertension, when surgery is contraindicated or when ventricular dysfunction persist following surgery. Exercise testing is more useful in aortic stenosis and follow-up would be acceptable if the aortic root was not dilated.

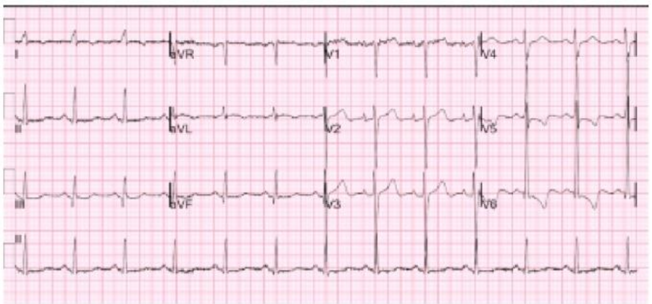
Question 18 of 328

A 52-year-old is brought from the renal outpatient clinic to the emergency department (ED) after appearing very short of breathing having walked up the stair. His breathlessness was associated with some mild, generalised chest discomfort, but the patient puts this down to a recent chest infection he has just recovered from.

When assessed in the ED the patient's breathlessness has improved and he denies any further chest discomfort. He reports he has been experiencing more frequent similar episodes, gradually increasing in severity over several months. He reports he recently had a productive cough with coryzal symptoms for the last 3-4 days but he feels it is improving. On questioning he confirms his symptoms of breathlessness are often worse during and post these chest infections.

He has a past medical history of chronic renal artery stenosis and resultant poorly controlled hypertension for which he is on multiple medications including an ACE inhibitor, beta-blocker and a diuretic.

An ECG is performed as seen below.



What is the patient's most likely diagnosis?

A. Brugada syndrome
B. Hyperkalaemia
C. Left ventricular hypertrophy
D. Pericarditis
E. Wellen's syndrome

C. Left ventricular hypertrophy

This patient has presented with symptoms and typical ECG changes in keeping with left ventricular (LV) hypertrophy. The thickening of the cardiac muscle is most likely a result of chronic uncontrolled hypertension, hence the prolonged, progressive history. LV hypertrophy results in signs and symptoms of LV failure including breathlessness, angina and reduced exercise tolerance. The patient's ECG demonstrates left ventricular hypertrophy with ST elevation in V2-3, without reciprocal lead changes and the combination of the S wave in V1 with the R wave in V5 or V6 clearly exceeding 40 mm.

Brugada syndrome is a genetic cardiac disorder resulting in electrical activity disorders and not structural issues. Features include syncope and sudden cardiac death. There are several types of Brugada syndrome with variation seen on ECG however the only potentially diagnostic ECG abnormality is of coved ST-segment elevation in 2 or more of V1-3 followed by a negative T wave. This is classical of Brugada type 1 syndrome and is commonly known as Brugada sign.

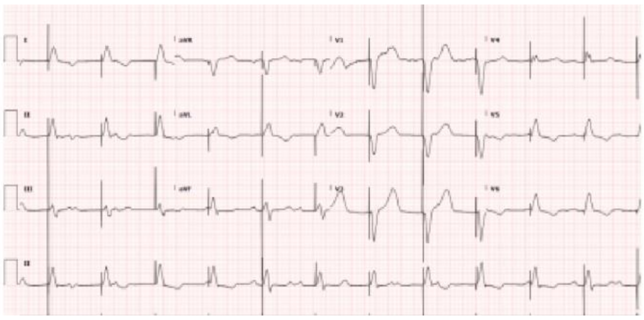
There is no clear reason why this patient would be hyperkalaemia. Although he has renal impairment it is not indicated that he has endstage renal failure or that he is on renal replacement therapy therefore hyperkalaemia would not be expected. Common ECG changes noted in hyperkalaemia included peaked T waves, P wave widening/flattening, PR prolongation progressing to the bradyarrhythmias and conductions blocks. No clear features of hyperkalaemia can be seen on this patient's ECG.

Pericarditis is inflammation of the tissue surrounding the heart and can be due to infections. Patients normally present with pleuritic, constant chest pain and fevers. Although this patient has a very recent chest infection, pericarditis normally develops a few weeks post the resolution of infection as it is an immune response and not a direct result of the infection itself. ECG findings associated with pericarditis include widespread concave ST elevation and PR depression which are not present on this patient's ECG.

Wellen's syndrome is the ECG pattern of biphasic or deeply inverted T waves in the chest leads V2-3. It is highly specific for critical stenosis ofthe left anterior descending (LAD) artery and therefore is normally seen in patients presenting with ischaemic like symptoms. You would expect the patient's chest pain to be continuous and to be more severe if he was experiencing stenosis of the LAD.

Question 19 of 328

A 62-year-old man is recovering from an aortic valve replacement via median sternotomy in the high-dependency unit. You are called to see him overnight as he is experiencing chest pain, worse on breathing. An ECG is performed and displayed below. He had temporary trans-venous pacing wires inserted during the procedure.



What does the ECG show?

A. AAI pacing
B. Complete heart block with loss of capture
C. DDD pacing
D. Myocardial infarction
E. VVI pacing

E. VVI pacing

Although atrial pacing wires are commonly inserted following cardiac surgery, it is particularly important to insert ventricular wires following aortic valve surgery due to the significantly higher incidence of complete heart block postoperatively. The ECG shows pacing spikes immediately preceding QRS complexes, indicating ventricular (VVI) pacing with good capture. This patient has a degree of heart block underlying his paced rhythm, hence the discordance between the p waves and the QRS complexes, and the reason why the patient is paced.AAI pacing would show pacing spikes prior to p waves, and in instances of heart block as shown here there would be abnormal or potentially absent conduction to the ventricles. As the pacing spikes are associated with the QRS complex here, AAI cannot be the mode in use.

Although this patient may have complete heart block, there is good capture as shown by a 1:1 ratio between pacing spikes and subsequent QRS complexes. Therefore loss of capture is an incorrect answer.

DDD pacing refers to the pacing of both the atria and ventricles. Although this can be useful post-operatively to increase cardiac output, it is not being used here. DDD pacing would either show pacing of the atria (pacing spikes before p waves) or tracking of the patient's intrinsic atrial rate (ventricular pacing spikes that follow p waves and immediately precede QRS complexes).

Pain is very common following a sternotomy and is classically sharp and pleuritic. It can be difficult to interpret ischaemia on a paced ECG however the Sgarbossa criteria is a useful tool for this. As this patient does not fit this criterion, and the pain is classical for sternotomy pain rather than cardiac, myocardial infarction is unlikely.

Question 20 of 328

A 68 year-old man with a history of ischaemic heart disease and type 2 diabetes mellitus is 2 days post right curative hemicolectomy for bowel malignancy.

He has developed chest pain and shortness of breath. The surgical team have requested an urgent medical review.

On examination the patient appears distressed and is complaining of chest pain and breathlessness. Capillary refill time is 5 seconds centrally. The pulse rate is 200bpm and the blood pressure is 87/45mmHg. Oxygen saturations are 92% on fiO2 0.4 via Venturi mask.

There are crackles to the midzones bilaterally on chest auscultation.

A 12-lead ECG reveals a regular broad complex tachycardia with a monomorphic waveform.

Blood results from the morning reveal:

Hb	129 g/l
Platelets	564 * 10 /l
WBC	15.8 * 10 /l
Na	128 mmol/l
K	3.3 mmol/l
Urea	12.9 mmol/l
Creatinine	101 µmol/l
Bilirubin	27 µmol/l
ALP	125 u/l
ALT	34 u/l
Albumin	36 g/

What is the most appropriate initial management?

A. Adenosine 6mg IV
B. Amiodarone 300mg IV
C. Magnesium sulphate 2g IV
D. Synchronized DC shock
E. Metoprolol 5mg IV

D. Synchronized DC shock

In the context of a tachyarrhythmia, a systolic BP < 90 mmHg → DC cardioversion

This question outlines a case of ventricular tachycardia with adverse features including shock and heart failure.

According to the Resuscitation Council guidelines, if a patient with a tachyarrhythmia is unstable (i.e. has adverse features likely to be caused or made worse by the tachycardia - shock, syncope, heart failure or myocardial ischaemia) synchronised cardioversion is the treatment of choice.

If cardioversion fails to restore sinus rhythm, and the patient remains unstable, the next step is to give amiodarone 300 mg IV over 10-20 min and re-attempt electrical cardioversion. The loading dose of amiodarone may be followed by an infusion of 900 mg over 24 h.

If the patient is conscious, cardioversion should be carried out under sedation or general anaesthesia in the presence of an anaesthetist.

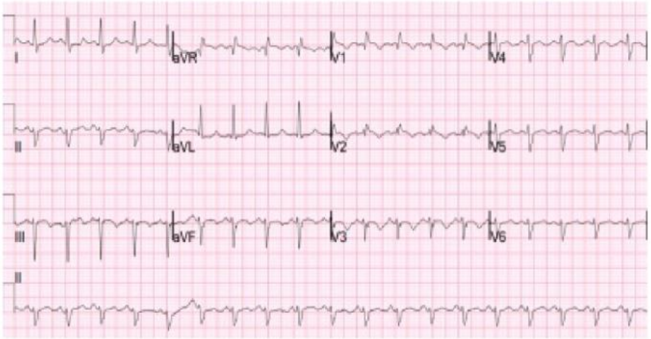
Question 21 of 328

You are called to review a 68-year-old who is complaining of chest pain on the ward. She was admitted 6 days ago after having a stroke which has left her with a mild degree of right-sided hemiparesis. She has some difficultly articulating the nature of the pain but describes it as central.

Her past medical history includes hypertension and hypothyroidism. She has not had a stroke or transient ischaemic attack previously.

On examination, the heart rate is 102/min, blood pressure 122/78 mmHg, respiratory rate 22/min and oxygen saturations 94% on room air.

The nurse recorded an ECG:



What is the most likely diagnosis?

A. Subarachnoid haemorrhage
B. Inferior myocardial infarction
C. Hypokalaemia
D. Posterior myocardial infarction
E. Pulmonary embolism

E. Pulmonary embolism

S1Q3T3 is a classic but uncommon ECG finding in PE

This woman is at risk of a pulmonary embolism given her relative immobility following the stroke.

The tachycardia and tachypnoea are also signs associated with pulmonary embolism.

The ECG shows some of the features that may be associated with pulmonary embolism (sinus tachycardia, RBBB, right ventricular strain). Other features such as the left axis deviation are atypical.

Question 22 of 328

A 76-year-old woman presents to the acute medical assessment unit with a two-month history of palpitations. She reports regularly feeling her heart racing, occasionally associated with pre-syncope. She denies dyspnoea, syncope and chest pain.

She has a medical history significant for asthma, for which she takes regular inhaled beclometasone dipropionate and formoterol, and montelukast, with as required salbutamol. There is no other medical history.

Her observations are:

A. Respiratory rate: 20/min
B. Blood oxygen saturation: 98% on room air
C. Heart rate: 116bpm
D. Blood pressure: 135/68mmHg
E. GCS: 15/15
F. Temperature: 37.4 C

An ECG is performed which shows atrial fibrillation. Blood tests are unremarkable. A transthoracic echocardiogram shows no evidence of valvular heart disease and no evidence of congestive cardiac failure.

What is the most appropriate management?

A. Apixaban + bisoprolol
B. Apixaban + digoxin
C. Apixaban + diltiazem
D. Warfarin + bisoprolol
E. Warfarin + diltiazem

C. Apixaban + diltiazem

Apixaban + diltiazem is the correct answer. This patient has atrial fibrillation with no valvular abnormalities. Her CHA DS -VASc score is 3 and therefore qualifies for anticoagulation with a direct-acting oral anticoagulant (DOAC) such as apixaban. If this patient had mitral stenosis, weighed over 120kg, or had a contraindication to a DOAC, warfarin would be considered. This patient would be suitable for rate control rather than rhythm control - the two-month history suggests this is not acute, there is no clear reversible cause and there is no evidence of heart failure. This patient's asthma is a contra-indication for a betablocker, as beta-blockers can increase airway reactivity. Calcium channel blockers are second-line for rate control in atrial fibrillation. Therefore apixaban + diltiazem is the correct combination of medications.

Apixaban + bisoprolol is the incorrect answer. Beta-blockers are the first-line treatment for rate control in atrial fibrillation, but are relatively contraindicated in asthma - given this patient is on fourth-line treatment for her asthma, beta-blockers are best avoided in this scenario.

Apixaban + digoxin is the incorrect answer. Digoxin is the third-line treatment for rate control in atrial fibrillation. Therefore a calcium channel blocker such as diltiazem is more appropriate. Digoxin would be more appropriate if this patient had evidence of heart failure due to the negative inotropic effects of calcium channel blockers.

Warfarin + bisoprolol is the incorrect answer. DOACs, such as apixaban, are used first line when anticoagulation is indicated for atrial fibrillation. Warfarin is recommended if there is evidence of mitral stenosis however, this patient's echocardiogram has no evidence of valvular disease. As above, diltiazem is more appropriate than bisoprolol due to this patient's asthma.

Warfarin + diltiazem is the incorrect answer. As above, a DOAC is preferable to warfarin in this situation. Diltiazem is the correct drug for rate control in this scenario.

Question 23 of 328

A 78-year-old female was referred to the clinic after several episodes of chest pain. The chest pain came on exertion and was relieved by sitting down. The pain was centrally located and described as crushing in character.

She has a past medical history of hypercholesterolaemia, systemic lupus erythematosus and hypertension.

A myocardial technetium (99mTc) MIBI scan demonstrates the following results:

At rest Normal perfusion Under stress Lateral perfusion defect

What is the most likely explanation of these results?

A. Myocardial infarction of left circumflex artery territory
B. Myocardial infarction of left anterior descending coronary artery territory
C. 70% stenosis of left circumflex artery
D. 70% stenosis of left anterior descending coronary artery
E. Libman-Sacks endocarditis

D. 70% stenosis of left anterior descending coronary artery

If a cardiac MIBI scan shows that the defect is present on stress, but not at rest, this is suggestive of a reversible defect (e.g. myocardial ischaemia secondary to coronary artery stenosis)

A myocardial perfusion (MIBI) scan is carried out to investigate the blood supply to the heart. By comparing the rest with stress images, any areas of ischaemia can classified as reversible or fixed.If the defect is present on stress, but not at rest, then this is suggestive of a reversible defect (e.g. myocardial ischaemia secondary to coronary artery stenosis).

As demonstrated in this case, the defect is present only on stress, which is indicative of a reversible defect such as coronary artery stenosis.

The lateral part of the heart is usually supplied by the left circumflex artery, making this the best answer.

Question 24 of 328

A 46-year-old man is referred to the acute medical assessment unit with a fever of 38.9 C. He reports a three-week history of general malaise, reduced appetite, intermittent sweating, and occasional rigors. The patient has no medical history and has had no previous operations or procedures. He denies any drug allergies.

o

His observations are: Respiratory rate: 24/min

Oxygen saturation: 95% on room air Heart rate: 105bpm BP: 101/62mmHg Temperature: 38.9 C GCS: 15/15

On examination, the patient has a loud early diastolic murmur over the right second intercostal space. Splinter haemorrhages are noted in three of his fingernails.

What is the most appropriate antibiotic to start?

- A. Amoxicillin
- B. Benzylpenicillin
- C. Flucloxacillin
- D. Vancomycin + low-dose gentamicin
- E. Vancomycin + low-dose gentamicin + rifampicin

A. Amoxicillin

IV amoxicillin is the empirical treatment of choice in native valve endocarditis

Fever + a new cardiac murmur raises suspicion of infective endocarditis. A loud early diastolic murmur over the right second intercostal space is suspicious for aortic regurgitation secondary to endocarditis. This will cause the patient's shortness of breath.

Amoxicillin is the correct answer. Given the patient has no medical or surgical history, he should be treated for native valve endocarditis by starting high-dose intravenous amoxicillin. Ideally, six sets of blood cultures should be sent, with as many as possible before the initiation of treatment (although this should not delay treatment if the patient is unstable).

Benzylpenicillin is the incorrect answer. Benzylpenicillin is the treatment for infective endocarditis caused by a fully sensitive Streptococcus organism.

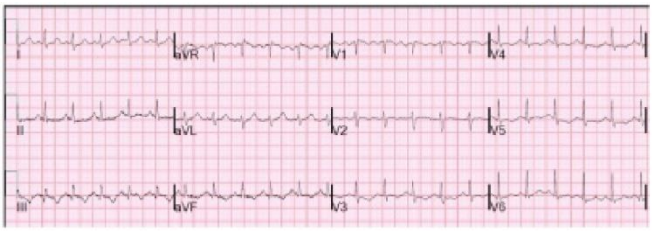
Flucloxacillin is the incorrect answer. Flucloxacillin is used to treat infective endocarditis caused by a fully sensitive Staphylococcus (usually Staphylococcus aureus.)

Vancomycin + low-dose gentamicin is the incorrect answer. Vancomycin + low-dose gentamicin can be used for infective endocarditis in penicillin-allergic patients or patients with methicillinresistant Staphylococcus aureus (MRSA) infective endocarditis.

Vancomycin + low-dose gentamicin + rifampicin is the incorrect answer. Vancomycin + low-dose gentamicin + rifampicin is the treatment for infective endocarditis on a prosthetic valve. This patient has no history of valve replacement.

Question 25 of 328

A 19-year-old student is brought to the Emergency Department by her friends. Around one hour ago she 'collapsed' whilst playing hockey. Her friends describe her complaining that she felt light-headed and then 'fainting' to the ground. She lost consciousness for a few seconds before returning to normal quite quickly. There is no past medical history of note other than the use of Microgynon 30 (a combined oral contraceptive pill). For the past 4-5 days the patient has experienced shortness-of-breath and a central chest pain which is worse when she coughs. On examination her pulse is 120/min, blood pressure 96/60 mmHg and chest auscultation reveals scattered wheezes. An ECG done on admission is shown below:



What is the most likely diagnosis?

- A. Hypertrophic obstructive cardiomyopathy
- B. Vasovagal attack
- C. Acute coronary syndrome
- D. Pulmonary embolism
- E. Asthma attack

D. Pulmonary embolism

It is usual taught that pulmonary embolism (PE) presents with pleuritic chest pain, dyspnoea and haemoptysis. This combination of symptoms is however only found in less than 20% of cases. As PE is a potentially lifethreatening condition it is important to be aware of the wide variety of symptoms and signs that may accompany cases.A lot of patients who develop a PE have risk factors. There is one present in this case - combined oral contraceptive pill use. Tachycardia is also a common sign.

It would be unusual to develop an asthma attack with no previous history of asthma. Occasional wheezes are a relatively common finding in patients following a pulmonary embolism.

The ECG shows a sinus tachycardia and a partial S1Q3T3 - the S wave is not particularly convincing.

Question 26 of 328

A 72-year-old man presents to the emergency department with palpitations. An ECG confirms atrial fibrillation. His CHA2DS2-VASc score is 2 and he is commenced on bisoprolol and edoxaban.

He is referred and attends for catheter ablation, which he undergoes successfully. Post-procedure ECG confirms sinus rhythm.

He is followed up 4 weeks later in the clinic and a repeat ECG confirms the maintenance of sinus rhythm.

What is the best course of action at this stage?

- A. Continue edoxaban for a further 4 weeks then stop
- B. Continue edoxaban for a further 8 weeks then stop
- C. Continue edoxaban long-term
- D. Discontinue edoxaban
- E. Switch to aspirin

C. Continue edoxaban long-term

Patients who've had a catheter ablation for atrial fibrillation still require long-term anticoagulation as per their CHA DS -VASc score

The correct answer is continue edoxaban long-term. Although the ablation has been successful and rhythm control has been achieved, patients in this setting have the same stroke risk as those remaining in atrial fibrillation. Anticoagulation should, therefore, be continued longterm.

Continue edoxaban for a further 4 weeks then stop is incorrect. Continue edoxaban for a further 8 weeks then stop is incorrect.

Question 27 of 328

A 43-year-old lady with a history of schizophrenia presents to the emergency department with palpitations, headaches and dizziness for 3 days. She says that she can feel her heart pounding after which she becomes dizzy and feels faint. She has noted these bouts about three to four times per day. She does not suffer from diabetes and is not hypertensive.

Regarding her headaches, she has had them for a long period of time and is quite convinced that there is some sort of sinister problem with her brain, although her doctors do not believe so. She has a younger sister who has had epilepsy for the last 15 years.

She was diagnosed as having a UTI 4 days ago which is currently being treated with ciprofloxacin. Her medication history includes olanzapine and occasional paracetamol for her headaches.

On examination, her pulse was 135 bpm with an irregular rhythm. Blood pressure was 90/60mmHg. Her systemic examination was unremarkable.

Attachment to a cardiac monitor revealed runs of ill sustained polymorphic tachycardia. She was immediately given IV lidocaine to which there was no response.

Which is the most appropriate next step in management?

A. Immediate DC cardioversion
B. IV amiodarone
C. IV flecainide
D. IV magnesium sulphate
E. IV labetalol

D. IV magnesium sulphate

The scenario above is an acquired long QT syndrome secondary to both ciprofloxacin and olanzapine. Patients with long QT syndromes developsyncope and palpitations as a result of polymorphic ventricular tachycardia (torsades de pointes). Episodes may terminate spontaneously which is usual, but may also evolve into fatal ventricular fibrillation. The corrected QT interval in between the arrhythmia is usually >0.5s.

Question 28 of 328

A 78-year-old gentleman is seen in clinic with long-standing heart failure with reduced ejection fraction (32%). He has had numerous admissions this year with heart failure decompensation and is wondering if there is anything else that you can do for him. You review his ECG, which is in sinus rhythm with a heart rate of 64/min with a QRS of 136 msec and left bundle branch block. His blood pressure is: 98/55 mmHg. You also review his bloods:

Hb	128 g/L	Male: (135-180)Female: (115 - 160)
Platelets	335 * 10 /L	(150 - 400)
WBC	6.8 * 10 /L	(4.0 - 11.0)
Na	136 mmol/L	(135 - 145)
K	4.7 mmol/L	(3.5 - 5.0)
Urea	6.8 mmol/L	(2.0 - 7.0)
Creatinine	126 µmol/L	(55 - 120)

He is currently on carvedilol 25 mg BD, enalapril 10 mg BD, bumetanide 2 mg BD, aspirin 75 mg, ivabradine 2.5 mg BD.

What alteration to this gentleman's management could potentially decrease this gentleman's probability of being re-admitted?

A. Arrange cardiac resynchronisation therapy-pacemaker (CRT-P) implantation
B. Increase ivabradine
C. Increase bumetanide
D. Increase spironolactone
E. Initiate sacubitril/valsartan

A. Arrange cardiac resynchronisation therapy-pacemaker (CRT-P) implantation

Cardiac resynchronisation therapy can be used in patients with a QRS duration of >130 msec and LBBB morphology to improve symptomatology

The European society of cardiology (ESC) guidelines have highlighted the importance of cardiac resynchronisation in patients with heart failure. Evidence exists indicating the importance of implantation of such devices in patients with intraventricular delay with most studies focusing on the impact of left bundle branch block. The stronger evidence related CRT-P in such patients include left bundle branch block (LBBB) and QRS >150 msec; however the presence of LBBB on its own provided that the QRS is longer than 120 msec is associated with improved mortality/morbidity. There is no evidence of benefit from CRT for patients with QRS <120 msec, while the presence of right bundle branch block (RBBB) implies a worse disease state where CRT may not necessarily benefit such patients.

The possibility of altering medications in this case remains attractive however one should be aware of the potential of introducing detriment to the patient in doing so.

This patient is already tolerating a relatively slow heart rate therefore ivabradine could potentially cause further slowing.

He is borderline hypotensive with raised potassium (making the possibility of increasing spironolactone less favourable.

Sacubitril/valsartan remains an option, however the ESC guidelines dictate that in such cases with noted intraventricular delay the first port of call would be the provision of cardiac resynchronisation therapy.

Question 29 of 328

A 54-year-old presents to his general practitioner with fatigue and shortness of breath. He has no significant past medical history and is on no medications.

Clinical examination reveals a mid-diastolic murmur, louder in expiration, and a loud S1. There is no peripheral oedema. The jugular venous pressure is not raised and chest auscultation is normal. The pulse is regular.

From the options listed, what is the most likely diagnosis?

A. Tricuspid stenosis
B. Moderate mitral stenosis
C. Mitral regurgitation
D. Severe mitral stenosis
E. Mitral valve prolapse

B. Moderate mitral stenosis

Mitral stenosis - loud S1

Moderate mitral stenosis is the correct answer. This is characterized by a mid-diastolic murmur with a loud S1 heart sound. Left-sided murmurs are louder in expiration. The most common cause is rheumatic fever. This may not always be apparent from the history if it happened in childhood.

Tricuspid stenosis is incorrect. This can also cause a diastolic murmur with a loud S1. However, it will be louder in inspiration as it is a rightsided murmur.

Mitral regurgitation is incorrect. This causes a pansystolic murmur and a soft S1.

6% 63% 6% 19% 6%Severe mitral stenosis is incorrect. In severe mitral stenosis, the S1 is soft due to lack of pliability and acoustic dampening from a severely calcified valve. Furthermore, there are no obvious clinical features of severe mitral stenosis such as evidence of pulmonary hypertension or right heart failure.

Mitral valve prolapse is incorrect. This murmur may be associated with a 'click' but it is a systolic murmur.

Question 30 of 328

A 37-year-old lady presents to the Emergency Department with a 4-hour history of sudden-onset chest pain and dyspnoea. Her symptoms started whilst she was watching television. She describes the pain as severe, central and non-radiating. She denies a cough and haemoptysis. Her past medical history is unremarkable and her only regular medication is the oral contraceptive pill.

Examination reveals an overweight lady with a pulse rate of 114/min and a blood pressure of 101/63mmHg. Her respiratory rate is 22 and her oxygen saturations are 94% on air. Her JVP is 5cm but there is no peripheral oedema. Auscultation of the chest reveals scattered crepitations. Her heart sounds are dual with no audible murmurs. A portable chest x-ray shows clear lung fields.

A short time later the patient's blood pressure is recorded at 90/59mmHg. A bedside echocardiogram shows elevated right ventricular filling pressures with evidence of strain.

What is the most appropriate management?

A. Surgical embolectomy
B. Alteplase
C. Unfractionated heparin
D. Aspirin, clopidogrel, low molecular weight heparin and furosemide
E. Low molecular weight heparin

B. Alteplase

This patient has presented with chest pain and dyspnoea in combination with tachycardia, hypotension, and hypoxia. This, in combination with the history of obesity and exogenous oestrogen, makes PE the most likely diagnosis. It is worth noting that PE does not always present with pleuritic chest pain and crepitations and wheeze can often be heard on auscultation.The management of PE depends on its classification. Intermediate risk (previously 'sub-massive') PE, characterised by the presence of myocardial injury or right ventricular dysfunction, is treated with low molecular weight heparin. High risk (previously 'massive') PE is characterised by shock or hypotension and is best treated by thrombolysis. The patient in this vignette has become hypotensive and would thus benefit from thrombolytic therapy.

Surgical embolectomy is an effective treatment for massive PE but only a few centres are able to offer this. Consequently, thrombolysis remains the preferred first-line treatment for massive PE.

Acute MI with cardiogenic shock would be a key differential, but the clear lung fields on chest x-ray and lack of peripheral oedema go against this. Therefore, treatment with aspirin and clopidogrel would not be beneficial.

Question 31 of 328

A 58-year-old man attends cardiology clinic with retrosternal chest pain on exertion. He reports the onset of his symptoms when walking more than 50 metres on flat ground or when playing on the floor with his grandchildren. The symptoms have been present at the current level for at least the previous 9 months and the patient denied any episodes of pain at rest. The patients GP has previously attempted treatment with bisoprolol and amlodipine but both medications were discontinued for unwanted effects (bradycardia and ankle swelling respectively). The patient has no other medical history except for hypercholesterolaemia. Regular medications are aspirin 75 mg daily and pravastatin 10 mg daily with nitrate spray and sildenafil used as required. He is formerly a heavy smoker but managed to quit the previous year.

Clinical examination is unremarkable with no evidence of cardiac failure. Blood pressure is 102 / 72 mmHg.

Please see below for the results of previous investigations.

Electrocardiogram: sinus rhythm at 58 beats per minute; borderline left axis deviation; normal QRS complex; non-specific lateral ST segment abnormalities; normal T waves.

Transthoracic echocardiogram: normal valvular function; no regional wall motion abnormality; ejection fraction 55-60 %.

Urea 6.7 mmol / L
Creatinine 80 micromol / L
Sodium 138 mmol / L
Potassium 4.1 mmol / L

Cardiac stress magnetic resonance imaging: significant evidence of ischaemia in the region of the lateral left ventricle; estimated 20 % of LV myocardium with significant evidence of ischaemia.

What is the most appropriate management of the patients chest pain?

A. Ticagrelor
B. Nicorandil
C. Percutaneous coronary intervention
D. Ivabradine
E. Long-acting isosorbide mononitrate

C. Percutaneous coronary intervention

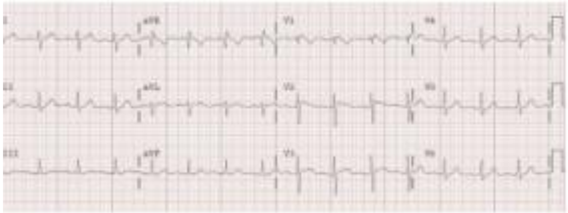
The role of percutaneous coronary intervention in stable angina is a controversial subject. Revascularisation has not been shown to reduce mortality or the rate of myocardial infarction in such patients. However, sub-group analysis of randomised controlled trials suggests some benefit for the above outcomes over medical therapy. Therefore, ESC guidelines recommend offering revascularisation to patients with stable coronary artery disease and ischaemia in > 10 % of the left ventricle. Also, the patient's age and lack of co-morbidities make this the most appropriate strategy in this case.

The other possible answers that are options for the medical management of stable angina are contraindicated in this patient. Both nicorandil and ISMN are contra-indicated for this patient due to his borderline hypotension and use of sildenafil. Ivabradine is contraindicated due to the patient's history of bradycardia. It is also not used in patients with moderate to severe angina symptoms as has been shown to increase the rate of cardiovascular events in such patients.

Ticagrelor is a P2Y12 adenosine diphosphate receptor blocker with utility as an oral antiplatelet agent.

Question 32 of 328

A 30-year-old man is admitted to the Emergency Department after suffering a 'blackout' whilst at work. His colleagues report him collapsing without warning whilst waiting at the water machine. There has never happened before and he is normally fit and well. On examination blood pressure is 102/68 mmHg, pulse 88/min, oxygen saturations 99% on room air and respiratory rate 16/min. An ECG is taken:



What is the most likely diagnosis?

A. Hypertrophic obstructive cardiomyopathy
B. Anterior myocardial infarction
C. Long QT syndrome type 1
D. Arrhythmogenic right ventricular dysplasia
E. Brugada syndrome

E. Brugada syndrome

The ECG shows a Brugada pattern, most marked in V1, which has an incomplete RBBB, a downsloping ST segment and an inverted T wave.

Question 33 of 328

A 66-year-old gentleman presented to the Emergency Department complaining of severe abdominal pain for the last day. The pain occurred immediately after he ate, and he described it as an excruciating pain in the central of his abdomen. It lessened during periods of starvation, and a few hours ago passed dark red blood within his stools on one occasion, leading to his admission. His bowels were otherwise normal and prior to the pain had been relatively well in himself. In addition, he had been feeling unwell for the last couple of days, complaining of night sweats and tiredness. His past medical history comprised angina which had been confirmed three days ago upon undergoing a coronary angiogram which demonstrated diffuse triple vessel disease. In addition, he had a past medical history of hypertension, hypercholesterolaemia and COPD. He smoked 30 cigarettes per day for 25 years and did not consume alcohol and was taking amlodipine 5mg OD, ramipril 7.5mg OD, bisoprolol 2.5mg OD, atorvastatin 20mg OD, beclometasone 200mcg BD & salbutamol PRN.

Examination revealed an obese gentleman with a temperature of 37.6ºC, a heart rate of 118bpm, respiratory rate of 18/min and blood pressure of 142/82 mmHg. Examination of his cardiovascular and respiratory systems revealed the absence of his pedal pulses bilaterally, with no evidence of ulceration or gangrene. A purpuric rash was noted on both halluxes.

Initial investigations revealed the following results:

Hb	122 g/l
Platelets	132 * 10 /l
WBC	11.2 * 10 /l
Eosinophils	0.6 * 10 /l
ESR	52 mm/hr
Na	136 mmol/l
K	5.8 mmol/l
Urea	13.3 mmol/l

Creatinine	158 µmol/l
CRP	38 mg/l

Urinalysis protein +, otherwise nil else

ECG: heart rate 118bpm sinus tachycardia, Q waves II/III/aVR
Blood results 4 weeks ago (done by GP when titrated ramipril)

Na +	142 mmol/l
K +	4.8 mmol/l
Urea	6.6 mmol/l
Creatinine	86 µmol/l

Which one of the following is the most likely diagnosis?

A. Contrast induced nephropathy
B. Cholesterol atheroemboli
C. Polyarteritis nodosa
D. Infective endocarditis
E. Microscopic polyangiitis

B. Cholesterol atheroemboli

Cholesterol atheroemboli may occur as a consequence of manipulation of arteries lined with atheromatous plaques, such as a result of angiography. Cholesterol emboli may involve multiple organs, as a result of widespread dissemination. Signs and symptoms often involve a lowgrade inflammatory response with fever and malaise. Cutaneous emboli lodging may affect the distal extremities (as is evident here by a vasculitic rash). Organs involved include the respiratory system, gastrointestinal system (as is manifest by mesenteric ischaemia here) and the renal system resulting in renal failure, with often proteinuria upon urinalysis. A key differential is contrast nephropathy, but cutaneous involvement and other organ involvement is very uncommon and the renal failure tends to be transient.

Question 34 of 328

A 68 year old gentleman with a past medical history of type 2 diabetes mellitus and hypertension attends the Emergency Department complaining of general malaise and feeling unwell. He had undergone successful primary coronary intervention (PCI) post myocardial infarction 5 weeks previously. He has a temperature of 38.2 degrees Celsius, blood pressure 164/87 mmHg and the doctor notes a lacy reticular rash extending over his legs.

Hb	12.2g/dl
Eosinophils	1.2 * 10 /l9
WBC	14.5 * 10 /l9
Urea	8mmol/l
Creatinine	142µmol/l

What is the most likely cause of his symptoms?

A. Allergic rash to contrast
B. Infected femoral site
C. Cholesterol embolus
D. Infective endocarditis
E. Polyarteritis nodosa

C. Cholesterol embolus

This is a classic story for a cholesterol embolus post an invasive arterial procedure, whereby a plaque has been ruptured and there is lodging of debris in small/medium arteries causing mechanical occlusion, inflammation and end-organ damage. Patients may have the lacy reticular rash of livedo reticularlaris and this, together with acute renal failure and an eosinophilia should lead the clinician to consider this diagnosis.

Patients tend to present with non-specific symptoms such as fever, weight loss and myalgia for weeks to months before developing endorgan damage or stroke.

The current mainstay of treatment is supportive.

Question 35 of 328

A 70-year-old man is admitted to hospital with chest pain associated with nausea and vomiting. There was no recent history of shortness of breath or wheeze. ECG demonstrated inferolateral ST depression and troponin was significantly elevated at 12 hours after symptom onset.

His past medical history included chronic obstructive pulmonary disease, hypertension and a previous MI 3 years previously. The patient reported two exacerbations of his COPD within the last 18 months, neither of which required hospital admission. He had never required intubation or non-invasive ventilation due to his COPD.

Regular medications included bendroflumethiazide 2.5 mg OD, inhaled tiotropium 18 microg OD and inhaled salbutamol 100 microg PRN. There were no known drug allergies. The patient was a retired engineer, an exsmoker and lived independently with his wife.

The initial impression was of non-ST elevation myocardial infarction and treatment was initiated with aspirin, clopidogrel, fondaparinux, ramipril and atorvastatin. The patient subsequently mobilised pain-free on the ward and was discharged home with a plan for outpatient stress echocardiogram.

Recent pulmonary function tests are given in the table below.

Forced vital capacity	105 % predicted
Forced expiratory volume (1s)	67 % predicted
FVC / FEV1	64 % predicted

Transthoracic echocardiogram: no valvular abnormality; mild-moderate systolic impairment of lateral left ventricle; normal right ventricular function

What is the most appropriate plan for beta-blockade therapy for this patient?

A. Low dose bisoprolol with slow up-titration of dose
B. Low dose carvedilol with slow up-titration of dose
C. Low dose bisoprolol
D. Low dose carvedilol
E. Beta-blockade therapy inappropriate

A. Low dose bisoprolol with slow up-titration of dose

NICE guidance on secondary prevention following myocardial infarction includes a recommendation for beta-blocker therapy as it has been shown to reduce mortality. In patients with COPD or asthma, betablockers have sometimes been held due to concerns abut precipitating bronchospasm. However, a large observational study has shown substantial survival benefits for patients with COPD who have suffered a myocardial infarction in starting beta-blockers during hospital admission (adjusted hazard ratio 0.50).

Therefore, beta-blocker therapy is indicated in this patient. The most appropriate choice is the cardio-selective agent (bisoprolol) started at a low dose and slowly up-titrated as per current guidance.

Question 36 of 328

A 34-year-old Afro-Caribbean female presents to the heart failure clinic and would like your advice. Two years ago, she presented with shortness of breath and reduced exercise tolerance three months after her second pregnancy. A chest x-ray demonstrated bilateral alveolar shadowing. The symptoms were improved following diuresis and she remains on an ACE inhibitor, beta blocker and furosemide. She continued to be followed up with echocardiograms, her last demonstrating LV EF of 30%. She would like to try for a third pregnancy.

What would you recommend regarding pregnancy?

A. Pregnancy is advised as normal
B. Pregnancy is not advised
C. Pregnancy is advised with increased ACEi
D. Pregnancy is advised with monthly echocardiograms
E. Pregnancy is advised with increased beta blockade

B. Pregnancy is not advised

The patient presented with peripartum cardiomyopathy, defined as LV ejection fraction reduction in the last month or within 5 months of giving birth, particularly prevalent in black women. The key in this question is whether LV dysfunction was persistent. The last echo demonstrates persistent reduced EF and in this case, pregnancy is NOT advised due to the risk of LV failure progression and significant mortality risk of up to 20% . Those with recovered LV function are likely to also experience deterioration in LV function but pregnancy is not absolutely contraindicated.

Treatment is similar to that of heart failure, consisting of beta blockade, diuretics but avoidance of ACEi due to the risk to the foetus or breastfeeding baby. Digoxin may also have a greater role in PPCM than in classic heart failure. The degree of residual LV dysfunction is difficult to predict but a number of factors appear to be predictive of worse outcomes, namely LVEF <30% at presentation, raised troponin I and LV end-diastolic diameter > 6cm.

Question 37 of 328

A 67-year-old woman with mitral regurgitation attends a cardiology clinic for her regular follow-up. She is asymptomatic, can perform daily tasks, and has a New York heart association (NYHA) functional classification of I. On examination, there is a loud rumbling pan-systolic murmur heard at the apex. She is noted to be in atrial fibrillation, which was not previously known. A recent echocardiogram shows an ejection fraction of 62% with mild left atrial dilatation.

Which feature from the history would be the strongest indication for referral for valve replacement?

A. Age
B. Atrial fibrillation
C. Left atrial dilatation
D. Loud murmur
E. Ejection fraction <65%

B. Atrial fibrillation

New AF in mitral regurgitation -> refer for mitral valve replacement

Surgery is indicated in patients with signs of left ventricular (LV) dysfunction. If LV function is preserved, surgery should be considered in asymptomatic patients with new-onset atrial fibrillation (AF) or pulmonary hypertension. Severe symptomatic mitral regurgitation is also an indication for replacement.

AF develops as a consequence of increased left atrial pressures (due to backflow), resulting in left atrial dilatation. This is, therefore, the strongest indication for replacement.

In the absence of AF, left atrial dilatation only becomes an indication for replacement if it is significant (≥60 mL/m2 body surface area [BSA]).

Age itself is not an indication for mitral valve replacement. Age will be considered alongside other co-morbidities and functional baseline when deciding if she will be suitable for a general anaesthetic for the procedure.

The loudness of a murmur typically doesn't impact the decision to proceed with surgery.

Ejection fraction becomes relevant in asymptomatic patients. 2017 European society of cardiology (ESC) and European association for cardiothoracic surgery (EACTS) guidelines for the management of valvular heart disease suggest that replacement should be considered in asymptomatic patients with an ejection fraction of less than 60%.

Question 38 of 328

An 84-year-old man comes for review. Four weeks ago an opportunistic blood pressure reading was taken and recorded as 150/92 mmHg. You therefore arranged ambulatory blood pressure monitoring (ABPM) along with a standard hypertension work-up. You did not calculate his 10-year cardiovascular risk on account of his age. The following results were obtained:

Na +	141 mmol/l
K +	4.2 mmol/l
Urea	6.5 mmol/l
Creatinine	101 µmol/l
Total cholesterol	4.9 mmol/l
HDL cholesterol	1.2 mmol/l
Fasting glucose	5.5 mmol/l

Urine dipstick was normal. The ECG showed sinus rhythm, 72 bpm and first degree heart block.

The daytime average blood pressure reading was 145/80 mmHg.

What is the most appropriate course of action?

A. Diagnose stage 1 hypertension and advise about lifestyle changes
B. Start treatment with an ACE inhibitor
C. Start treatment with a calcium channel blocker
D. Start treatment with a thiazide-like diuretic
E. Repeat the ABPM

A. Diagnose stage 1 hypertension and advise about lifestyle changes

Stage 1 hypertension is defined by an ABPM reading of >= 135/85 mmHg, with stage 2 hypertension having a cut-off of >= 150/95 mmHg

This patient therefore has stage 1 hypertension. As they are > 80 years they do not need treatment

Question 39 of 328

An 80-year-old lady is brought in by ambulance having been found unresponsive by her granddaughter.

She has a history of vascular dementia, hypertension, atrial fibrillation and mild congestive cardiac failure. She lives alone and is visited by her granddaughter, who is her main carer, once per day. Her granddaughter states she has been more confused recently, finding it hard to know the time of day, or whether she had had meals or medications, but has been otherwise well.

She takes aspirin 75mg once daily, amlodipine 5mg once daily, bisoprolol 5mg once daily, ramipril 2.5mg once daily, and simvastatin 40mg at night.

On examination in the Emergency Department she has a Glasgow coma scale of 10. Her heart rate is 30/min and her blood pressure is 68/36 mmHg. Her saturations are 95% on 60% oxygen with a respiratory rate of 30 breaths per minute.

She has a regular heart rhythm and normal first and second heart sounds with an S3 gallop. Her jugular venous pressure is raised at 6 cm and she has pitting oedema to the knees. Her capillary refill is 4 seconds and she is peripherally cool. She has fine bibasal crepitations in both lungs. Her abdomen is soft and non-tender. Her temperature is 36.1ºC.

Her ECG shows complete heart block with a ventricular rate of 30/min. Her QRS complexes are broad with a right bundle branch block pattern. There are signs of left ventricular hypertrophy but no T wave or ST segment abnormalities.

She is given 3mg of IV atropine in 500 mcg boluses but the heart rate remains 30/min with a blood pressure of 67/40 mmHg.

Which further medication is most likely to result in a sustained improvement in her heart rate?

A. Aminophylline
B. Dopamine
C. Glucagon
D. Isoprenaline
E. Noradrenaline

C. Glucagon

This lady has severe bradycardia and is haemodynamically unstable with signs of acute heart failure.

Resus Council Advanced Life Support Guidelines advocate atropine, up to 3mg, as the initial therapy for this. However, this lady has not responded to this initial measure. The next step would be a choice of a number of drugs or transcutaneous pacing.

This lady had a history of dementia with a recent cognitive decline, lives alone and is on both a beta blocker and a calcium channel blocker. This combined with new complete heart block on her ECG, without signs of new ischaemia, is suggestive of an accidental overdose. This makes glucagon the most logical choice to further improve heart rate as it bypasses the beta blockade.

Aminophylline, isoprenaline and dopamine are all other recommended second-line drugs in unstable bradycardia, along with adrenaline (rather than noradrenaline) and could be administered if there is an unsatisfactory response to glucagon.

In practice, it is worth preparing to transcutaneously pace while furthers drugs are given, in case the bradycardia proves refractory to all medication or the clinical condition worsens further.

Question 40 of 328

A 72-year-old gentleman with previous myocardial infarctions and coronary stents presents with increased leg swelling. This has arisen over two weeks. He is still well and denies any shortness of breath but has an occasional cough at night time. He is managed on aspirin, clopidogrel, ramipril and atorvastatin.

On further questioning, he admits feeling cold at times and feels low in energy for which his GP started citalopram 2 months ago.

He denies any further chest pains. His previous records indicate disease in the circumflex and right coronary arteries previously. He admits to drinking 20-30 units of alcohol a week.

On examination, he appears well. He has bilateral pitting leg oedema with extension into the sacral area. There is a pansystolic murmur over the sternum. His abdomen is soft with a tender hepatomegaly. There is no splenomegaly, ascites, jaundice or asterixis. His JVP is raised with a double flicker pattern which peaks up to the ear lobe on the second flicker. Current blood pressure is 145/86 mmHg.

Hb	95 g/l	Na	137 mmol/l
Platelets	476 * 10 /l	K	4.2 mmol/l
WBC	10.2 * 10 /l	Urea	4.3 mmol/l
Neuts	9.6 * 10 /l	Creatinine	112 µmol/l
Bilirubin	4 µmol/l	ALP	165 u/l
ALT	103 u/l	Albumin	34 g/l
PT 12.2 (14-18 normal range)	APTT 34 (34-42 normal range)		

Chest x-ray	enlarged cardiac shadow, no effusions or lung field shadowing
ECG	sinus rhythm with Q waves in V2-V3
Urine dip	2+ protein, no blood

What is the likely diagnosis?

F. Tricuspid regurgitation
G. Cirrhosis
H. Hypertensive cardiomyopathy
I. Nephrotic syndrome
J. Hypothyroidism

A. Tricuspid regurgitation

Prominent V waves on JVP → tricuspid regurgitation

This gentleman presents with leg swelling and a raised JVP which are both signs of right-sided heart failure. Nephrotic syndrome and hypothyroidism can present with swollen legs but would not have a raised JVP. Hypertensive cardiomyopathy does occur but there is no mention of previous hypertension and we do not know his blood pressure. He does not have cirrhosis given there are no signs of liver disease and his clotting is normal though his LFTs are raised. This leaves tricuspid regurgitation as the answer. He most likely developed this after suffering a posterior myocardial infarction. The gaint V waves on JVP are a classic sign.

Question 41 of 328

A 45-year-old woman develops severe central chest pain. An ECG in the ambulance shows ST segment elevation in leads: I, aVL, V2-6. Shortly after she arrives in hospital the pain resolves and a second ECG is entirely normal. She has had three similar episodes of chest pain in the past. All episodes of chest pain have come on at rest. Blood pressure is 140/80 mmHg, heart rate is 90 beats per minute and hearts sounds are normal. She underwent coronary angiography following a previous episode of chest pain three weeks ago, which showed no significant coronary artery disease. An echocardiogram is normal.

What is the likely diagnosis?

A. Acute anterolateral myocardial infarction
B. Crescendo angina
C. Da Costas syndrome
D. Prinzmetal's variant angina
E. Takotsubo cardiomyopathy

D. Prinzmetal's variant angina

This woman is likely to be suffering from Prinzmetal's variant angina. Classically her pain occurs at rest and the ECG demonstrates ST segment elevation that disappears as the pain abates. Normal coronary angiography supports this diagnosis, however in many Prinzmetal's angina patients there is co-existing coronary artery disease. Symptoms and ECG changes are unlikely to be reproduced with exercise testing in Prinzmetal's angina.

Prinzmetal's angina is caused by coronary artery spasm, however the underlying pathophysiology causing spasm is not currently well understood. In some patients it is associated with other vasospastic disorders such as Raynaud's phenomenon.

The main stay of treatment is:

- Avoiding precipitants of spasm such as smoking.
- Calcium channel blockers, nitrates and/or nicorandil.

Question 42 of 328

You are reviewing in clinic a 67-year-old man who has type 2 diabetes. His glycaemic control is reasonable with metformin therapy; the latest HbA1c is 54 mmol/mol (7.1%). A few weeks ago he was noted to have a clinic blood pressure reading of 152/90 mmHg. A 24 hour blood pressure monitor was requested. The report shows his average blood pressure was 142/88 mmHg.

What is the most appropriate course of action?

A. Do nothing for now, monitor his blood pressure regularly
B. Start an ACE inhibitor
C. Start a calcium channel blocker
D. Repeat the 24 hour blood pressure monitor in 4-8 weeks time
E. Request an ultrasound of his kidneys

B. Start an ACE inhibitor

Hypertension in diabetics - ACE inhibitors/A2RBs are first-line regardless of age

This patient has stage 1 hypertension as defined by NICE. He should however be treated because he has underlying diabetes.

The first-line treatment for a patient aged > 55 years is a calcium channel blocker. However, in patients with diabetes ACE inhibitors are used firstline due to their renoprotective effect.

Question 43 of 328

A 54-year-old woman presents to her general practitioner complaining of a rash on her chest, face and upper arms. The rash is transient and seems to mostly occur in the summertime. She has a past medical history of hypertension, palindromic rheumatism and heart failure with preserved ejection fraction. She takes amlodipine, furosemide, indapamide and hydroxychloroquine.

On examination, there is a maculopapular rash on her chest and distal forearms. It is not itchy.

Bloods tests:

Antinuclear antibody	negative

What is the likely explanation for her symptoms?

A. Furosemide
B. Hydroxychloroquine
C. Indapamide
D. Solar urticaria
E. Systemic lupus erythematosus

C. Indapamide

Thiazides may cause photosensitivity

Indapamide is correct. Thiazide diuretics are a well-recognised cause of a photosensitive rash. The patient's rash occurs mostly in the summertime and on sun-exposed areas and is therefore consistent with this reaction.

Furosemide is incorrect. This is mainly associated with bullous skin reactions.

Solar urticaria is incorrect. The rash, in this case, is maculopapular and non-pruritic and therefore is not consistent with a diagnosis of urticaria.

Hydroxychloroquine is incorrect. This medication is associated with rashes but is actually used to treat conditions associated with photosensitive rashes and is therefore not the likely cause.

SLE is incorrect. Photosensitive rashes are common in lupus. However, the ANA is negative, which makes a diagnosis of the systemic form of lupus, overwhelmingly unlikely.

Question 44 of 328

A 40-year-old man with a family history of sudden cardiac death presents to the emergency department with palpitations and exerciseinduced syncope. There is no associated chest pain.

An ECG is performed and shows T wave inversion in leads V1-3.

What is the most likely underlying pathology is in this case?

A. Arrhythmogenic right ventricular cardiomyopathy
B. Brugada syndrome
C. Hypertrophic obstructive cardiomyopathy
D. Myocardial infarction
E. Pulmonary embolism

A. Arrhythmogenic right ventricular cardiomyopathy

Arrhythmogenic right ventricular cardiomyopathy - T wave inversion in V1-3

The ECG described here is characteristic of **arrhythmogenic right ventricular cardiomyopathy**, which is consistent with the clinical presentation. This includes T wave inversion in precordial leads V1-3 (possibly also V4/5). Epsilon waves (small waves noted immediately after the QRS complex) are also seen in some cases however is not as common as T wave inversion.

Brugada syndrome is a potential hereditary cause of sudden cardiac death but is associated with ST elevation in leads V1-3 rather than the changes described here.

Hypertrophic obstructive cardiomyopathy is another potential cause of sudden cardiac death but is associated with increased precordial voltages and Q waves in the lateral, and possibly inferior, leads rather than the changes described here.

This presentation would be atypical for a **myocardial infarction**. The patient is not suffering from chest pain and instead reports symptoms that are more consistent with an arrhythmic cause. Although a myocardial infarction cannot be ruled out from this information alone, arrhythmogenic right ventricular cardiomyopathy is a more likely explanation, particularly given the strong family history.

Pulmonary emboli are associated with RV strain which can cause the T wave changes in V1-4 described in this question, however, epsilon waves are characteristic of arrhythmogenic right ventricular cardiomyopathy. Pulmonary emboli may also be associated with sinus tachycardia, right bundle branch block, right axis deviation, and S1Q3T3 patterns.

Question 45 of 328

A 52-year-old female who you see regularly represents to your clinic after routine blood tests having recently been started on ramipril for her hypertension. Her blood pressure (BP) prior to initiation of ramipril in the clinic was 145/98mmHg and her baseline creatinine prior to treatment was 100umol/L. On review today her clinic BP appears to be well controlled at 132/84mmHg and her bloods show her creatinine has risen to 125umol/L.

What is the most appropriate step in the management of her hypertension?

A. Reduce dose of ramipril
B. Continue current dose of ramipril
C. Stop ramipril and consider angiotensin receptor blocker
D. Increase dose of ramipril
E. Stop ramipril and consider calcium channel blocker

B. Continue current dose of ramipril

The main consideration here is the renal function, which shows an increase in creatinine by 25%. Her BP appears to be well controlled. The BNF recommends the angiotensin-converting enzyme inhibitors should only be stopped if the creatinine increases by 30% or eGFR falls by 25% or greater. This lady's results are within these limits and have shown good effect. It would therefore be pertinent to continue the ramipril at the current dose and monitor the renal function as per normal protocol

Question 46 of 328

A 43-year-old with no past medical history presents with 6 months history of exertional dyspnoea. Her echocardiogram demonstrated impaired right ventricular function with raised pulmonary arterial pressure of 78 mmHg, good systolic function and ejection fraction 80%. Right and left heart catheterisation demonstrated pulmonary arterial hypertension, with a pulmonary arterial pressure of 49/24 mmHg, V/Q scan demonstrated no chronic pulmonary emboli, CTPA no acute emboli, ultrasound abdomen no portal hypertension. She is at present comfortable at rest but is short of breath on minimal activity.

What is the appropriate management?

A. No treatment
B. IV prostaglandin
C. Oral sildenafil
D. Atrial septostomy
E. List for lung transplantation

C. Oral sildenafil

Treatment for pulmonary arterial hypertension is guided by the patients functional status. Patients with class I require no symptomatic treatment while those with class IV require prostaglandins, most likely intravenously. This patient is short of breath on minimal activity and hence functional class III. Endothelin receptor antagonists (e.g. bosentan) or phosphodiesterase V inhibitor (e.g. sildenafil) are appropriate. Atrial septostomy and lung transplantation are appropriate for patients who remain symptomatic at functional class IV despite intravenous prostaglandins and subsequent combination therapy with bosentan or sildenafil.

Question 47 of 328

A 47-year-old woman presents to an endocrinology clinic with a history of hypertension. She was first diagnosed with hypertension following a private health assessment 8 years ago and at that time underwent testing to exclude any secondary causes of her hypertension. She was subsequently commenced on ramipril, which was up titrated to 10mg. When her blood pressure remained above 150/100mmHg, her GP added amlodipine 10mg and then bendroflumethiazide 2.5mg. During a recent medication review, the patient's blood pressure was noted to be 170/100mmHg. Her GP thus referred her to the endocrine team for a reconsideration of secondary hypertension. On questioning, she denies any chest pain, palpitations, change in vision, or symptoms consistent with postural hypotension. She has no family history of hypertension, does not smoke and drinks minimal alcohol.

On clinical examination, heart sounds are normal and chest clear, abdomen soft and non-tender with no organomegaly.

Her test results are as follows:

Na	137 mmol/L	(135 - 145)
K	4.6 mmol/L	(3.5 - 5.0)
Bicarbonate	25 mmol/L	(22 - 29)
Urea	3.0 mmol/L	(2.0 - 7.0)
Creatinine	68 µmol/L	(55 - 120)
Hba1c	48 mmol/mol	(42-47)

CT chest / abdomen / pelvis shows no abnormalities. Echocardiogram demonstrates left ventricular hypertrophy only. Urine dip:

Blood	-
Protein	-
Glucose	Trace

Given the diagnosis, which of the following agents would be most appropriate?

A. Doxazosin
B. Spironolactone
C. Furosemide
D. No indication for further anti-hypertensive agents
E. Candesartan

A. Doxazosin

Poorly controlled hypertension, already taking an ACE inhibitor, calcium channel blocker and a standard-dose thiazide diuretic. K+ > 4.5mmol/l - add an alpha- or beta-blocker

This patient has resistant hypertension, that is to say, hypertension which has not responded to three appropriately dosed anti-hypertensive agents. Effective treatment is needed given her evidence of end-organ damage (left ventricular hypertrophy).

Given that her potassium is > 4.5, spironolactone would not be recommended.

Furosemide would not be recommended in a patient without evidence of heart failure prior to the trial of alpha blockade.

Candesartan should not be co-prescribed with ramipril.

Doxazosin (an alpha-blocker) or alternatively a beta-blocker, would be the most appropriate 4th line antihypertensive agent.

Question 48 of 328

A 64-year-old male presents sudden onset back pain while painting a wall at home, radiating to his left anterior chest and jaw, associated with nausea and vomiting. It appears to be constant for the past 3 hours since onset and is his first ever episode. His past medical history includes hypertension, hypercholesterolaemia and one previous transient ischaemic attack.

His ECG demonstrates left ventricular hypertrophy by voltage criteria and T-wave inversion in I, aVF and V5 and V6. On examination, his heart sounds are both present with a soft systolic murmur. The chest is clear and abdomen is soft and non-tender without a pulsatile mass. Both radial pulses are intact with no delay. There is a mild radio-femoral delay. His blood pressure is stable at 134/80 mmHg, heart rate 124/min and sinus rhythm. His bloods are as follows:

Hb	123 g/l
MCV	82 fl
Platelets	204 * 10 /l9
WBC	9.2 * 10 /l9
Troponin T	240 (normal range < 32)

His pain settles transiently with 2.5mg of subcutaneous morphine.

What is the most appropriate immediate action?

A. Aspirin 300mg and clopidogrel 300mg and treatment dose low molecular weight heparin
B. Intravenous fluids
C. Blood transfusion
D. Coronary angiogram +/- stent as appropriate
E. CT aorta

E. CT aorta

The combination of an atypical history of back and chest pain, persistent pain, non-specific ECG changes, mildly raised troponin and an unexplained sinus tachycardia should raise suspicion that this is not a simple diagnosis of acute coronary syndrome. The clue lies in the distal pulses: the lack of radial-radial delay suggests any aortic pathology occurs below the subclavian artery origin. The mild radial-femoral delay suggests a descending artery lesion. The patient is unwell but not actively exsanguinating as evidenced by the stable Hb and blood pressure. An underlying diagnosis of acute aortic syndrome, a new term classifying all acute aortic pathology including dissections, ulcers and aneurysms, must be ruled out by high definition CT aorta with contrast.

Question 49 of 328

A 54-year-old man is reviewed on the cardiac ward some 2 days after admission with an acute coronary syndrome event. He has suffered an inferior myocardial infarction some 4 years ago, for which he was stented, and takes aspirin together with atorvastatin, ramipril, bisoprolol and indapamide. Examination on the ward is unremarkable, and he is deemed fit for discharge.

Which of the following is the most appropriate option for anti-platelet therapy on discharge?

A. Aspirin and clopidogrel
B. Aspirin only
C. Aspirin and ticagrelor
D. Aspirin and dipyridamole
E. Aspirin and prasugrel

C. Aspirin and ticagrelor

Nice guidelines now recommend secondary prevention with ticagrelor and aspirin, not clopidogrel and aspirin after ACS

NICE guidance is clear for patients who are medically managed for acute coronary syndrome, it states that:

Prescribe DAPT (dual anti-platelet therapy). This is aspirin 75 mg daily plus ticagrelor 90 mg twice a day for 12 months. Ticagrelor is the preferred choice (even in those previously treated with clopidogrel) unless the bleeding risk outweighs the benefit. For people at high risk of bleeding, continue DAPT for at least one month.

For people with MI at high ischaemic risk who have tolerated DAPT without a bleeding complication, consider treatment with DAPT (ticagrelor 60 mg twice daily) in addition to aspirin for longer than 12 months and up to 36 months. If ticagrelor is not suitable, consider clopidogrel 75 mg daily (as well as aspirin) for longer than 12 months.

Aspirin and prasugrel is only recommended for patients undergoing percutaneous intervention and stenting, and aspirin and dipyridamole was formerly used for secondary prevention of stroke. This has now been replaced for secondary prevention of stroke by clopidogrel alone.

Question 50 of 328

A 58 year-old man with a background of is ischaemic heart disease and Crohn's disease has developed colonic enterocutaneous fistulae. He is admitted to hospital under the surgical team and a temporary ileostomy is formed to defunction the bowel and promote healing.

Two days post-operatively he develops palpitations and the surgical team request your assistance.

On examination the pulse rate is 220bpm and the blood pressure is 135/90mmHg. Oxygen saturations are 96% on 2L nasal oxygen.

The chest is clear to auscultation.

A 12-lead ECG reveals a wide-complex tachycardia with a polymorphic waveform.

Blood tests from the morning reveal:

Hb	129 g/l
Platelets	643 * 10 /l
WBC	13.8 * 10 /l
Na	129 mmol/l
K	3.3 mmol/l
Phosphate	0.63 mmol/l
Mg	0.59 mmol/l
Urea	8.1 mmol/l
Creatinine	97 µmol/l
Bilirubin	15 µmol/l
ALP	143 u/l
ALT	53 u/l
Albumin	31 g/l

What is the most appropriate initial management?

A. Adenosine 6mg IV
B. Magnesium sulphate 2g
C. Metoprolol 5mg IV
D. Synchronised DC shock
E. Amiodarone 300mg IV

B. Magnesium sulphate 2g

In this scenario this patient has developed polymorphic ventricular tachycardia with no adverse features. This is likely to be secondary to electrolyte derangement in this case.

Initial management steps include stopping all drugs known to prolong the QT interval. Correct electrolyte abnormalities, especially hypokalaemia. Give magnesium sulphate 2 g IV over 10 min (= 8 mmol).

If adverse features are present (shock, syncope, myocardial ischaemia or heart failure) arrange immediate synchronised cardioversion.

Question 51 of 328

A 83 year old lady presents to heart failure follow up clinic. She has a history of NSTEMI and gallstones. Her symptoms are reasonably poorly controlled. She is able to walk around her bungalow, but struggles to walk to her local shops 100m away due to breathlessness.

She had recently seen her GP who added spironolactone to her regular medications due to persistent hypokalaemia. Her potassium has since normalised.

Her latest echo reveals an ejection fraction of 25%. Her ECG is sinus rhythm Her last BNP was 1000 pg/ml.

She is currently taking senna, ramipril 10mg, aspirin 75m, frusemide 40mg bd, simvastatin 40mg, and spironolactone 50mg.

Her observations at clinic are:

- oxygen saturations: 94% on room air
- blood pressure: 126/66 mmHg
- heart rate: 84/min

Which additional medication would be beneficial?

A. Ivabradine
B. Bisoprolol
C. Digoxin
D. Diltiazem
E. Atenolol

B. Bisoprolol

This patient is already taking some prognostically beneficial medications; ACE inhibitor and aldosterone antagonist. The additional of a beta blocker would be a beneficial medication; both from a preventative of re-modelling and a reduction in heart rate.

Heart rate is a well established modifiable risk factor, which when appropriately controlled can improve morbidity and mortality in heart failure. This patient's heart rate is not well controlled.

Bisoprolol, carvedilol, nebivolol and metoprolol are the only evidencebased cardioselective beta blockers for heart failure patients.

Ivabradine is an If (funny) channel blocker, which reduces heart rate by blocking the If current in the sinoatrial node. NICE guidelines advocate the use of ivabradine for heart failure in a select group of patients:

- Ejection fraction <35%
- Heart rate >75/min
- Sinus rhythm
- NYHA class 2-4
- Maximally titrated beta blocker therapy.

Since this patient is not on maximal beta blocker therapy, it would not be appropriate to commence ivabradine. Diltiazem may be used as an antianginal medication, but this patient does not have angina. Digoxin may be of benefit in heart rate, especially if the patient has atrial fibrillation, but should only be considered following failure of first and second line therapies.

Question 52 of 328

A 72-year-old man attends the cardiology clinic. He has a past medical history of heart failure. He advises you that he has had worsening shortness of breath over the last few weeks. He is now symptomatic at rest. His regular medications include ramipril, bisoprolol, furosemide and eplerenone. His observations are as follows: heart rate 70 beats per minute, blood pressure 125/75 mmHg, respiratory rate 22 breaths per minute, and SpO2 94% on air. On examination, you note pitting oedema to the mid calves and bilateral coarse crepitations.

An echocardiogram and ECG is performed:

Echo	Left ventricular failure with an ejection fraction of 25%
ECG	Sinus rhythm; QRS 140 msec; Dominant S wave in V1 and a broad monophasic R wave in lateral leads (I, aVL, V5-6)

What treatment is most likely to be beneficial to long-term survival?

A. Bendroflumethiazide
B. Bumetanide
C. Cardiac resynchronisation therapy with defibrillator (CRT-D)

D. Cardiac resynchronisation therapy with pacing (CRT- P)
E. Ivabradine

D. Cardiac resynchronisation therapy with pacing (CRT- P)

Cardiac resynchronisation therapy can be used in patients with a QRS duration of >130 msec and LBBB morphology to improve symptomatology

The patient has symptomatic (NYHA class IV) cardiac failure requiring escalation of treatment. He is already on an ACE-I, beta blocker, mineralocorticoid receptor antagonist, and loop diuretic. He has severe left ventricular systolic dysfunction (LVSD) with an ejection fraction of 25%. It is important to note the presence of LBBB with a significantly prolonged QRS. The dominant S wave in V1 and a broad monophasic R wave in the lateral leads are classic of LBBB.

Cardiac resynchronisation therapy with pacing (CRT- P) is correct. Cardiac resynchronisation therapy (CRT) is recommended as a treatment option for people with heart failure who have left ventricular dysfunction with a left ventricular ejection fraction (LVEF) of 35% or less, and LBBB with a QRS duration > 130 msec. The patient in this clinical case, therefore, fulfils these criteria. Many randomised controlled trials have shown that CRT significantly prolongs survival in patients with heart failure. The NICE guidelines have criteria on whether to choose CRT-D or CRT-P. In patients with NYHA class IV (as seen in this case), CRT-P is the preferred modality. In this case, it would also be important to consider the use of sacubitril/valsartan. The PROVE-HF trial (as well as case reports and observational studies) have shown that sacubitril/valsartan is able to induce reverse cardiac remodeling, which has the potential to avoid ICD or CRT implantation.

Bendroflumethiazide is incorrect. Bendroflumethiazide can be used for mild or moderate heart failure. Although diuretics can have major benefits for patient symptomatology, they have no benefit to long-term survival.

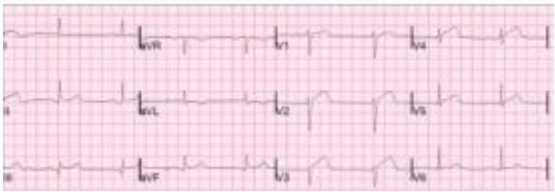
Bumetanide is incorrect. Bumetanide is a type of loop diuretic which has been shown to have greater bioavailability compared to furosemide due to increased absorption in patients who have oedematous bowels. Although diuretics can have major benefits for patient symptomatology, they have no benefit to long-term survival.

Cardiac resynchronisation therapy with defibrillator (CRT-D) is incorrect. As the patient has NYHA class IV symptomatology, CRT-P is the correct option as per NICE guidelines.

Ivabradine is incorrect. Ivabradine is recommended as an option for treating chronic heart failure for patients who are in sinus rhythm with a heart rate of 75 beats per minute or more and who are already on standard therapy with ACE inhibitors, beta-blockers and aldosterone antagonists. The patient in this clinical case has a heart rate of 70 beats per minute and ivabradine is therefore not indicated.

Question 53 of 328

A 79-year-old man presents with severe central chest pain which started around 90 minutes ago. He is known to have ischaemic heart disease and had a coronary artery bypass graft (CABG) five years ago. On arrival in the Emergency Department he is clammy and vomiting. An ECG is taken:



What is the most accurate description of what is shown on this ECG?

(MI = myocardial infarction, STEMI = ST elevation myocardial infarction, NSTEMI = non-ST elevation myocardial infarction)

A. Normal ECG
B. Current inferior NSTEMI
C. Current anterior NSTEMI
D. Current inferior STEMI
E. Current anterior STEMI

A. Current anterior NSTEMI

Ischaemic changes in leads V1-V4 - left anterior descending

In the anterior leads (V2-4) ST elevation can clearly be seen indicating a STEMI. Angiogram revealed an acutely occluded saphenous vein graft to the left anterior descending which was opened with percutaneous coronary intervention.

In the inferior leads there is a pathological Q wave in lead III and nondiagnostic Q waves in leads II and aVF (see below for definitions). These changes suggest that this patient has previously had an inferior MI.

The older and simpler definition of a pathological Q wave:

- Q-wave of >=0.04 s and an amplitude >=25% of the R-wave in that lead

The new (and much more complicated) Joint European Society of Cardiology/American College of Cardiology definition of a pathologic Q wave is:

- any Q-wave in leads V2 - V3 >= 0.02 s or QS complex in leads V2 and V3
- Q-wave >= 0.03 s and > 0.1 mV deep or QS complex in leads I, II, aVL, aVF, or V4 - V6 in any two leads of a contiguous lead grouping (I, aVL,V6; V4 - V6; II, III, and aVF)
- R-wave >= 0.04 s in V1 - V2 and R/S >= 1 with a concordant positive T-wave in the absence of a conduction defect

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A 45-year-old woman presents to the emergency department after a road traffic accident where she sustains multiple injuries including an open fracture of her left tibia and fibula. The following day she has an open reduction and internal fixation of the left tibia and fibula and remains in hospital for physiotherapy. She is quite immobile during this period and then develops subsequent painful swelling and erythema of the left calf. Subsequent ultrasonography confirms a left-sided above knee deep vein thrombosis.

Before treatment starts, she develops sudden onset weakness in her right leg and right arm, dysarthric speech and a reduction in conscious level. Subsequent CT scanning confirms the presence of a left-sided infarct in the middle cerebral artery territory. Doppler investigation of the carotids shows a 20% stenosis on the left side and 10% on the right side. The 24- hour tape shows average heart rate 52bpm with 1.5s pauses maximum, sinus bradycardia

Which of the following features from further investigations would best explain this woman's presentation?

A. Anti phospholipid antibody positive
B. Protein C deficiency
C. Patent foramen ovale (PFO)
D. Hypercholesterolaemia
E. Dilated cardiomyopathy on ECHO

C. Patent foramen ovale (PFO)

This unfortunate lady has developed a thrombus as a result of her recent operation and subsequent immobility. Part of the thrombus has embolised into her left middle cerebral artery.

One would normally expect the source of the embolus to be from the carotids or from the heart. However, the minimal carotid stenosis and lack of history of atrial fibrillation make this less likely. The finding of a

PFO on an ECHO would explain how a thrombus from the leg would reach the brain rather than the lungs.

PFOs are extremely common and have been found in 25-30% of people at autopsy. PFOs have been linked to an increased risk of stroke. There is also some evidence that subclinical DVTs in the presence of a PFO may be one cause for cryptogenic stroke http://content.onlinejacc.org/article.aspx?articleid=1120253

The evidence is however limited that PFO closure will reduce the risk of stroke.

The other options except E are all risk factors for stroke but cannot explain the mechanism of embolism with near-normal carotids and sinus rhythm. E is not associated with an increased risk of embolic stroke.

Question 55 of 328

A 52-year-old man of Afro-Caribbean descent is reviewed after having ambulatory blood pressure monitoring (ABPM). This had been ordered after a works medical recorded a blood pressure of 156/86 mmHg. He smokes 20 cigarettes/day and has a family history of ischaemic heart disease. The results show the following:

Average daytime blood pressure: 142/88 mmHg QRISK 10 year cardiovascular risk: 23%

Urine dipstick: NAD

ECG: sinus rhythm, rate 78/min

Na +	141 mmol/l
K +	4.6 mmol/l
Urea	4.5 mmol/l
Creatinine	82 µmol/l
Total cholesterol	5.4 mmol/l
HDL cholesterol	0.9 mmol/l
Fasting glucose	5.3 mmol/l

What is the most appropriate course of action?

A. Start treatment with a calcium channel blocker
B. Diagnose stage 1 hypertension and advise about lifestylechanges
C. Start treatment with an ACE inhibitor
D. Start treatment with a thiazide-like diuretic
E. Repeat the ABPM

A. Start treatment with a calcium channel blocker

Stage 1 hypertension is defined by an ABPM reading of >= 135/85 mmHg, with stage 2 hypertension having a cut-off of >= 150/95 mmHg.

This patient therefore has stage 1 hypertension. We should therefore treat if < 80 years of age AND any of the following apply; target organ damage, established cardiovascular disease, renal disease, diabetes or a 10-year cardiovascular risk equivalent to 20% or greater, as is present in this case.

ACE inhibitors are usually the treatment of choice for patients < 55-years-old but as this patient is of Afro-Caribbean descent a calcium channel blocker should be used instead.

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A patient with a severe rheumatic heart disease is about to attend a gastroscopy with oesophageal dilatation. He has no current active gastrointestinal infection.

What endocarditis prophylaxis is recommended?

A. Ciprofloxacin PO 30 min pre-procedure
B. None
C. IV ampicillin at time of surgery
D. IV cefazolin at time of surgery
E. Erythromycin PO 30 min pre-procedure

B. None

Prophylaxis is only indicated when one of the following are met:

Prosthetic cardiac valve or prosthetic material used for cardiac valve repair
Previous infective endocarditis
Cardiac transplantation with the subsequent development of cardiac valvulopathy
Congenital heart disease but only if it involves:Unrepaired cyanotic defects, including palliative shunts and conduitsCompletely repaired defects with prosthetic material or devices whether placed by surgery or catheter intervention, during the first 6 months after the procedure (after which the prosthetic material is likely to have been endothelialised) Repaired defects with residual defects at or adjacent to the site of a prosthetic patch or device (which inhibit endothelialisation

Congenital heart disease but only if it involves:

- Unrepaired cyanotic defects, including palliative shunts and conduits Completely repaired defects with prosthetic material or devices whether placed by surgery or catheter intervention, during the first 6 months after the procedure

(after which the prosthetic material is likely to have been endothelialised)

- Repaired defects with residual defects at or adjacent to the site of a prosthetic patch or device (which inhibit endothelialisation)

Active or not active gastrointestinal infection does not alter your management of prophylaxis, but would be advisable to defer the procedure if possible.

Question 57 of 328

A 56-year-old gentleman was admitted to the Emergency Department with a three-hour history of severe central chest pain. The pain came on suddenly whilst he was walking. The pain has remained severe in intensity since onset. He also felt short of breath at rest with associated clamminess. There was no history of trauma. His past medical history comprised of hypertension, hypercholesterolaemia and gout. He was prescribed amlodipine 5mg OD, ramipril 7.5mg OD, atorvastatin 20mg OD, allopurinol 100mg and orlistat 120mg TDS. He smoked 20 cigarettes per day and consumed 30 units of alcohol per week.

On examination, he appeared pale and clammy. His blood pressure was 196/78 mmHg in his right arm and 152/65 mmHg in his left arm. His heart rate was 92bpm. His oxygen saturations were 96% on air. Examination of his cardiovascular system revealed the presence of a prominent early diastolic murmur in the aortic area. Examination of his respiratory system revealed tachypnoea but no added sounds on auscultation. Examination of his gastrointestinal and neurological systems was unremarkable.

He was admitted to the resuscitation bay and commenced on 15 litres of oxygen per minute. He was administered IV diamorphine 2.5mg OD. Investigations conducted revealed the following results:

ECG: normal sinus rhythm 96 bpm, ST elevation in leads II, III and aVF Chest x-ray: normal appearances of heart and lung

What is the best immediate management step?

A. Commence thrombolysis
B. Transfer for immediate percutaneous coronary intervention
C. Commence aspirin 300mg OD and clopidogrel 300mg OD
D. Conduct myocardial perfusion scan
E. Commence IV labetalol infusion

E. Commence IV labetalol infusion

This patient has had an aortic dissection involving the ascending aorta and arch of the aorta, as manifested by the presence of severe chest pain, aortic regurgitation, unequal blood pressures and evidence of an inferior myocardial infarction on ECG. In this situation, thrombolysis is absolutely contraindicated as the myocardial infarction is due to dissection of the vessels and not due to an atherosclerotic thrombotic occlusion. Options 2 and 3 are therefore also not suitable. This gentleman requires urgent intravenous beta blockade for aggressive blood pressure control and to act as a negative inotrope. He will also require an urgent cardiothoracic surgical opinion and further imaging to confirm the diagnosis.

Question 58 of 328

A 75-year-old female is admitted to hospital with community-acquired pneumonia. She is currently being treated with intravenous benzylpenicillin and oral clarithromycin. On the third day of admission, she complains of a sensation of fullness on the lateral aspect of her right leg.

Her past medical history includes osteoarthritis, temporal arteritis (now off steroids) and a uterine prolapse.

Bloods performed the day before show:

Hb	131 g/l
Platelets	520 * 10 /l9
WBC	14.2 * 10 /l9
Na +	141 mmol/l
K +	4.1 mmol/l
Urea	6.2 mmol/l
Creatinine	86 µmol/l
CRP	78 mg/L

A doppler ultrasound scan is performed that shows a superficial vein thrombosis in the right leg.

What is the most appropriate management?

A. Treatment dose low molecular weight heparin (LMWH) and warfarin until warfarin therapeutic then anticoagulate for three months
B. Prophylactic dose low molecular weight heparin (LMWH) for 30days.
C. Treatment dose low molecular weight heparin (LMWH) for 30days.
D. Non-steroidal anti-inflammatory drugs for 8-12 days.
E. Low molecular weight heparin (LMWH) and warfarin untilwarfarin therapeutic then anticoagulate for six months.

B. Prophylactic dose low molecular weight heparin (LMWH) for 30days.

Superficial vein thrombosis (also referred to as thrombophlebitis) is a common condition and in the majority of cases is self-limiting. Patients presenting with a superficial vein thrombosis are at increased risk of a venous thromboembolic event and up to 20% of patients will already have a deep vein thrombosis (DVT) on presentation. Patients are more likely to have thrombus extension into the deep venous system if they have more than 5cm of thrombus within 10cm of the saphenofemoral junction (i.e. within the proximal long saphenous vein).

The evidence for how superficial vein thrombosis should be managed comes from a Cochrane review and is used to support the Scottish intercollegiate guidelines network (SIGN) guidelines on the prevention and management of venous thromboembolism (guide 122). The guidelines recommend that all patients with clinical signs of a superficial vein thrombosis should have an ultrasound scan to exclude a DVT.

Patients with a confirmed diagnosis of a superficial vein thrombosis should be considered for treatment with anti-embolism stockings and prophylactic doses of low molecular weight heparin (LMWH) for 30 days or fondaparinux for 45 days. In cases where LMWH is contraindicated, 8- 12 days of oral non-steroidal anti-inflammatory drugs should be offered as this has been shown to reduce the risk of thrombus extension.

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A 76-year-old man presents to the emergency department with complaints of shortness of breath on exertion, difficulty walking up stairs, and significant weight gain over the last 2 weeks. His daughter who is with him expresses concerns about him and says that he is needing to lay upright in his armchair in order to get any sleep.

His medical history includes 2 previous non-ST-segment elevation myocardial infarctions for which he was treated with coronary stenting and hypertension. Physical examination shows a heart rate of 111 beats per minute and blood pressure of 113/76 mmHg. He is visibly short of breath at rest and is using accessory muscles. His JVP is 7cm above the angle of Louis, his apical impulse is laterally displaced, and heart sounds are audible with a holosystolic murmur heard loudest at the apex. Auscultation of his chest reveals audible crepitations bi-basally and he has 2+ peripheral oedema extending to his mid-thighs. An ECG shows Q-waves in his inferior leads, and his bloods show elevated troponins.

Which of the following is the most appropriate next step in the management of this patient?

A. Obtain an urgent echocardiogram
B. Prescribe oral furosemide and discharge from the emergencydepartment with a view to follow up with his GP
C. Load him with aspirin and clopidogrel
D. Commence bisoprolol to control his heart rate
E. Administer a bolus of intravenous furosemide and then start himon an infusion

E. Administer a bolus of intravenous furosemide and then start himon an infusion

The most appropriate medication in patients with acute heart failure and a preserved ejection fraction who have signs of volume overload is addition/up-titration of a loop diuretic

This patient presents with acute decompensated congestive heart failure. He has evidence of significant volume overload on exam, with an elevated JVP and signs of pulmonary and peripheral oedema. The aetiology of his heart failure is likely to be ischaemic cardiomyopathy related to his history of myocardial infarctions and manifests with a displaced apical impulse and a holosystolic murmur heard loudest at the apical region, suggestive of significant ventricular dilatation. His symptoms are severe and he requires inpatient hospitalisation and treatment with intravenous loop diuretics to improve his symptoms and to optimise his volume status.

An echocardiogram would be useful in the evaluation of this patient but is not the most appropriate next step as he is currently significantly symptomatic and needs intravenous loop diuretics to improve his volume status.

Although this patient does need to be treated with loop diuretics, he is in significant distress and likely needs to be admitted as an inpatient and should be treated more aggressively with intravenous loop diuretics.

This patient does have a history of ischaemic heart disease and is at risk of having further ischemic events. However, the findings on his ECG represent an old inferior infarct, and there a no features to suggest an acute event. In addition, he may have elevated troponins simply as a result of having ischaemic cardiomyopathy. He does not necessarily need to be loaded on dual antiplatelet therapy at this moment, but he should have his troponins trended to ensure there is no significant rise in their values.

Starting a beta-blocker in acute decompensated heart failure is contraindicated.

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An 89-year-old gentleman presented to the general medical clinic with complaints of palpitations. He was diagnosed with atrial fibrillation (AF) three months ago and started on bisoprolol. This was part of a plan to rate control, rather than rhythm control, his AF. The bisoprolol has been up-titrated as his symptoms did not come under control with his dose. Palpitations occur almost every day but are not associated with any other symptoms. He also has a history of ischaemic heart disease with a myocardial infarction five years ago and type 2 diabetes mellitus. He currently takes bisoprolol, metformin, aspirin and simvastatin. He has declined anticoagulation with warfarin or NOAC despite the risk as his wife died from an intracranial bleed whilst taking warfarin.

On examination, his heart rate is 94/min and irregular. His chest is clear on auscultation and there is no peripheral oedema.

How should he be further managed?

A. Amiodarone
B. Dronedarone
C. Amlodipine
D. Diltiazem
E. Left atrial ablation

D. Diltiazem

The correct answer is diltiazem. This is a gentleman with symptomatic, but not decompensating, permanent AF which is not responding to firstline treatment with a beta-blocker (bisoprolol). NICE advises that first line treatment for a rate-control strategy in AF should be either a betablocker or a rate-limiting calcium channel blocker, but digoxin can be considered for non-paroxysmal AF in patients who are not very active. If the first-line treatment fails, either with continuing symptoms or poor response of ventricular rate then a combination therapy with any of the following two can be used: beta-blocker, diltiazem, digoxin. Amiodarone, dronedarone and left atrial ablation are all strategies for cardioversion. Amlodipine is a calcium channel blocker but is used in hypertension rather than in AF as it is not rate-limiting, and would therefore not help.

Question 61 of 328

A 65-year-old Caucasian man attends the dermatology clinic with a 3- week history of a new rash. He complains of a discolouration of his forearms and hands that initially appeared whilst on holiday in the south of France. It is mildly itchy but not painful. There is no history of a change in diet or detergents and he has no known allergies. His past medical history includes hypertension, type 2 diabetes, ischaemic heart disease and atrial fibrillation.

On examination, there is a purplish discolouration of his hands up to his elbows bilaterally. His face and scalp are mildly erythematous. There is no blistering or crusting.

Which of the following is most likely to be responsible for this patient's presentation?

A. Aspirin
B. Digoxin
C. Indapamide
D. Metformin
E. Ramipril

C. Indapamide

Thiazides may cause photosensitivity

This patient has drug-induced photosensitivity, a skin reaction secondary to UV radiation causing expected burns or dermatitis in patients taking photosensitising medications. Drugs which cause phototoxicity include: antibiotics (tetracyclines, sulfonamides, fluoroquinolones), NSAIDs, diuretics, sulfonylureas, antipsychotics as well as amiodarone, quinine and hydroxychloroquine. The clinical features of photosensitivity can vary according to the medication taken as well as the type of reaction. Some reactions may be phototoxic whilst others are photoallergic.

A phototoxic reaction results from direct damage mediated by UV activation of the photosensitising agent. These reactions can appear within minutes to hours after exposure and appears as a sunburn reaction with reddening and swelling. Rarely, the skin may change pigmentation (e.g. blue-green discoloration seen with amiodarone). In severe reactions, blisters and vesicles may be seen. Other symptoms may include itch, as seen in this scenario. Thiazides typically cause a phototoxic reaction. In contrast, a photoallergic reaction is a cellmediated response. These are less common and present as an eczematous, itchy skin reaction within 24-72 hours after exposure to sunlight. Unlike phototoxic reactions, photoallergic reactions are capable of spreading to areas that have not been exposed to sunlight. The treatment of drug-induced photosensitivity is to avoid the trigger, if possible. If the medication is essential to be taken then protective measures should be taken including sunscreen or protective clothing.

In toxic doses, digoxin is capable of causing side effects including arrhythmias, dizziness, yellow or green visual changes and skin reactions. However, they do not commonly cause photosensitivity. If there are any concerns regarding digoxin toxicity, a digoxin level can be taken with the therapeutic window ranging between 0.7ng/mL and 2.0ng/mL.

Aspirin is a common cause of acute or chronic urticaria. However, it does not cause photosensitivity.

Metformin and ramipril do not commonly cause a rash or skin discolouration. In rare cases, patients may have an allergic reaction to these medications which results in the eruption of urticaria and, in extreme cases, lip and tongue swelling and airway compromise. However, they do not cause photosensitivity.

Question 62 of 328

An 89-year-old lady presents acutely short of breath and distressed. She has a background of previous myocardial infarction (MI) and hypertension. She is now coughing up white sputum. Examination reveals bilateral course crepitations throughout the lung fields. She has a raised jugular venous pressure and peripheral oedema. Observations are a respiratory rate of 35/min, oxygen saturations of 92% on 15 litres of oxygen per minute, blood pressure 135/90 mmHg, heart rate 100/min. Chest X-ray reveals widespread interstitial shadowing. Intravenous furosemide has been given but the patient fails to improve.

Which of the following would be useful in treating this patient?

A. Intravenous antibiotics
B. Oral furosemide
C. Bilevel positive airway pressure
D. Continuous positive airway pressure
E. Chest physiotherapy

D. Continuous positive airway pressure

Acute heart failure not responding to treatment - consider CPAP

The patient is suffering from severe pulmonary oedema with bilateral course crackles and cough productive of white sputum. The patient has signs of right sided heart failure with raised JVP and peripheral oedema. They also have a history of MI and hypertension that are two risk factors for heart failure.

NICE guidance on Acute Heart Failure 2014 states that a patient who has failed medical management of pulmonary oedema with severe dyspnoea should be considered for CPAP. BIPAP is not used in acute pulmonary oedema.

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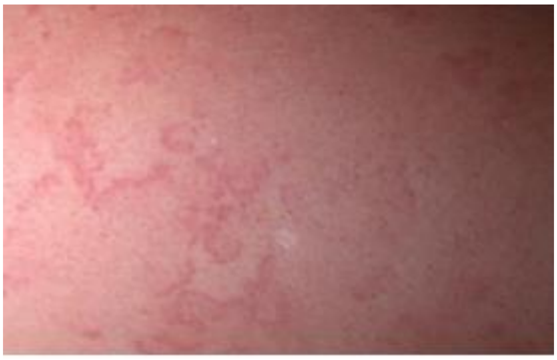
A 10-year-old girl is reviewed. Her mother describes her as being 'generally unwell'. For the past week she has been having joint pains and fever. Her mother also describes episodes of her making jerky, irregular movements. She recently went on holiday to the Lake District and had a severe sore throat around 4 weeks ago.

On examination her temperature is 37.9ºC, pulse 90/min, blood pressure 100/62 mmHg. Auscultation of the heart is unremarkable. A rash is noted on her torso:



What does the rash most likely represent?

A. Erythema marginatum
B. Erythema multiforme
C. Erythema chronicum migrans
D. Janeway lesions
E. Erythema toxicum

A. Erythema marginatum

The underlying diagnosis is rheumatic fever. This is supported by the recent sore throat, chorea (jerk, irregular movements) and polyarthralgia.

Erythema toxicum is a non-specific rash seen in newborns

Question 64 of 328

A 22-year-old man is brought to the emergency department after being granted asylum in the UK. Immigration officials had been concerned about the patient's well-being immediately on his arrival in the country. The patient reported feeling progressively more unwell over the previous 3 months after he had fled from his village in Syria. The primary symptoms reported by the patient were fevers, severe night sweats and profound fatigue. In addition, the patient had found that his exercise tolerance has greatly reduced in the previous four weeks and he became markedly breathless on minimal exertion.

The patient explained that he had experienced a prolonged period of inadequate food and shelter due to the war in his home country and while travelling across Europe. The patient had previously lived in a small village in Syria and had worked as a sheep farmer. Prior to fleeing his home the patient had regularly consumed food products made with unpasteurised sheep's milk. The patient had previously been in good physical health and had suffered no significant medical problems during his life.

On general examination, the patient appeared unwell and diaphoretic. The patient's sweat was noted to have an unusually unpleasant odour. Examination of the cardiovascular system identified multiple splinter haemorrhages under the patient's fingernails and toenails. Jugular venous pressure was elevated by 3-4 cm and pitting oedema was present to the patient's knees. A harsh systolic murmur was heard over the aortic area on auscultation. Bilateral basal crackles were heard on auscultation of the patient's lungs. The patient's spleen was palpable on examination of the abdominal system. No palpable lymphadenopathy was identified.

Please see the below table for results of initial investigations.

Haemoglobin	123 g / dL
Mean cell volume	80.3 fl
Lymphocytes	4.9 x 10>3 / microlitre (reference 1.5-4.0)
Platelets	109 x 10>3 / microlitre
Urea	8.9 mmol / L
Creatinine	126 micromols / L
Sodium	137 mmol / L
Potassium	4.1 mmol / L
Erythrocyte sedimentation rate	102 mm / h (reference 0-35)
C-reactive protein	99 mg / L (reference < 7)
Urinalysis	Positive for blood +++
Chest x-ray	Upper lobe blood diversion; generalised patchy airspace shadowing; bilateral shallow pleural effusions
ECG	Sinus rhythm; borderline right bundle branch block
Focused bedside transthoracic echocardiogram	0.6 cm vegetation on aortic valve; evidence of moderate-severe aortic stenosis; ejection fraction 30 %

Which of the following organisms is the most likely cause of the patient's presentation?

A. Clostridium burnetti
B. Bartonella quintana
C. Haemophilus parainfluenzae
D. Kingella kingae
E. Brucella melitensis

E. Brucella melitensis

The patient presents with clear symptoms, signs and investigation results consistent with aortic valve endocarditis and secondary congestive cardiac failure. It is likely that the patient's prolonged exposure to inadequate living conditions has weakened his immune system allowing the development of an opportunistic infection.

Of the available options, *Brucella melitensis* is the most likely to be the causative microorganism. Endocarditis - most commonly of the aortic valve - is a rare complication of Brucella; however, it is responsible for the majority of mortality from this disease. Of the different *Brucella* species known to cause endocarditis *Brucella melitensis* is known to be associated with the most severe complications.

Brucellosis is the most likely diagnosis in this patient due to his history of consumption of unpasteurised milk products and close contact with livestock from an endemic region. The clinical sign of malodorous sweat is considered to be nearly pathognomonic for Brucellosis. In addition, the patient's blood results are typical for the condition featuring lymphocytosis and thrombocytopenia.

The patient requires blood cultures and serological testing to confirm the diagnosis. Given the severity of the patient's condition, he would require treatment with empirical antibiotics with consideration of aortic valve replacement at an appropriate point in the future.

The other available answers are all organisms known to be rare causes of endocarditis that usually will not be identified without specialist microbiological investigations. There is no information in the case presentation that makes any of these microorganisms a more likely causative agent of endocarditis than *Brucella melitensis*.

Question 65 of 328

A 64-year-old hypertensive male presents to the emergency department with a history of sudden onset tearing chest pain. He is a chronic smoker with a 25 pack year history. He takes valsartan 160mg daily and amlodipine 5mg daily.

On examination, he is distressed and in severe pain. Blood pressure is 200/120 mmHg. The peripheral pulses are weak and his heart rate is 125 bpm.

Sublingual nitrates and oral aspirin are given to him in the emergency department and his twelve-lead ECG is performed which reveals tachycardia, left ventricular strain and deep S waves in lead V1-V3 and tall R waves in V4-V6.

Chest x-ray shows widening of the mediastinum with an irregular aortic contour.

Contrast-enhanced CT scan of the chest reveals an aortic intimal flap distal to left subclavian artery.

Which of the following is the most appropriate treatment option for this patient?

A. Blood pressure control with IV beta-blockers
B. Percutaneous stent insertion
C. Surgical repair

D. Blood pressure control with IV hydralazine
E. Continuous infusion of isosorbide dinitrate

A. Blood pressure control with IV beta-blockers

The diagnosis in this question is aortic dissection type B (distal to the left subclavian artery), the management of which is blood pressure control. The most appropriate drugs used are IV beta-blockers.

Aortic dissections can be classified according to the anatomic site of the dissection. Type A involves the aortic arch and the aortic valve while type B involves the descending thoracic aorta distal to the left subclavian artery. (Stanford Classification)

Stanford types A and B are further sub-classified according to the De Bakey classification.

Stanford Type A constitutes De Bakey I (extending to the abdominal aorta) and De Bakey II (localised to the ascending aorta). Stanford type B is synonymous with De Bakey type III.

The importance of these classifications is the difference in treatment. Universally, blood pressure control in mandatory with a target reduction in systolic BP to 120mmHg. Type A dissection should undergo surgery (aortic arch replacement) if the patient is fit enough since medical management alone carries a high risk of mortality (50% within 2 weeks). Type B dissections carry a better prognosis with 89% survival at one month and should thus initially be managed medically, with surgical interventions only if complications develop.

Indications for endovascular stenting include:

- Rapidly expanding dissections (>1cm per year)
- Critical diameter (>5.5cm)
- Refractory pain Malperfusion syndrome
- Blunt chest trauma
- Penetrating aortic ulcers

Question 66 of 328

A 32-year-old woman with pyrexia of unknown origin is admitted to the medical assessment unit.

Examination reveals a pale patient with a new cardiac murmur, evidence of splinter haemorrhages and splenomegaly. She looks unwell.

She has no past medical history, takes no regular medication and has no allergies.

The patient has high inflammatory markers and positive blood cultures which are growing Gram-positive cocci in clusters.

What is the single most appropriate antibiotic treatment?

A. IV amoxicillin
B. IV flucloxacillin
C. IV gentamicin
D. IV vancomycin
E. Oral doxycycline

B. IV flucloxacillin

IV flucloxacillin is the treatment of choice for native valve endocarditis caused by staphylococci

The most likely diagnosis here is infective endocarditis given the widespread clinical signs and the positive blood culture is most likely a *Staphylococcus aureus*, which is often described as Gram-positive cocci in clusters before full identification is completed.

The most appropriate antibiotic, therefore, is **IV flucloxacillin** which will adequately treat *S aureus*. This is the recommended treatment for any native valve staphylococcal endocarditis and is the correct answer in this case.

IV amoxicillin is incorrect. Although it is the recommended empirical antibiotic for endocarditis, it is incorrect to use if there is a known staphylococcal organism in the blood culture.

IV gentamicin is often added to amoxicillin in native valve endocarditis but it is unnecessary as the organism has been identified in a blood culture.

You may see a patient with a prosthetic valve where you would need to consider using **IV vancomycin** to cover an MRSA, however, in this case, the patient has no medical history so this is not correct.

Oral doxycycline would treat *S aureus* but is inappropriate as IV antibiotics are always required in infective endocarditis.

Question 67 of 328

A 55-year-old obese man with a history of hypertension, diabetes and angina develops central crushing chest pain whilst gardening alone. He is short of breath and clammy. He takes two puffs of his glyceryl trinitrate spray but has only mild relief from this.

He calls an ambulance, but before the ambulance arrives he suffers a cardiac arrest. The ambulance crew commence cardiopulmonary resuscitation (CPR). Heart monitoring shows him to be in ventricular tachycardia and after a DC shock and a further round of CPR, he recovers cardiac output.

He is taken to the local hospital intensive care unit where he remains unconscious. The doctors explain to his family that because of the time between his cardiac arrest and CPR commencing, his brain function may not recover well.

What is the most reliable indicator of this patient's prognosis at 72 hours post-arrest?

A. Electroencephalogram
B. Glasgow coma scale motor score
C. Pupillary reflex
D. Somatosensory evoked potential
E. Gag reflex

C. Pupillary reflex

Ability to predict poor outcome (death or severe disability) in a comatosed patient followings cardiac arrest is important for decision making regarding withdrawal of treatments as well as communicating with families.

No clinical neurological signs have been shown to correlate reliably with poor outcomes when measured less than 24 hours after cardiac arrest.

Work by Rittenberger and colleagues (2010) looked at patients who suffered ventricular tachycardia or ventricular fibrillation cardiac arrests. They found that GCS motor score did not correlate with cardiac arrest outcome when measured at 24 or 72 hours. However, lack of pupillary light or corneal reflex at 72 hours was a reliable predictor of death.

Vestibulo-ocular reflexes are also used but are less reliable. No biochemical, imaging or neurophysiological studies are currently recommended for the assessment of prognosis.

Question 68 of 328

A 74-year-old gentleman presents with oedema. He also states that his urine has been frothy over the last few weeks. He has no past medical history and is on no regular medications.

On examination he has pitting oedema up to the mid thighs. His JVP is 8cm from the sternal angle, and rises with inspiration. His pulse is irregularly irregular, and ECG shows fast atrial fibrillation with a rate of 145 bpm. He is otherwise systemically well with a BP of 135/85mm Hg. On neurological exam you note weakness and a sensory deficit in the distribution of the right ulnar nerve and left posterior tibial nerve.

You administer a loading dose of 1mg IV digoxin over 2 hours. Shortly after this the patient deteriorates and loses consciousness. A repeat ECG shows ventricular tachycardia, which then develops into ventricular fibrillation. Unfortunately the patient did not survive resuscitation attempts.

What is the most likely underlying disease?

A. Hypertrophic obstructive cardiomyopathy
B. AL amyloidosis
C. Brugada disease
D. AA amyloidosis
E. Cryoglobulinaemia

B. AL amyloidosis

Digoxin administration is not recommended in cardiac amyloidosis owing to a higher risk of digoxin toxicity, as the drug binds avidly to amyloid fibrils

The presence of Kussmauls sign (JVP which rises on inspiration) and significantly raised JVP is suggestive of constrictive pericarditis or restrictive cardiomyopathy. Together with the presence of nephrotic syndrome, and mononeuritis multiplex, one must consider amyloidosis.

AA amyloidosis occurs due to chronic inflammation e.g. rheumatoid arthritis. There is no evidence of a past medical history that would predispose to AA amyloidosis. Furthermore, cardiac manifestations are very rare in AA amyloidosis. This is in contrast to AL amyloidosis, which occurs due to light chain disease, where cardiac manifestations are very common.

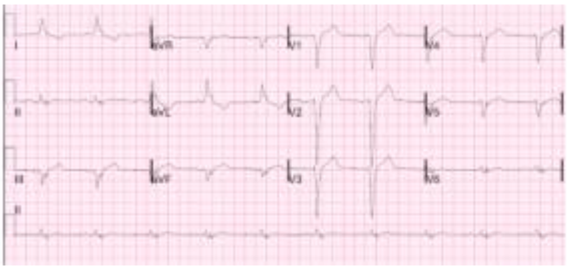
Digoxin administration is not recommended in cardiac amyloidosis owing to a higher risk of digoxin toxicity, as the drug binds avidly to amyloid fibrils. In this case, administration of digoxin has led to cardiac arrhythmia due to the enhanced effects of digoxin on an amyloid heart

Question 69 of 328

A 49-year-old man presents to the emergency department of a tertiary centre with a 4-hour history of central chest pain. There is no radiation of pain, but he reports to be feeling breathless and light-headed. Other than a recent viral upper respiratory tract infection, he has been well in himself recently. His past medical history includes hypertension, hyperlipidaemia and a post-operative deep vein thrombosis 2 years ago. Current medications include ramipril, amlodipine and atorvastatin and he denies any allergies.

On examination, he appears clammy to touch. His chest is clear on auscultation, although he is mildly tachypnoeic with saturations of 94%. Heart sounds are normal and his peripheral pulses are intact. He has a soft and non-tender abdomen.

An ECG is recorded, as shown below:



Given the likely diagnosis, what is the most appropriate next step in the management of this patient?

A. Discharge with NSAIDs
B. Fibrinolysis
C. Insulin and dextrose infusion
D. Percutaneous coronary intervention
E. Rivaroxaban

D. Percutaneous coronary intervention

This patient's ECG shows a left bundle branch block (LBBB) with broadened QRS complexes (> 120ms), a dominant S wave in V1 (creating 'W' impression) and broad monophasic R waves in leads I, aVL, V5 and V6 (creating 'M' impression). In patients with an ECG showing LBBB, it is important to establish whether or not this is a new finding as a new presentation of LBBB is concerning for myocardial ischaemia. In combination with a history of chest pain, shortness of breath, and being clammy to touch, it is likely that this patient is having a myocardial infarction. For patients presenting to the hospital within 12 hours of a STEMI with the availability of primary percutaneous coronary intervention (PCI) within 2 hours, PCI is the most appropriate and definitive management of this patient where the obstructed artery can be stented. As this patient has presented within 4 hours to a tertiary centre, it is likely that PCI can be achieved and is, therefore, the correct answer.

It would be inappropriate to discharge this patient with analgesia as a new finding of LBBB and a clinical history of chest pain is highly suggestive of myocardial ischaemia and needs urgent treatment.

Fibrinolysis is an alternative option for managing acute coronary syndrome. However, if PCI is available, it should be prioritised as the most appropriate treatment.

Insulin and dextrose are used in the management of hyperkalaemia. ECG changes in hyperkalaemia commonly include peaked T waves. However, in severe hyperkalaemia, ECG changes can include bundle branch blocks including LBBB. This is much rarer than myocardial ischaemia and given the history of chest pain, shortness of breath and sweating, an acute coronary syndrome is the more likely cause for which PCI is indicated.

Rivaroxaban is used in the treatment of a pulmonary embolism (PE). Although this patient has a higher risk of venous thromboembolism with a previous DVT, it is very unlikely for a PE to cause LBBB

Question 70 of 328

An 84-year-old gentleman is admitted to hospital with a short history of episodes of loss of consciousness. The patient has no clear recollection of the events, but his family informs you that they have not identified any specific pattern to them. Most of them have been unwitnessed, with the exception of the last one, where according to his wife he became pale, his eyes rolled backwards and fell on the chair. Some minor flickering was noted. Once he regained consciousness he was briefly confused and anxious, but soon realised where he was. Cardiovascular examination was unremarkable, with no postural hypotension but a blood pressure of 103/55 mmHg, and no focal neurology was isolated. His bloods were unremarkable.

His ECG was that of sinus rhythm: 67/min with Right Bundle Branch Block (RBBB). You see from a previous echocardiogram in his notes, that he has a degree of heart failure with an ejection fraction of 45%. A 72 hour holter monitor revealed no significant pauses.

Which action would you take next?

A. Arrange MRI head
B. Arrange EEG
C. CRT-D placement
D. Arrange insertion of Reveal Device
E. Arrange performance of Carotid sinus and Tilt-test

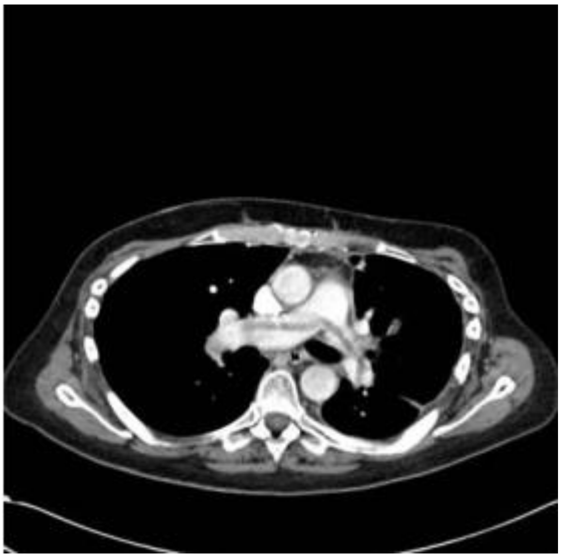
E. Arrange performance of Carotid sinus and Tilt-test

In patient with BBB and unexplained syncope but an ejection fraction >35% proceed with further testing (e.g. carotid sinus massage, electrophysiological studies) prior to initiation of management

As per the ESC Guidelines on Cardiac Pacing and Cardiac Resynchronization therapy (2013) it is important to exclude any other probable causes of collapse/syncope prior to installing any device. It is important to note that 'Less than half of the patients with BBB and syncope have a final diagnosis of cardiac syncope, albeit the probability is different among different type of BBB'. Given his symptomatology and the examination findings alongside the 72-hour holter monitor this is unlikely to be purely cardiogenic therefore further investigation is needed. With his age and presentation, autonomic dysfunction could be the cause, therefore carotid sinus and tilt-test may provide the answer. An MRI head and an EEG remain plausible options, but given the lack of solid seizure-like activity, can be arranged later on, provided that no answer is given by other tests.

Question 71 of 328

A 72-year-old man is admitted to hospital with shortness-of-breath. On examination his pulse is 96/min, BP 100/64 mmHg, respiratory rate 20/min, temperature 37.5ºC, oxygen saturations 96% on room air. A 12- lead ECG shows sinus rhythm, at a rate of 94/min with no diagnostic ST-T changes. The troponin I level is < 0.05 µg/L. A CT chest (with contrast) is ordered:



What is the most likely diagnosis?

A. Superior vena cava obstruction
B. Aortic dissection (Stanford type A)
C. Infective endocarditis
D. Aortic dissection (Stanford type B)
E. Pulmonary embolism

E. Pulmonary embolism

The CT shows a large saddle embolus where the pulmonary trunk splits to form the right and left pulmonary arteries.

Most image-based questions can be answered without looking at the image. If we therefore concentrate on the history a few clues point towards a pulmonary embolism (PE). The first is tachypnoea. Around 95% of patients with a PE have a respiratory rate > 16/min. Tachypnoea is not as common in any of the other diagnoses. A low-grade temperature is also an under appreciated sign of pulmonary embolism. This could of course be consistent with infective endocarditis but there are no other features to support this diagnosis.

Question 72 of 328

A 19-year-old woman is being reviewed in the cardiology clinic for recurrent syncopal attacks in the last 2 years. The attacks mainly occur during or after strenuous exercise.

Her vital signs are a temperature of 37 ºC, blood pressure 105/65 mmHg, pulse 83 beats/minute, and oxygen saturation 98% on room air. Cardiac examination is unremarkable. She has thickened skin over her palms and soles. An electrocardiogram (ECG) shows deep t-wave inversions in V1 to V3.

What is the most appropriate anti-arrhythmic drug for this patient?

A. Bisoprolol
B. Digoxin
C. Magnesium sulphate
D. Metoprolol
E. Sotalol

E. Sotalol

Arrhythmogenic right ventricular cardiomyopathy - T wave inversion in V1-3

Syncopal attacks precipitated by exercise and deep T-wave inversions from V1 to V3 point towards arrhythmogenic right ventricular cardiomyopathy (ARVC). ARVC, as described in this scenario, can occur with hereditary Naxos disease in which woolly hair and palmoplantar skin thickening can be seen. **Sotalol** is the most effective anti-arrhythmic drug for patients with ARVC. Sotalol is superior to amiodarone in preventing and treating ventricular arrhythmias in ARVC.

Bisoprolol is incorrect because the anti-arrhythmic properties of bisoprolol are not equivalent to sotalol in preventing arrhythmias in ARVC. Bisoprolol is a widely used rate-controlling agent and less frequently used as an anti-arrhythmic.

Digoxin is unlikely to benefit this patient with ARVC. Digoxin is used as an anti-arrhythmic in patients with concomitant heart failure.

Magnesium sulphate has no role in treating ARVC. It is an antiarrhythmic of choice in torsades de pointes.

The patient in this scenario has features similar to hypertrophic cardiomyopathy (HCM), in which **metoprolol** is the first-line drug of choice. The skin keratosis described in this scenario makes Naxos disease and ARVC more likely.

Question 73 of 328

A 72-year-old male presents with a five hour history of left sided chest pain, radiating to his left arm. He also complains of sweatiness in both his hands and has vomited three times in the last 2 hours. ECG demonstrates ST depression in II, III and aVF, troponin is 0.78 (normal range <0.03). On examination, he is alert but extremely clammy. You note his pulse at the radial artery is 35/ minute and regular. His BP on the cardiac monitor is 120/67. An NSTEMI is diagnosed and the patient is taken to the catheter laboratory for reperfusion therapy.

Which coronary artery is most likely to be occluded?

A. Left anterior descending artery
B. Left circumflex artery
C. Obtuse marginal branch artery 1
D. Obtuse marginal branch artery 2
E. Right coronary artery

E. Right coronary artery

The sino-atrial node (SAN) is supplied by the right coronary artery in 60- 80% of patients, with the left circumflex supplying the remainder of the population. The OM1 and OM2 arteries are branches of the left circumflex artery, classically supplying the apex of the heart.

Question 74 of 328

A 17-year-old man from Romania is seen on the acute medical take. He is usually fit and well but for the past week has had a sore throat and fevers. He was unsure of his vaccination status and a throat swab taken from pustular exudate on his tonsils grew *Corynebacterium diphtheriae* for which he has started erythromycin. His maternal grandfather is known to have glucose-6-phosphate deficiency but he has never received testing.

He presents today with one day of shortness of breath. He is experiencing fevers and having difficulty swallowing. On examination, he is tachypnoeic with a respiratory rate of 34 breaths per minute. He had bibasal crepitations and a JVP can be seen at 4cm above the angle of Louis. He is coughing white frothy material and saturating at 90%.

Na +	137 mmol/l
K +	4.2 mmol/l
Urea	6.4 mmol/l
Creatinine	60 µmol/l
Troponin	29,000 ng/ml (normal range <20)
Hb	110 g/l
Platelets	300 * 10 /l9
WBC	9.6 * 10 /l9
Neutrophils	4.3 * 10 /l9
Lymphocytes	8.2 * 10 /l9
Throat swab	Corynebacterium diphtheriae resistant to erythromycin, sensitive to penicillin

What complication has arisen?

A. Acute haemolytic crisis
B. Myocarditis
C. Diphtheria pneumonitis
D. Interstitial lung disease
E. Lemierre's Syndrome

B. Myocarditis

Myocarditis can be a secondary complication of diphtheria

The picture shows a large troponin rise with the rapid onset of acute heart failure. Myocarditis is a rare recognised complication of diphtheria and is due to exotoxin produced. The organisms resistance to the macrolide antibiotic has undoubtedly allowed it to persist despite medical therapy. Diphtheria pneumonitis, Lemierre's syndrome and interstitial lung disease could all conceivably present with bibasal crepitations but no troponin rise. An acute haemolytic crisis would usually have a very low haemoglobin and signs of jaundice that are not present here.

Question 75 of 328

A 23-year-old gentleman presents with sudden shortness of breath, lethargy and fevers. He is currently homeless and has been brought in by a friend. He has vomited three times since arrival but has no diarrhoea.

He is usually well and has a metal plate in his right wrist for a fracture sustained in an assault a few weeks prior. He does not take any regular medications and is not registered with a local GP. He denies any alcohol or substance use.

He is currently taking rifampicin and isoniazid for pulmonary tuberculosis diagnosed 4 months ago and is compliant with his directly observed therapy.

On examination, he has a pan-systolic murmur over the left sternal edge. He has bilateral lung crepitations to the midzones with dullness to percussion at the base. His heart rate is 96/min, temperature is 37.8ºC and he is saturating at 94% with 4 litres oxygen by nasal cannulae. He has a bounding JVP that reaches the ear lobe with cannon V-waves seen. Tracking marks can be seen in the arms and there are injection sites in his groin. He has poor dentition with tenderness under the left jaw. His right wrist is tender over the plate and there is no redness and a mildly reduced range of motion.

Hb	96 g/l	Na	139 mmol/l
Platelets	156 * 10 /l	K	4.2 mmol/l
WBC	14.2 * 10 /l	Urea	5.6 mmol/l
Neuts	12.0 * 10 /l	Creatinine	96 µmol/l
CRP	156 mg/l		

Chest x-ray	bilateral alveolar shadowing, small right sided effusion
ECG	T wave inversion in V1-V4
Blood cultures	Staph aureus

What is the underlying cause of this presentation?

A. Poor dental hygiene
B. Intravenous drug use
C. Infected orthopaedic plate
D. Tuberculosis
E. Prolonged vomiting

B. Intravenous drug use

Tricuspid valve endocarditis can cause tricuspid regurgitation, which may manifest with a new pan-systolic murmur, large V waves and features of pulmonary emboli

This gentleman has infective endocarditis. The cannon V waves and pan systolic murmur narrow this down to the tricuspid valve being affected which is very common in intravenous drug users as this is the first valve reached from venous blood. The tracking marks are a clue even though the patient denies this. Tuberculosis does not cause murmurs and usually only affects the pericardium of the heart. Poor dental hygiene can cause a *strep* endocarditis that is less florid and more insidious. An infected orthopaedic plate is a rare cause of endocarditis. Prolonged vomiting would not cause this. The lack of pitting oedema suggests this is a rapid onset right sided heart failure.

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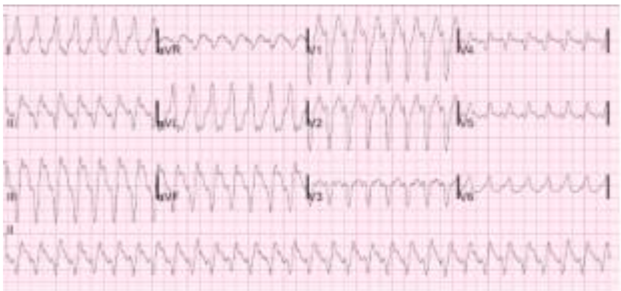
A 68-year-old man attends the emergency department with palpitations and dizziness. This had a sudden onset approximately one hour ago. He denies chest pain. His observations are as follows: heart rate 170 beats per minute, BP 100/70 mmHg, respiratory rate 18 breaths per minute, and temperature 37.2 ºC.

An ECG is performed in the department:



What is the most likely diagnosis?

A. Atrial fibrillation with aberrant conduction
B. Monomorphic ventricular tachycardia (VT)
C. Polymorphic ventricular tachycardia (VT)
D. Supraventricular tachycardia with aberrant conduction
E. Ventricular flutter

B. Monomorphic ventricular tachycardia (VT)

The ECG demonstrates a broad-complex tachycardia. It can be difficult to distinguish between ventricular tachycardia and supraventricular tachycardia with aberrant conduction (e.g. LBBB). Therefore it is always safest to assume that the arrhythmia is ventricular in origin until proven otherwise. Electrocardiographic features that increase the likelihood of VT include:

- Absence of typical RBBB or LBBB morphology
- Extreme axis deviation ('northwest axis'): QRS positive in aVR and negative in I and aVF
- Very broad complexes > 160ms
- AV dissociation Capture beats Fusion beats
- Positive concordance throughout the precordial leads
- Negative concordance throughout the precordial leads
- RSR' complexes with an R>R'

Monomorphic ventricular tachycardia is the correct option. The ECG demonstrates RSR' complexes with an R>R' which is the most specific finding in favour of VT. Since the morphology of the QRS waves is similar beat to beat then the diagnosis is monomorphic VT rather than polymorphic VT.

Atrial fibrillation with aberrant conduction is incorrect. The presence of P waves (seen best in lead II) and the regular rhythm make this an unlikely diagnosis.

Polymorphic ventricular tachycardia is incorrect. This would also present with a broad complex tachycardia with similar criteria as discussed above. However, in this condition, the morphology of the ventricular complex would vary from beat to beat.

Supraventricular tachycardia with aberrant conduction is incorrect. Although this is a possibility, the extremely broad QRS duration and the presence of RSR' complexes with R>R' favour the diagnosis of monomorphic ventricular tachycardia.

Ventricular flutter is incorrect. This condition is an extreme form of ventricular tachycardia (VT) with loss of organised electrical activity. The ECG would demonstrate a continuous sine wave with no identifiable P waves, QRS complexes, or T waves.

Question 77 of 328

A 65-year-old with exertional dyspnoea over the past 3 months is referred to the pulmonary hypertension team. An initial echocardiogram demonstrated 65% ejection fraction, preserved left ventricular function with a pulmonary arterial pressure of 72 mmHg. She undergoes a right and left heart catheter, revealing the following saturations:

Right atrium high	60%
Right atrium mid	89%

Right atrium low	70%
Right ventricle high	70%
Right ventricle mid	73%
Right ventricle low	72%
Pulmonary artery	71%
Capillary wedge	96%

What is the most likely diagnosis?

A. Aortic stenosis
B. Atrial septal primum defect
C. Atrial septal secundum defect
D. Pulmonary stenosis
E. Mitral stenosis

C. Atrial septal secundum defect

The key to questions regarding saturations and cardiac catheters is to spot the 'step-up' in oxygen saturation. It is present between the high and mid right atrium, demonstrating the presence of a left-to-right shunt. Primum ASD defects typically occur lowering in the septum than secundum defects, which typically can be found in the mid-atrial region.

Question 78 of 328

A 30-year-old man presents with a fever. He complains that he has had a fever, headache and lethargy for three weeks. On examination, his temperature is 38.5C. You notice a healed human bite mark on his right forearm, he tells you he was bitten in a pub brawl about a couple months ago. Diastolic and systolic murmurs are heard on auscultation. A transoesophageal echo demonstrates an oscillating mass on a bicuspid aortic valve, with aortic regurgitation. Three blood cultures are taken before administration of empirical antibiotics, two grow small colonies of tiny pleomorphic gram-negative bacilli.

What is the likely causative organism?

A. Eikenella corrodens
B. Escherichia coli
C. Phoenicoparrus andinus
D. Staphylococcus aureus
E. Streptococcus viridans

A. Eikenella corrodens

This patient fits the Duke criteria for infective endocarditis as there is: echocardiographic evidence of an intra-cardiac mass associated with a valve, a fever of >38 and two blood cultures of an endocarditis causing microorganism. Additionally, this patients bicuspid aortic valve is a risk factor for developing endocarditis.

Eikenella corrodens is a gram-negative bacilli, which is a commensal of the human mouth. It is a member of the HACEK group, an acronym for a group of organisms that can cause gram-negative endocarditis (*Haemophilus* species, *Actinobacillus actinomycetemcomitans*, *Cardiobacterium hominis*, *Eikenella corrodens*, and *Kingella* species.) The human bite injury and gram-negative culture make *Eikenella corrodens* the most likely causative organism.

Streptococcus viridans and *Staphylococcus aureus* are commoner causes of infective endocarditis, which are easily cultured and are gram-positive.

Escherichia coli could cause endocarditis and is a gram-negative bacilli, but is not the most likely in this case.

Phoenicoparrus andinus is the binomial nomenclature for the rare Andean Flamingo, which are not known to cause infective endocarditis.

Question 79 of 328

A 30-year-old pregnant woman (24 weeks) is admitted to the acute medical unit with sudden onset shortness of breath and pleuritic chest pain. She has no past medical history other than eczema and this is her first pregnancy.

On examination, auscultation of her chest reveals only a mild wheeze with oxygen saturations of 94% on room air and a respiratory rate of 25/min. Her heart sounds are normal, with a heart rate of 97bpm and blood pressure is 105/60 mmHg. An ECG shows sinus rhythm.

The FY1 doctor on the ward has requested a D-dimer to try and 'speed up the diagnosis', which comes back positive.

What is the next appropriate investigation?

A. Ventilation-Perfusion (V/Q) scan
B. Chest x-ray
C. Peak flow measurement
D. CT Pulmonary Angiogram (CTPA)
E. Ultrasound doppler of the lower legs

B. Chest x-ray

It is likely that this woman has had a pulmonary embolism given the acute nature of the shortness of breath and pleuritic chest pain, together with tachycardia and reduced oxygen saturations.

The correct way to investigate a pregnant woman with a possible pulmonary embolism is to first perform a chest x-ray. This is vital to ensure other diagnoses such as pneumonia and pneumothorax are identified

Ultrasound doppler of the lower legs is the next step as this can demonstrate a venous thromboembolism of the lower limb vasculature without radiation. If a thrombus is found, appropriate management with low molecular weight heparin can be initiated.

If the chest x-ray and the ultrasound doppler are normal, a ventilationperfusion scan should be performed. The presence of a normal chest xray can essentially function as the ventilation part of the V/Q scan. This allows for a half-dose radiation V/Q scan.

The radiation dose to the foetus of a V/Q scan is slightly higher than that of a CTPA, however both are well below the threshold of specific harm to the foetus. Please note that a CTPA should only be used in the presence of lung disease including asthma or an abnormal chest x-ray, as the radiation dose to the proliferating maternal breasts may be significant in the development of carcinogenesis later in life.

A d-dimer is not appropriate as it may be raised in pregnancy and may lead to further inappropriate tests based on the positive result alone.

Question 80 of 328

A 23-year-old Afro-Caribbean female presents with a four-month history of fatigue, transient fever lasting hours, 12kg weight loss and nonspecific bilateral headaches. She has presented to the Emergency Department four times in the past 3 months with non-specific abdominal pains that are worse after eating and also blue-lighted into her local hyperacute stroke unit as a thrombolysis patient after sudden onset loss of monocular blindness in her left eye, which resolved before any treatment was given. She has no other past medical history, does not smoke and drinks minimally.

On examination, cranial nerve examination and fundoscopy are both unremarkable. Upper and lower limb neurology are intact with downgoing plantars. Her heart sounds demonstrates a gallop rhythm, an early diastolic murmur and a mild radial-radial delay. Chest auscultation is clear. She appears very warm and measures 38.4ºC in your clinic with facial flushing. Blood tests are as follows:

Hb	94 g/l
Platelets	245 * 10 /l
WBC	18.4 * 10 /l
Eosinophil	0.1 * 10 /l
ESR	121 mm/hr
Na	141 mmol/l
K	4.0 mmol/l
Urea	5.2 mmol/l
Creatinine	68 µmol/l
CRP	56 mg/l

Complement levels were reported as normal and an antibody screen including ANCA and ANA was negative. Urine dip is negative. Chest Xray demonstrates focal consolidation. Her heart rate is 95 and regular, her blood pressure is 185/110 mmHg.

What is the unifying diagnosis?

A. Systemic lupus erythematosus
B. Polyarteritis nodosa
C. Poorly controlled primary hypertension
D. Ascending aortic aneurysm
E. Takayasu arteritis

E. Takayasu arteritis

A systemic condition is clearly present in this patient: there is a combination of fever, weight loss, pyrexia, ischaemic abdominal pain, transiently ischaemic cerebral occlusion, raised inflammatory markers and hypertension associated with radial-radial delay. The key differentials are between PAN and Takayasu arteries, which are medium-sized and large vessel vasculitis respectively. Ischaemic colitis is a common presentation in PAN but almost always associated with one of positive ANCA, eosinophilia, peripheral neuropathy and hepatitis B. In addition, the involvement of temporal arteries and it appears the ascending aorta to produce radial-radial delay suggests Takayasu arteritis to be the most likely diagnosis. Immediate CT or MRI imaging is necessary with acute immunosuppression with high-dose steroids, followed by maintenance immunosuppression with azathioprine, methotrexate or cyclophosphamide.

Question 81 of 328

A 73-year-old man was admitted as an emergency suffering from severe, central and crushing chest pain. ECG revealed evidence of anterior ST elevation MI and primary percutaneous coronary intervention was performed with the deployment of two drug-eluting stents to the left anterior descending artery. The patient had no previous coronary artery disease or significant family history. He is an ex-smoker and drinks approximately 10 units of alcohol per week.

Post-procedure the patient was well and mobilised on the ward without further chest pain or shortness of breath. Clinical examination was unremarkable.

The patient was commenced on aspirin, clopidogrel, ramipril, bisoprolol and atorvastatin therapy. Further investigations performed following intervention are summarised as below.

Haemoglobin	13.9 d / dL
White cell count	8.6 * 10 /l
Platelets	199 * 10 /l
Urea	7.2 mmol / L
Creatinine	110 micromol / L
Sodium	137 mmol / L
Potassium	4.3 mmol / L
Total cholesterol	5.4 mmol / L
LDL cholesterol	3.1 mmol / L
HDL cholesterol	1.2 mmol / L
Triglycerides	2.5 mmol / L

Transthoracic echocardiogram: no valvular abnormality; mild anterior dyskinesia with overall normal left ventricular systolic function; ejection fraction 55-60 %

Primary PCI report: total occlusion of mid-LAD, two DES deployed with good angiographic result; 50 % mid-vessel occlusion of the right coronary artery; circumflex unobstructed.

Prior to discharge, the patient asks to speak to you about any other possible strategies to reduce his risk of suffering a further heart attack.

Which of the following interventions is recommended for this patient by NICE guidance on secondary prevention of myocardial infarction?

A. Percutaneous coronary intervention to right coronary artery
B. Mediterranean-style diet
C. Abstinence from alcohol
D. Omega-3 fatty acid supplements
E. Aldosterone antagonist (for example, eplerenone)

B. Mediterranean-style diet

NICE recommends several lifestyle changes after a myocardial infarction. These include smoking cessation, a Mediterranean-style diet, achievement of a healthy weight, limitation of alcohol consumption to recommended healthy limits and 20-30 minutes of physical activity daily. NICE does not recommend routinely eating oily fish or the consumption of omega-3 supplements, although it is noted that there is no evidence of harm from these interventions.

Aldosterone antagonists are recommended for use in patients with symptoms and signs of heart failure or with evidence of systolic impairment on echocardiography. There is no evidence that percutaneous treatment of mild asymptomatic coronary artery disease (such as in this patient) reduces the future risk of myocardial infarction.

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A 69-year-old man with a background of ischaemic heart disease attends his routine cardiology appointment. He reports a 2-month history of progressively worsening shortness of breath and is only able to walk 100 yards before needing to stop for rest. He has occasional exertional chest pain which resolves with sublingual glyceryl trinitrate. He is currently taking aspirin, atorvastatin, lansoprazole, bisoprolol, ramipril, spironolactone and sublingual glyceryl trinitrate as required.

On examination, his heart rate is 80bpm and regular with a blood pressure of 130/75mmHg. His chest is clear with good air entry bilaterally and saturations of 94% on room air. He has a soft and nontender abdomen with no palpable organomegaly. There is pitting oedema to his knees.

ECG: Sinus rhythm, Q waves and T wave inversion in V1-V4, QRS duration 110ms.

Echocardiogram: Left ventricular ejection fraction 33%, pulmonary arterial pressure 20mmHg.

What is the most appropriate step in the management of this patient?

A. Cardiac resynchronisation therapy
B. Digoxin
C. Furosemide
D. Ivabradine
E. Nicorandil

D. Ivabradine

Ivabradine should be considered in heart failure if the patient has sinus rhythm > 75/min and a LVEF < 35% and have not responded to to ACE-inhibitor, beta-blocker and aldosterone antagonist therapy

Ivabradine is a 3rd line agent in the treatment of heart failure. It works by selectively inhibiting the 'funny current' I*f*, also known as the pacemaker current found in the sinoatrial node. The drug acts to lower the heart rate and therefore the oxygen demand needed to supply to the myocardium. Criteria for prescribing ivabradine include a heart rate > 75bpm and an ejection fraction of < 35%.

Cardiac resynchronisation therapy (CRT) is another 3rd line treatment agent for heart failure. It is indicated for patients with left ventricular dysfunction and consequently a reduced LV stroke volume which is complicated by a dyssynchronous ventricular contraction secondary to a conduction delay (usually LBBB). It involves inserting electrodes into the walls of the ventricle to coordinate their contraction and ensure a synchronised contraction of the left ventricle to increase the ejection fraction. An indication for its use is a reduced ejection fraction (< 35%) and widened QRS complexes (>120ms) on the ECG.

Digoxin can be used as a 3rd line agent in the treatment of heart failure. It is strongly indicated in patients with co-existing atrial fibrillation.

Furosemide can provide symptomatic relief from fluid overload in heart failure. However, it does not improve mortality and is therefore not the next recommended treatment option.

Nicorandil is a potassium channel agonist that causes vasodilatation and is therefore used in the treatment of stable angina. Due to the effects of vasodilation on blood pressure and ejection fraction, its use is contraindicated in those with left ventricular dysfunction.

Question 83 of 328

A 29-year-old man is admitted to the cardiology ward following a collapse whilst playing football. He has no recollection of events other than running on the pitch and waking up in the ambulance. In the last 2- 3 months, he has been complaining of intermittent palpitations but put this down to work-related stress. There is no history of chest pain and he has no past medical history of note.

His observations are recorded within normal limits. On auscultation, chest sounds are clear and heart sounds are normal. He has a soft and non-tender abdomen and his neurological examination is unremarkable.

A bedside echocardiogram demonstrates a hypokinetic right ventricle.

Given the likely diagnosis, what is the most characteristic finding on this patient's ECG?

A. Left ventricular hypertrophy
B. Positive deflection at the J point
C. Right bundle brach block
D. Slurred upstroke in QRS
E. Small positive deflection at the end of QRS complex

E. Small positive deflection at the end of QRS complex

The most characteristic ECG finding in arrhythmogenic right ventricular dysplasia (ARVD) is the epsilon wave (a small positive deflection at the end of the QRS complex)

This patient has arrhythmogenic right ventricular dysplasia (ARVD), a type of inherited cardiovascular disease. ARVD can present as palpitations, syncope, or sudden cardiac death similar to hypertrophic obstructive cardiomyopathy (HOCM). Over time, the right ventricular myocardium is replaced by fibrofatty tissue and patients develop signs of right ventricular failure. This patient's echocardiogram finding of a hypokinetic right ventricular wall is suggestive of the diagnosis. The most characteristic ECG finding in ARVD is an **epsilon wave**. An epsilon wave is a positive deflection following the end of the QRS complex caused by post-excitation of right ventricular myocytes.

Left ventricular hypertrophy is a common ECG finding of HOCM. HOCM is the most common cause of sudden cardiac death and can also present with palpitations and syncope, as seen in this patient. However, this patient's echocardiogram findings are in keeping with ARVD, of which the epsilon wave is the most characteristic ECG finding.

An **Osborn wave (or J wave) is a positive deflection at the J point**. There are multiple causes, of which the most common is hypothermia. Other causes of J waves include left ventricular hypertrophy and Brugada syndrome. However, an Osborn wave is not associated with ARVD.

There are multiple causes of **right bundle branch block**, including right ventricular hypertrophy. In ARVD, rather than hypertrophy of the right ventricle, the myocardium is replaced by fatty tissue. Right bundle branch block is not a sign of ARVD. Instead, the left bundle branch block is more likely to feature on an ECG of a patient with ARVD.

A **delta wave** is a slurred upstroke in the QRS complex that is commonly associated with pre-excitatory activity, such as that seen in WolffParkinson White syndrome. It is not associated with ARVD.

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A medical opinion was sought from the obstetrics team regarding a 38- year-old 28 weeks pregnant lady. A routine blood pressure check revealed a blood pressure of 158/98 mmHg. Other than suffering from hyperemesis gravidarum, her pregnancy had proceeded without complication. She specifically denied the presence of headaches, vomiting, change in vision, abdominal pain, seizures or bleeding per vagina. She had noticed no change in the frequency of foetal movements, and her 20-week antenatal scan revealed the presence of a healthy foetus with a rate of growth within the expected range. Her past medical history was unremarkable; she was a non-smoker and did not consume alcohol. Her blood pressure at the booking antenatal appointment was 148/88 mmHg. Her sister suffered from pre-eclampsia during her pregnancy necessitating delivery by caesarean section.

Examination revealed the presence of a well 28-week old pregnant lady. Her blood pressure was indeed 158/98 mmHg, her heart rate was 86 bpm and temperature 36.5ºC. Examination of the cardiovascular system revealed normal heart sounds, a JVP of 3cm and the absence of pedal oedema. Examination of the respiratory system was unremarkable. Examination of the gastrointestinal system revealed the presence of a symphysiofundal height appropriate for the stage of gestation with easily obtainable foetal heart sounds on hand held Doppler examination. Examination of the neurological system was unremarkable with normal reflexes, cranial nerve function and peripheral motor and sensory function. Urinalysis revealed no abnormality.

What is the next best management step?

A. Commence ramipril
B. Commence labetalol
C. Commence indapamide
D. Commence magnesium sulphate
E. Transfer to high dependency unit to observe for signs of preeclampsia

B. Commence labetalol

This lady has pre-existing hypertension; her blood pressure at the antenatal booking clinic was elevated and continues to be elevated throughout (as opposed to gestational hypertension in which hypertension develops after 20 weeks). If untreated hypertension is associated with adverse maternal and foetal outcomes including intrauterine growth restriction, placental abruption, cerebrovascular accidents and prematurity. There are no clinical features of preeclampsia, including notably the presence of proteinuria and peripheral oedema, and there is, therefore, no indication for admission to a high dependency unit or to commence magnesium sulphate. Of the remaining options, labetalol is the safest antihypertensive to use in pregnancy; methyldopa is an alternative. The usual first line ACE inhibitors are absolutely contraindicated in pregnancy.

Question 85 of 328

A 38-year-old female of Asian descent, with no significant past medical history, presents after a syncopal event while pruning hedges. She has had a 6-month history of fever, arthralgia and for the past few weeks has had multiple episodes of vertigo and one syncopal event. She denies headaches or visual complaints. Her examination reveals a diminished radial pulse in the left arm and a systolic blood pressure difference in the upper extremities of 14 mmHg. A bruit is auscultated along the left upper extremity. Dopplers of the upper extremities indicate a stenotic area along the subclavian that is later confirmed by Magnetic Resonance Angiography (MRA). She is diagnosed with subclavian steal syndrome. Laboratory tests reveal a normocytic normochromic anaemia, elevated CRP and ESR, negative ANA and ANCA, and all other laboratory tests are within normal range.

Of the following, What is the most likely diagnosis?

A. Fibromuscular dysplasia
B. Ehlers-Danlos syndrome
C. Takayasu arteritis
D. Giant cell arteritis
E. Wegener's granulomatosis

C. Takayasu arteritis

This patient likely has Takayasu arteritis which is essentially a chronic vasculitis primarily of the aorta and its branches. These patients can present in a variety of different ways depending on the vessels affected, however, they all typically have a prodrome of systemic symptoms including fatigue, weight loss and low-grade fevers prior to developing any vascular complaints. From a pathology standpoint biopsies of vessels are very similar to giant cell and are typically not performed.

There are 6 criteria for the diagnosis of Takayasu. Presence of 3 of the 6 has 90% sensitivity and specificity for diagnosis:

1. Age onset <=40 years

2. Claudication of the extremities

3. Decreased pulsation of one or both brachial arteries

4. Difference of at least 10 mm Hg in systolic blood pressure

between the arms

5. Bruit over one or both subclavian arteries or the abdominal aorta

6. Arteriographic narrowing or occlusion of the entire aorta, its primary branches, or large arteries in the proximal upper or lower extremities, not due to arteriosclerosis, fibromuscular dysplasia, or other causes

Fibromuscular dysplasia typically affects the renal arteries leading to renal artery stenosis and hypertension, and it not accompanied by other systemic manifestations like fever and malaise. Ehlers-Danlos syndrome is a genetic defect in type III collagen and can lead to aneurysms along with hyperelasticity of the skin and hypermobile joints, but other systemic manifestations are typically not present. Giant cell arteritis is most similar to Takayasu in pathology, however typically affects older patients and usually presents with headaches and tenderness over the temporal artery. Lastly, Wegener's is actually a small vessel vasculitis and its most common presenting symptoms include persistent rhinorrhoea, purulent/bloody nasal discharge, oral and/or nasal ulcers, polyarthralgias, myalgias, or sinus pain. Most with Wegener's are ANCA positive.

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A 72-year-old woman with a 30 year history of type 2 diabetes mellitus comes for review. She was diagnosed with chronic kidney disease (secondary to diabetes) 8 years ago and has seen declining renal function since. Her current medication includes ramipril 10mg od, amlodipine 10mg od, simvastatin 40mg on and Novomix 30 insulin bd.

Her most recent renal function tests show the following:

Na +	139 mmol/l
K +	5.3 mmol/l
Urea	10.2 mmol/l
Creatinine	123 µmol/l
eGFR	40 ml/min/1.73m²

Blood pressure in clinic is 156/88 mmHg and this is confirmed on a second reading.

What should be done regarding her blood pressure medication?

- A. Add bisoprolol
- B. Add indapamide
- C. Add doxazosin
- D. Add spironolactone
- E. Add an angiotensin II receptor blocker

B. Add indapamide

Poorly controlled hypertension, already taking an ACE inhibitor and a calcium channel blocker - add a thiazide-like diuretic

Tight blood pressure remains a key management aim in patients with diabetic nephropathy. ACE inhibitors are clearly the most evidence based management in this arena but her blood pressure is persistently high. If we look at the NICE guidelines the next step would be the addition of a thiazide based diuretic (e.g. indapamide) and there is no reason not to follow these recommendations in this situation.

If the eGFR is less than 30 ml/min/1.73m² then thiazides should be avoided as the BNF states:

Thiazides and related diuretics are ineffective if eGFR is less than 30 mL/minute/1.73 m2 and should be avoided; metolazone remains effective but with a risk of excessive diuresis

Spironolactone and angiotensin II receptor blockers may risk precipitating hyperkalaemia.

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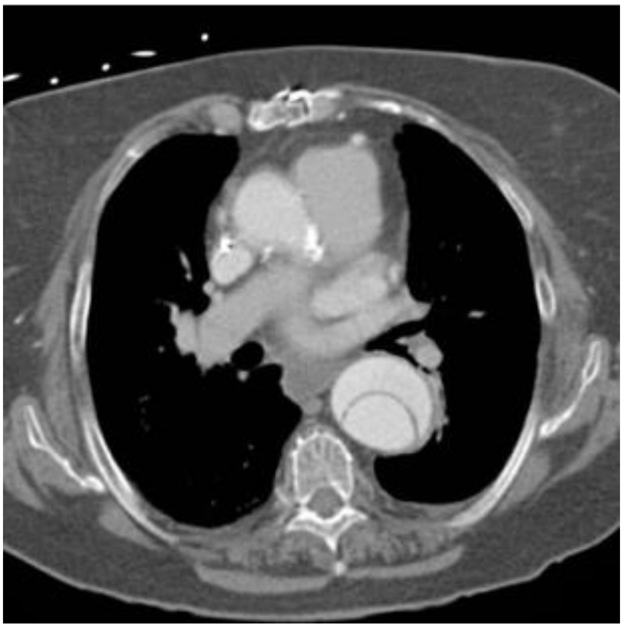
A 74-year-old man with a history of depression, chronic obstructive pulmonary disease and hypertension is admitted to hospital with severe central chest pain. He has no history of ischaemic heart disease or similar chest pains in the past. On admission blood pressure is 160/98 mmHg, pulse 110/min, respiratory rate 18/min and oxygen saturations are 93% on room air. A CT chest (with contrast) is shown below:



What is the most appropriate management?

- A. Chest drain
- B. Cardiothoracic surgery
- C. Intravenous labetalol
- D. Thrombolysis with tenecteplase
- E. Low-molecular weight heparin

C. Intravenous labetalol

Dissection of the descending aorta can be seen. As this is a Stanford type B dissection it is treated conservatively with intravenous beta-blockers to lower blood pressure and help prevent any extension.

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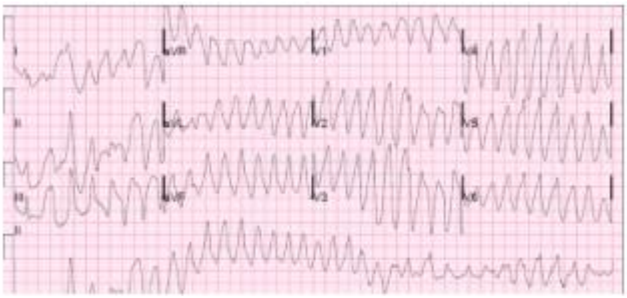
A 44-year-old woman presents to the emergency department with dyspnoea. She has been feeling intermittently dizzy and short-of-breath for the past 2 weeks. On examination her pulse is 180/min, blood pressure 100/66 mmHg, oxygen saturations 98% on room air. Her chest is clear and she appears well perfused. An ECG is obtained:



What is the most appropriate treatment? ,

- A. Intravenous amiodarone
- B. Intravenous adenosine
- C. Intravenous magnesium
- D. Intravenous labetalol
- E. Unsynchronised DC shock

C. Intravenous magnesium

IV magnesium sulfate is used to treat torsades de pointes

The ECG shows an irregular wide-complex tachycardia, a form of polymorphic ventricular tachycardia or more specifically torsades de pointes. This patient had an underlying long QT interval secondary to a combination of medications. The acute treatment for this is intravenous magnesium.

Precipitating medications should, of course, be reviewed and electrolyte abnormalities corrected.

If the patient was in shock or periarrest then the ALS tachycardia should be followed, i.e. SYNCHRONISED DC shocks. There are, however, no 'adverse' signs in this patient.

The ALS periarrest guidelines state the following:

Treat torsade de pointes VT immediately by stopping all drugs known to prolong the QT interval. Do not give amiodarone for definite torsade de pointes. Correct electrolyte abnormalities, especially hypokalaemia. Give magnesium sulfate 2 g IV over 10 min (= 8 mmol, 4 mL of 50% magnesium sulfate).

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A 67-year-old woman is complaining of a six-hour history of chest pain and dizziness in the high-dependency unit. She underwent a mitral valve replacement five days ago and had some temporary trans-venous pacing wires removed earlier today.

On examination, she has oxygen saturations of 93% on 2 litres via nasal cannula, has a heart rate of 110/min, a blood pressure of 76/43mmHg, has a temperature of 37.9ºC, and is responsive to voice. She feels cool peripherally. An ECG is performed at the bedside which shows sinus rhythm with QRS complexes of alternating amplitude.

Blood tests from this morning show the following:

Hb	89 g/L	(115 - 160)
Platelets	121 * 10 /L 9	(150 - 400)
WBC	13.1 * 10 /L 9	(4.0 - 11.0)
Na +	143 mmol/L	(135 - 145)
K +	3.1 mmol/L	(3.5 - 5.0)
Urea	18.2 mmol/L	(2.0 - 7.0)
Creatinine	151 µmol/L	(55 - 120)
CRP	282 mg/L	(< 5)
Magnesium	0.83 mmol/L	(0.85 - 1.10)

What is the most appropriate management given the likely diagnosis?

A. Electrolyte replacement
B. Emergency percutaneous coronary intervention
C. Pericardiocentesis
D. Prolonged course of antibiotics
E. Urgent temporary pacing wire insertion

C. Pericardiocentesis

Electrical alternans is suggestive of cardiac tamponade

This woman has clinical signs of cardiac tamponade which is confirmed by electrical alternans on ECG. Electrical alternans occurs due to the movement or 'swinging' of the heart within the pericardium as a result of the fluid present. This results in a variable amplitude of the QRS complex depending on the hearts proximity to the ECG leads. In this case, this was likely precipitated by a traumatic removal of trans-venous pacing wires causing bleeding into the pericardial space. As a result, she requires an urgent echocardiogram to confirm the diagnosis and subsequent **pericardiocentesis** or re-sternotomy to drain the blood. It is worth noting that her acute kidney injury is the result of poor cardiac output as a result of her tamponade.

Although there are electrolyte abnormalities on this patient's blood tests that should be replaced, this is not likely to be the cause of her acute presentation. An arrhythmia post-operatively could cause a reduction in cardiac output however the ECG performed here demonstrates sinus rhythm and therefore **electrolyte replacement** would not reverse her current condition.

An acute coronary syndrome must be considered in cardiac postoperative patients complaining of chest pain however the electrical alternans on ECG with no remarkable ST changes to speak of is more consistent with a diagnosis of cardiac tamponade. Therefore, **emergency percutaneous coronary intervention** is not the most appropriate option here.

A **prolonged course of antibiotics** may be employed if a diagnosis of infective endocarditis is suspected however this is not the case here for a variety of reasons. Firstly, infective endocarditis post-operatively is unlikely to present within 5 days. Furthermore, the electrical alternans on ECG is indicative of cardiac tamponade rather than infective endocarditis. Finally, although the patient has a fever and a raised CRP, these are common findings post-operatively due to the systemic inflammatory response to surgery and are not specific for infective endocarditis.

Arrhythmias are common in the post-operative period for patients who have undergone cardiac surgery, particularly valve surgery, however, this patient is in sinus rhythm at an acceptable rate. Therefore, as this patient's pathology is mechanical rather than electrical, **urgent temporary pacing wire insertion** will not help with her current issue.

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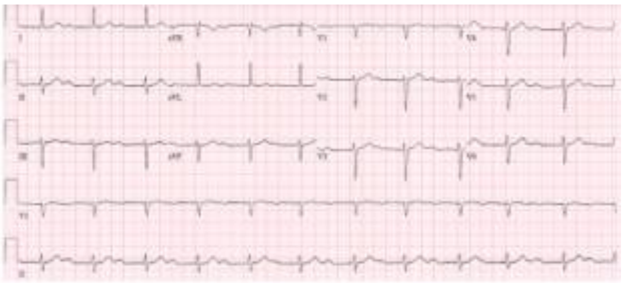
A 58-year-old man is referred to the cardiology clinic for a review. He complains of a 4-week history of intermittent palpitations and chest discomfort for approximately 1-2 minutes. He works as a stockbroker and has been recently promoted. There is no past medical history of note and he is an active tennis player on the weekends. He takes no regular medications and drinks 1 bottle of wine per week.

On examination, he has a heart rate of 65 beats/min and is regular with a blood pressure of 131/68mmHg. He has normal heart sounds and equal air entry on auscultation. His calves are soft with no pitting oedema.

An ECG is taken, as shown below:



What is the recommended management for this patient?

A. Catheter ablation of accessory pathway
B. Oral aspirin
C. Oral bisoprolol
D. Reassurance and discharge
E. Referral for pacemaker insertion

D. Reassurance and discharge

Reassurance and discharge is correct. This patient's ECG shows firstdegree heart block with a prolonged PR interval. First-degree heart block is not of any clinical concern and does not warrant further management. Taking a closer look at this patient's presentation, it seems that his chest discomfort and palpitations have coincided with a new job promotion and could be related to stress or anxiety. This history is atypical for cardiac ischaemia and there are minimal risk factors present to suggest that this patient's presentation was related to sinister cardiac pathology.

Catheter ablation of accessory pathway is incorrect. An accessory pathway (e.g. Bundle of Kent) is capable of creating a re-entry circuit that predisposes the patient to tachyarrhythmias. An example of a re-entry tachycardia associated with an accessory pathway is Wolff-ParkinsonWhite (WPW) syndrome. ECG features of WPW include PR-interval shortening, QRS prolongation and the classic 'delta wave' (the slow, slurred uprising of the QRS complex). These features are not seen on this patient's ECG. Catheter ablation is also used in the treatment of atrial fibrillation, but this patient has a regular rhythm with visible p waves, making atrial fibrillation very unlikely.

Oral aspirin is incorrect. This is used in the management of ischaemic heart disease that appears as the presence of Q waves, ST-segment elevation or depression or T wave inversion. None of these symptoms are seen, making ischaemic heart disease unlikely and, therefore, the use of aspirin incorrect.

Oral bisoprolol is incorrect. This is used in the management of ectopic burden and tachyarrhythmias. However, it is not used in the management of first-degree heart block.

Referral for pacemaker insertion is incorrect. Indications for pacemaker insertion include complete heart block, second-degree mobitz type 2 heart block, ventricular pauses >3 seconds and previous asystole. This patient has first-degree heart block which is not an indication to be referred for permanent pacemaker insertion.

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An 83-year-old male presents with ischaemic sounding chest pain that has persisted for the past one hour. A 12-lead ECG is performed and shows deep T wave inversion in leads V1 and V2.

Which is the most likely implicated coronary artery?

A. Left circumflex artery
B. Left main stem artery
C. Proximal left anterior descending artery
D. Right coronary artery
E. Distal left anterior descending artery

C. Proximal left anterior descending artery

Ischaemic changes in leads V1-V4 - left anterior descending

Wellens' syndrome is an ECG manifestation of critical proximal left anterior descending (LAD) coronary artery stenosis in patients with unstable angina. It is characterized by symmetrical, often deep (>2 mm), T wave inversions in the anterior precordial leads.

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A 22-year-old woman presents to the emergency department with a 1- day history of abdominal pain and vomiting. She denies fever or diarrhoea and disclosed that she went out to a party the night before and consumed 7 glasses of wine. She reports that there is a heart condition that runs in her family, but she cannot remember the name of the condition.

Her observations are within normal limits. On examination, there is central and epigastric tenderness but no guarding or peritonism. Her respiratory and cardiovascular examinations were unremarkable.

Venous blood gas:

pH	7.35	(7.35 - 7.45)
Na +	138 mmol/L	(135 - 145)
K +	4.1 mmol/L	(3.5 - 5.0)
Bicarbonate	22 mmol/L	(22 - 29)
Lactate	3.5 mmol/L	(< 1.6)
Glucose	6.2 mmol/L	(4 - 8)

ECG: Sinus rhythm, normal axis, QRS duration 110ms, QTc 550ms.

What drug is it most important to avoid?

A. Codeine
B. Cyclizine
C. Hyoscine butylbromide
D. Omeprazole
E. Ondansetron

E. Ondansetron

5HT-3 receptor antagonists shouldn't be used as antiemetics in patients with long-QT syndrome

This patient has a prolonged QTc interval of 550ms. A normal QTc interval for women should lie between 350-460ms. Patients with a prolonged QTc interval are at particular risk of developing critical arrhythmias such as Torsades de pointes (a polymorphic ventricular tachycardia). It is therefore extremely important to avoid drugs that may prolong the QTc interval further. **Ondansetron** is a type of 5HT-3 receptor antagonist that is associated with QTc interval prolongation and should therefore be avoided.

Codeine is a weak opioid analgesic with common side effects of nausea, drowsiness and constipation. It does not prolong the QTc interval.

Cyclizine is a H1-receptor antagonist. Side effects of cyclizine include urinary retention, dry mouth, and blurred vision. Prolongation of the QTc interval is not a common feature of this medication.

Hyoscine butylbromide is used in the treatment of gastric spasm. It is an anticholinergic agent and is, therefore, contraindicated in myasthenia gravis, urinary retention and gastrointestinal obstruction. It does not prolong the QTc interval.

Omeprazole is a proton-pump inhibitor used in the treatment of gastritis and gastro-oesophageal bleeding. Complications include electrolyte disturbances (e.g. hypomagnesaemia) and increased risk of *Clostridium dificile* infections but not prolongation of the QTc interval.

Question 93 of 328

A 64-year-old man presents to his general practitioner (GP) with several months of chest pain that occurs only on exertion. When he pauses to rest, the pain dissipates within a few minutes. He has a past medical history of high cholesterol, for which he takes atorvastatin. The GP commences him on bisoprolol.

Several weeks later, the patient re-attends, stating that the symptoms are still not under control.

What should be added next?

A. Amlodipine
B. Isosorbide mononitrate
C. Ivabradine
D. Nicorandil
E. Verapamil

A. Amlodipine

If angina is not controlled with a beta-blocker, a longer-acting dihydropyridine calcium channel blocker should be added

This patient is experiencing stable angina - the chest pain is occurring on exertion, but not at rest. NICE recommend the use of a beta-blocker or calcium channel blocker (CCB) first-line. As this patient has been commenced on bisoprolol already, the next step is to add a CCB. A dihydropyridine CCB should be used, to avoid reducing the heart rate further, and so **amlodipine** is the most appropriate option here.

Isosorbide mononitrate would be appropriate to use as a third agent, if the combination of a beta-blocker and CCB was not adequate. However, at this stage, a CCB should be added.

Similarly, **ivabradine**, which modulates the 'funny current', would be appropriate as a third agent instead of isosorbide mononitrate. A CCB should be added first, as this patient is only on bisoprolol.

Nicorandil is another alternative agent to be added third, instead of isosorbide mononitrate or ivabradine. It would not be appropriate to add currently, as the patient is only on bisoprolol.

Verapamil is a non-dihydropyridine CCB. As such, it is negatively chronotropic and so should not be used alongside a beta-blocker; this may precipitate complete heart block. It would have been suitable as a first-line alternative to the beta-blocker.

Question 94 of 328

A 64-year-old man presents to his general practitioner 4 weeks after a permanent pacemaker (PPM) insertion. This was inserted for symptomatic first-degree heart block. He reports that since this was inserted, he has actually felt worse and his exercise tolerance has become more limited.

An ECG is performed and shows a paced rhythm on VVI at 70/min. Each pacing spike is followed by a QRS complex and there are no pacing spikes in between beats. He has regular p waves at a rate of 35/min which are not related to the QRS complexes.

What is the most appropriate intervention?

A. Convert to a dual chamber pacemaker
B. Increase the pacing output
C. Increase the pacing sensitivity
D. Reduce the pacing output
E. Reduce the pacing sensitivity

A. Convert to a dual chamber pacemaker

A VVI pacemaker will pace and sense the right ventricle. In the presence or organised atrial activity, a VVI pacemaker may pace the ventricles out of sync with the atria resulting in pacemaker syndrome.

This man currently has single-chamber pacing to the ventricles as identified by his mode (VVI). Although this guarantees a ventricular rate, this is at the expense of atrioventricular concordance. For some people, cardiac output may actually fall because of this reduced diastolic function (known as pacemaker syndrome). Hence the worsening of this man's symptoms. One way to correct this would be to **convert to a dual-chamber pacemaker** and establish DDD pacing. This would pace both the atria and ventricles and ensure atrioventricular concordance. Atrial pacing would be possible given that the patient has p waves and therefore is not in atrial fibrillation.

Increasing the pacing output is a useful intervention when there is insufficient capture (the pacing spikes are not at a sufficient voltage to elicit a corresponding QRS complex). As this man's ECG shows good capture, this is not the most appropriate intervention.

An **increase of pacing sensitivity** increases the voltage required to inhibit the pacemaker. This would be a helpful intervention if the pacemaker was being inappropriately inhibited (potentially by noise). As the pacemaker is currently delivering regular pacing spikes at an appropriate rate, over-inhibition is not the cause of this man's symptoms and so this intervention would not be appropriate.

A **reduction in the pacing output** can be helpful to preserve the pacemaker's battery life and therefore the lowest output required to safely and reliably achieve capture should be used at all times, however this will not help with this patient's complaint.

Reducing the pacing sensitivity reduces the voltage required to inhibit the pacemaker. This could be employed if the patient's native rhythm was faster than the rate of the pacemaker but was still not inhibiting the pacemaker from firing. This is particularly important to avoid R on T pacing. As the pacing spikes here are immediately followed by QRS complexes, this man is reliant on his pacemaker's rate and there is no evidence that native QRS complexes are not able to inhibit the pacemaker. Therefore this intervention is not indicated.

Question 95 of 328

A 58-year-old female reports having symptomatic episodes of palpitations for the last six months. She is normally fit and drinks the occasional small glass of red wine when she goes out for a meal. The last episode was four days ago when she was cooking in her kitchen. She describes that she felt light headed with central chest discomfort that eased after twenty minutes. In clinic, her observations are all stable and an initial twelve lead electrocardiogram shows sinus rhythm.

What would be the investigation of choice?

A. 24 hour ambulatory electrocardiogram
B. Electrocardiogram treadmill test
C. 24 hour blood pressure
D. 72 hour ambulatory electrocardiogram
E. Event recorder electrocardiogram

E. Event recorder electrocardiogram

Atrial fibrillation is a common arrhythmia that may be persistent or paroxysmal. It is more common in women compared to men.

All patients with suspected atrial fibrillation should have a manual pulse check followed by a twelve-lead electrocardiogram. Those who have episodes less than 24 hours apart should have a 24-hour ambulatory electrocardiogram. In patients who experience episodes more than 24 hours apart, an event recorder electrocardiogram would be the most suitable investigation of choice.

24-hour blood pressure monitoring would be considered in a patient with suspected hypertension.

Electrocardiogram treadmill test is useful for revealing abnormal heart rhythms induced by exercise and ischaemic changes suggestive of coronary heart disease.

A 72-hour ambulatory electrocardiogram is used in much the same way as a 24 and 48-hour test. However, current NICE guidelines suggest for atrial fibrillation of episodes more than 24 hours apart an event recorder device is most useful.

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A 24-year-old man presents to the emergency department with one hour of palpitations and dizziness. He has no significant past medical history and is on no regular medications. He works as an accountant. There is no history of illicit drug use.

His observations are heart rate 180 beats per minute, blood pressure 88/59 mmHg, respiratory rate 22/minute, oxygen saturations 96% on air and temperature 36.4ºC.

Clinical examination is unremarkable except for a regular bounding tachycardia.

An ECG demonstrates ventricular tachycardia with positive concordance throughout the chest leads and similar morphology throughout.

As he is haemodynamically unstable, emergency electrical cardioversion is undertaken in the emergency department resuscitation area, which is successful.

Post cardioversion, his observations are heart rate 84 beats per minute, blood pressure 120/84 mmHg, respiratory rate 18/minute, oxygen saturations 96% on air and temperature 36.6ºC.

A repeat ECG demonstrates sinus rhythm and T wave inversion in leads V1-V3 with no other significant abnormalities.

Blood tests:

Hb	137 g/L	Male: (135-180)Female: (115 - 160)
Platelets	188 * 10 /L	(150 - 400)
WBC	7.2 * 10 /L	(4.0 - 11.0)
Na	141 mmol/L	(135 - 145)
K	4.4 mmol/L	(3.5 - 5.0)
Urea	5.6 mmol/L	(2.0 - 7.0)
Creatinine	89 µmol/L	(55 - 120)
CRP	4 mg/L	(< 5)
Adjusted calcium	2.41 mmol/L	(2.2 - 2.6)
Magnesium	0.92 mmol/L	(0.85 - 1.10)
Troponin	17 ng/L	(<14)

A CT pulmonary angiogram excludes a pulmonary embolism.

What is the likely diagnosis?

A. Arrhythmogenic right ventricular cardiomyopathy
B. Brugada syndrome
C. Catecholaminergic Polymorphic Ventricular Tachycardia
D. Right ventricular outflow tract ventricular tachycardia
E. Romano-Ward syndrome

A. Arrhythmogenic right ventricular cardiomyopathy

Arrhythmogenic right ventricular cardiomyopathy - T wave inversion in V1-3

Arrhythmogenic right ventricular cardiomyopathy (AVRC) is the correct answer. AVRC is a condition inherited with an autosomal dominant pattern, which is characterised by replacement of the normal right ventricular myocardium with fibrofatty tissue. It typically presents in the 2nd-4th decade of life with palpitations, syncope or sudden cardiac death and is a cause of monomorphic ventricular tachycardia. The resting ECG is typically abnormal and may demonstrate T wave inversion in V1- V3. The mildly elevated troponin in this case is simply secondary to ventricular tachycardia. Imaging will demonstrate structural and functional abnormalities of the right ventricle.

Brugada syndrome is incorrect. This is also an inherited cause of sudden cardiac death (autosomal dominant) but it traditionally causes a polymorphic ventricular tachycardia and the resting ECG is characterized by 'coved' ST segment elevation >2mm in >1 of V1-V3 followed by a negative T wave.

Catecholaminergic Polymorphic Ventricular Tachycardia is incorrect. This is an inherited cause of arrhythmia and sudden cardiac death characterized by palpitations, syncope of cardiac arrest precipitated by exercise or emotions. It is typically associated with polymorphic ventricular tachycardia as suggested by the name. Usually, the resting ECG is normal.

Right ventricular outflow tract ventricular tachycardia (RVOT-VT) is incorrect. Idiopathic RVOT-VT is not an inherited condition and the resting ECG is normal.

Romano-Ward syndrome is incorrect. This is a cause of congenital long QT syndrome, which can cause a polymorphic ventricular tachycardia. We are told there are no abnormalities on the resting

ECG apart from T wave inversion. Therefore, we can assume the QT interval is normal.

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An 82-year-old man presents with loss of consciousness. His wife states that he complained of feeling dizzy, before going pale in colour and falling to the ground. He was unconscious for approximately one minute. On regaining consciousness he was not confused and recovered within minutes. He denies any chest pain, shortness of breath or palpitations. He states that over the past few months he has had several episodes of light headedness however he has never lost consciousness before.

A 7 day holter was performed:

Heart rate	3 episodes of bradycardia of 20-30 bpm which correlated withepisodes of pre-syncope
Other events	Absence of P waves for 3.5 seconds

How will you manage this patient?

A. VVI pacemaker
B. Biventricular pacemaker
C. AAIR pacemaker
D. Reassurance
E. Bisoprolol

C. AAIR pacemaker

In pure sinus node dysfunction without AF or evidence of AV block, an AAIR or DDDR pacemaker can be used. Most cardiologists would choose a DDDR pacemaker since many of these patients go onto develop AV block

The 7 day holter confirms symptomatic sinus bradycardia and an episode of sinus arrest (absence of P wave for > 3 seconds). This confirms the diagnosis of sick sinus syndrome Sick sinus syndrome can be difficult to manage, as it can present with bradyarrhythmias, tachyarrhythmias, or a mixture of both. Bradyarrhythmias can be well controlled with pacemakers, while tachyarrhythmias respond well to rate limiting drugs such as calcium channel blockers, digoxin and beta blockers. However drugs to control tachyarrhythmia may exacerbate bradyarrhythmias. Therefore, a pacemaker is often implanted before drug therapy is begun for the tachyarrhythmia.

This patient has been having symptomatic bradycardia episodes secondary to sinoatrial node disease. Therefore an AAIR pacemaker is indicated. AAI pacemakers both sense and pace the atria. They are useful for isolated sinoatrial node disease. These are not suitable in patients with atrial fibrillation.

VVI pacemakers will both sense and pace the ventricle. VVI or VVR pacemakers are useful for pure sustained slow atrial fibrillation.

Biventricular pacemakers are used in heart failure with left bundle branch block.

Bisoprolol is a rate limiting drug which would worsen the bradycardia and the patients symptoms.

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A 45-year-old male patient presents with a worsening shortness of breath to the medical assessment unit. His has an accompanying letter from his GP stating that his past medical history is significant only for a deteriorating renal function that is secondary to polycystic kidney disease.

On examination there is no evidence of fluid overload, with the lung fields remaining clear and the JVP not elevated. You do however discern a late systolic murmur heard best at the cardiac apex which is immediately preceded by a 'click'.

Observations

- heart rate 82bpm
- blood pressure 106/86mmHg
- Respiratory rate 16 per minute
- temperature 36.5
- oxygen saturation 97% on room air

What is the most likely diagnosis?

A. Mitral valve prolapse
B. Mitral stenosis
C. Pulmonary stenosis
D. Mitral regurgitation
E. Aortic stenosis

A. Mitral valve prolapse

Adult polycystic kidney disease can be associated with extra-renal complications. Up to 85% of patients with autosomal dominant polycystic kidney disease (ADPKD) will exhibit liver cysts by the age of 30. Other complications include intra-cranial aneurysm and heart valve disease. Affected heart valves can occur in up to 25% of ADPKD patients and of them the majority have mitral valve prolapse. The majority of patients with mitral valve prolapse with be asymptomatic but when symptoms are present mitral valve prolapse has to be considered and investigated with an echocardiogram.

The 'click' coupled with the knowledge of the underlying ADPKD makes the murmur heard in this patient that of a mitral valve prolapse. The prolapse can worsen and cause a secondary mitral regurgitation leading to symptoms such as shortness of breath. The narrow pulse pressure present also suggests an element of left ventricular dysfunction

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A 64-year-old caucasian male presented to heart failure clinic with shortness of breath on exertion. He has a history of heart failure, initially diagnosed three years previously.

His current medications included ramipril, aspirin, bisoprolol, simvastatin and spironolactone. He is concerned that despite all of these medications his breathlessness is worsening. In addition to this he is now using four pillows to sleep at night.

On examination he is comfortable at rest with fine crackles at both lung bases and pitting oedema reaching up to both knees. His observations reveal a respiratory rate of 16 breaths per minute, oxygen saturation 96% in room air, blood pressure 110/85 mmHg, heart rate 70 beats per minute, temperature 37.2ºC.

His ECG revealed a normal sinus rhythm with narrow QRS complexes. A recent echocardiogram completed two weeks previously demonstrated an ejection fraction of 30%. His current medications were reviewed and he was found to be on the maximum dose for each of these.

His baseline U&E results are as follows:

Na +	136 mmol/L	(135 - 145)
K +	4.5 mmol/L	(3.5 - 5.0)
Bicarbonate	26 mmol/L	(22 - 29)
Urea	6.7 mmol/L	(2.0 - 7.0)
Creatinine	110 µmol/L	(55 - 120)

What is the optimum treatment option for this patient?

A. Cardiac Resynchronisation Therapy
B. Digoxin
C. Hydralazine combined with a nitrate
D. Ivabradine
E. Sacubitril-Valsartan (after stopping ACE inhibitor)

E. Sacubitril-Valsartan (after stopping ACE inhibitor)

Sacubitril-valsartan is considered in heart failure patients with a LVEF < 35% who are still symptomatic on ACE-inhibitors & betablockers

This patient has heart failure, he is symptomatic despite maximum medical therapy with an ACE inhibitor, beta blocker and aldosterone antagonist. His heart rate is 70 bpm, therefore ivabradine cannot be used at this time. After a period of ACE inhibitor washout sacubitril-valsartan is the optimal therapeutic option.

Digoxin has been found to not have any improvement in long-term outcome and therefore is not the correct answer in this case.

Hydralazine with a nitrate is indicated in the afro-caribbean population; we are told this patient is caucasian, hence this answer is incorrect.

Ivabradine is contraindicated as the patients heart rate if 70 bpm, the guidelines state it should be > 75 bpm for ivabradine to be prescribed.

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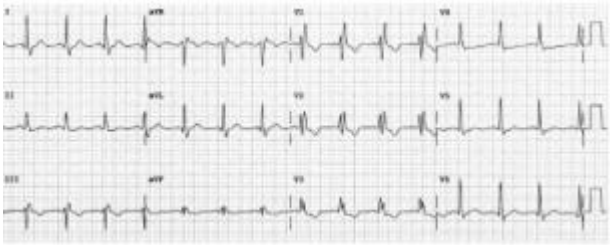
A 50-year-old man is brought into the emergency department following a collapse whilst gardening. He denies any presyncopal symptoms but now describes shortness of breath. His past medical history includes prostate cancer and hypertension.

On examination, there is good air entry bilaterally. His peripheral pulses are present bilaterally with a capillary refill time of < 2 seconds. There is no postural drop in blood pressure and his neurological examination is unremarkable.

An ECG is taken, as shown below:



What is the most likely cause of this patient's presentation?

A. Aortic stenosis
B. Hypercalcaemia
C. Hypokalaemia
D. Left ventricular hypertrophy
E. Pulmonary embolism

E. Pulmonary embolism

This patient's ECG demonstrates right bundle branch block (RBBB) with an RSR pattern in leads V1-V3 (creating an 'M-shaped' QRS complex), with wide slurred S waves in the leads I, aVL, V5-V6 (creating a 'Wshaped' QRS complex). A widened QRS interval (i.e. > 120ms) is also a feature of RBBB. There are multiple causes of RBBB and in some cases, it may be found incidentally as a normal variant. However, pathological causes commonly include increased right heart strain (e.g. right ventricular hypertrophy, cor pulmonale or pulmonary embolism). Given the history of malignancy, collapse and shortness of breath, a pulmonary embolism is the most likely cause of this patient's presentation to the hospital and ECG findings of RBBB.

Whilst aortic stenosis can cause the symptoms of collapse and breathlessness, it is not common for aortic stenosis to cause RBBB. Rather, aortic stenosis may be responsible for the appearance of left bundle branch block (LBBB) or left ventricular hypertrophy on an ECG.

The most common ECG finding in hypercalcaemia is QT interval shortening. In severe hypercalcaemia (> 3.4mmol/L) J waves may be seen. Although hypercalcaemia is more likely to be present in a patient with a diagnosis of malignancy, it would not be responsible for this patient's RBBB and is unlikely to be the underlying diagnosis.

Hypokalaemia causes the following ECG changes: prolonged PR interval, widespread ST depression and T wave flattening or inversion and prominent U waves (most clearly in leads V2-V3). It does not cause RBBB and is, therefore, incorrect.

Left ventricular hypertrophy (LVH) has numerous causes, most commonly hypertension. There are lots of specific voltage criteria to be met to accurately diagnose LVH on an ECG including:

- R wave in V4, V5 or V6 > 26mm
- R wave in V5 or V6 plus S wave in V1 > 35 mm
- Largest R wave plus largest S wave in precordial leads > 45 mm

Additional features include left axis deviation and left atrial enlargement. Although this patient has hypertension, there are no features of LVH on his ECG and it is unlikely to cause his episode of syncope, making it the incorrect answer.

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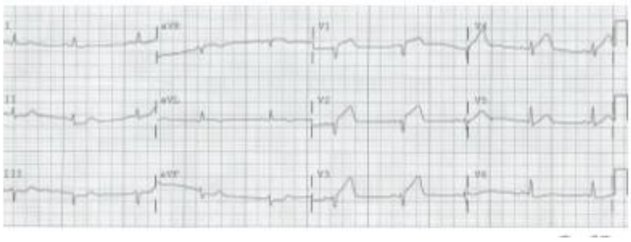
You review a 60-year-old man in resus who has been admitted with a 30-minute history of cardiac-sounding chest pain. His ECG is shown below:



Where is the lesion most likely to be?

A. Right coronary
B. Left main stem
C. Posterior intraventricular artery
D. Left anterior descending
E. Left circumflex

D. Left anterior descending

Ischaemic changes in leads V1-V4 - left anterior descending

ST elevation can be seen in V2-V4 which is highly suggestive of a left anterior descending lesion. A left main stem occlusion would result in more widespread changes.

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A 53-year-old woman has been admitted to the hospital with progressively worsening dyspnoea. She denies a cough but describes orthopnoea and the feeling of palpitations.

Observations are shown below:

Heart rate	112 bpm
Blood pressure	86/42 mmHg
Respiratory rate	28 /min
Oxygen saturations	99%
Temperature	36.8 ºC

On examination, she is overweight and appears dyspnoeic. Her airway is self-maintained and her chest appears clear on auscultations. Heart sounds are difficult to auscultate, her JVP is raised and her capillary refill time is normal. There is no evidence of pedal oedema.

Given the likely diagnosis, what might you see on the ECG?

A. Deep lateral Q waves with left ventricular hypertrophy
B. Electrical alternans
C. Global ST elevation
D. S1 Q3 T3
E. Trifascicular block

B. Electrical alternans

Electrical alternans is suggestive of cardiac tamponade

A history of dyspnoea, orthopnoea, and palpitations, associated with hypotension and tachycardia are classical symptoms of many cardiorespiratory illnesses. However, quiet or absent heart sounds with a raised JVP should raise concerns for cardiac tamponade. This can be confirmed with the ECG findings of sinus tachycardia, low QRS voltage, or electrical alternans.

Electrical alternans is a diagnostic feature of large pericardial effusion and cardiac tamponade. The alternate QRS amplitudes are due to the 'swinging' effect of the heart whilst contracting which subsequently gives rise to increased and decreased electrical impedance.

Deep lateral Q waves with left ventricular hypertrophy is a classical ECG finding of hypertrophic cardiomyopathy (HOCM). HOCM is the most common cause of sudden death in young people and is due to left ventricular hypertrophy without an identifiable cause. Patients can display all of the above symptoms. Dyspnoea is common due to left ventricular stiffness, resistance, and impaired filling. Patients may have a jerky pulse and may also present in arrhythmia, most commonly, atrial fibrillation. However, compared to the above scenario, patients have audible heart sounds, and may have a loud S1 and sometimes a 4th heart sound. There may be an ejection systolic murmur radiating to the apex which changes in intensity with a change in posture.

Pericarditis is associated with **global ST elevation**. Patients typically present with many of the above symptoms. Pericarditis is a common cause of pericardial effusion due to the accumulation of fluid in the pericardial sac due to inflammation. The distinguishing feature between pericarditis and pericardial effusion is the presence or absence of heart sounds, with pericarditis having audible heart sounds.

Symptoms of dyspnoea, palpitations, and orthopnoea, along with hypotension and tachycardia are common signs and symptoms associated with a pulmonary embolism (PE). The most common ECG finding in PE is sinus tachycardia, however, patients may have evidence of right axis deviation, atrial arrhythmia, right bundle branch block, P pulmonale, and **S1 Q3 T3**. S1 Q3 T3 is the presence of deep S waves in lead I along with Q waves and T wave inversion in lead 3.

Trifascicular block is the combination of third-degree atrioventricular block, right bundle branch block, and either left anterior fascicular block or left posterior fascicular block. These patients generally required pacemaker insertion. The most common causes are structural heart disease, particular fibrosis of the myocardium, and ischaemic heart disease. It may also occur in anterior myocardial infarction and aortic stenosis. Many of the pathological causes of trifascicular block may cause the symptoms described in the above scenario, however, heart sounds would still be audible. Trifascicular block is not associated with pericardial effusion.

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A 78-year-old woman attends the cardiology outpatient department after being referred by her general practitioner with angina. The patient reports consistent chest tightness after walking around 200 metres on flat ground but denies any episodes of pain at rest. Her symptoms have been stable at the current level for at least six months. Her GP has previously attempted treatment with both metoprolol and amlodipine but both were stopped after the patient reported dizziness and was found to be hypotensive.

The patient was formerly a heavy smoker but had stopped 15 years previously after suffering a non-ST elevation myocardial infarction (managed with medical therapy only). The patient had been diagnosed with type 2 diabetes mellitus 10 years previously and was known to have chronic kidney disease.

The patients current medications included aspirin 75 mg daily, simvastatin 20 mg daily, ramipril 1.25 mg daily and gliclazide 60 mg twice daily. She sometimes used a nitrate spray to relieve episodes of angina but found that it normally made her very dizzy.

Clinical examination was unremarkable except for a blood pressure of 98 / 65 mmHg.

Please see below for results of recent investigations.

Transthoracic echocardiogram: normal valvular function; moderately impaired left ventricular; ejection fraction 35 %.

Electrocardiogram: sinus rhythm at 85 beats per minute; normal axis; normal QRS complex, normal ST segments and T waves.

Urea	15.6 mmol / L
Creatinine	234 micromol / L
Sodium	135 mmol / L
Potassium	4.9 mmol / L

Cardiac stress MRI: evidence of mild myocardial ischaemia in left anterior descending territory, estimated < 10 % myocardium ischaemic.

What is the correct next line therapy for the patients angina?

A. Percutaneous coronary intervention
B. Nicorandil
C. Ranolazine
D. Ivabradine
E. Long-acting isosorbide mononitrate

D. Ivabradine

The patient has stable angina and has been intolerant of first line medical therapy. Due to her co-morbidities several of the second-line medical therapy options are contra-indicated. Ranolazine acts a late inward calcium channel antagonist but is contra-indicated in severe renal disease. Both nicorandil and ISMN are contra-indicated in this patient due to her history of hypotension.

Therefore, ivabradine is the most appropriate option. The drug acts as an If channel antagonist to reduce heart rate and is appropriate in this patient without a history of bradycardia. It should not be used in patients with moderate to severe angina as it has been shown to increase the incidence of cardiovascular events in these patients.

Revascularisation techniques such as PCI have not been show to reduce mortality or rate of MI in stable coronary artery disease. Therefore, medical therapy options should be exhausted before consideration of invasive treatment (particularly in this patient with renal failure making contrast administration very undesirable).

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A 43 year old man has been referred to endocrinology clinic for blood pressure investigation and management. Over the past year the patient has had a persistently raised blood pressure between 170/100mmHg and 180/110mmHg. Despite starting the patient on ramipril 5mg once daily four weeks ago the patient's blood pressure on this visit remained raised at 164/93mmHg.

During the history taking part of the consultation the patient mentions that he has been troubled with headaches for the past year, and has noticed that his stool frequency has increased and his stools have become looser. He tells you that he's rather embarrassed to admit that he's been having flushing episodes, and feels that his clothes are much looser than they were 1 year ago.

On discussing his family history he mentions that both his mother and his father had to have cancerous lumps removed in their middle ages. His mother had a breast lump removed, and his father had some form of pancreatic mass removed.

On examination the patient is tall with a wide arm span. Examination of the cardiovascular system reveals only a minor tachycardia of 95bpm and a quiet systolic flow murmur not radiating to the carotids. Abdominal examination reveals no palpable masses, and examination of his lungs is completely normal.

The GP had previously arranged a 24h urinary catecholamine test; results are as follows:

Total urine catecholamines 210mcg/24hr. This is raised.

As a follow up to this test the GP had arranged a CT of the patient's abdomen and pelvis which is reported as normal apart from a few incidental simple renal cysts.

Urinalysis in clinic today shows:

Leucocytes	-
Blood	-
Glucose	+
Ketones	-

Which test is most likely to elucidate the cause of the patient's hypertension?

A. Pentagastrin stimulation test
B. 24h urinary HIAA
C. MRI renal angiography
D. GH, IGF-1 and 'gut hormones'
E. MIBG (metaiodobenzylguanidine) scan

E. MIBG (metaiodobenzylguanidine) scan

This question tests the candidates knowledge of causes of secondary hypertension. The commonly listed causes of secondary hypertensions are listed below

Endocrine	Adrenal	Renal
Cushing's Syndrome	Conn's Syndrome	Diabetic nephropathy
Acromegaly	Adrenal hyperplasia	Chronic glomerulonephritis/tubul nephritis
Thyroid disease	Phaeochromocytoma	Adult polycystic kidney d
Hyperparathyroidism	-	Renovascular disease

The best way of narrowing down to the correct answer in this question is to actually look at what the answers are suggesting

Pentagastrin stimulation test	**Test for medullary carcinoma of the thyroid (associated with MEN2 - multiple endocrine neoplasia)**
24h Urinary HIAA	Screening for carcinoid
MRI renal angiography	Test for renal artery stenosis
GH, IGF-1 and 'gut hormones'	Test for pancreatic malignancy associated with MEN1

MIBG scan	Test for phaeochromocytoma

While this patient might well have MEN2 (as suggested by the patients body habitus and features consistent with phaeochromocytoma), medullary thyroid cancer would be unlikely to cause his blood pressure issues. Therefore the pentagastrin stimulation test, where one injects the patient with pentagastrin and looks for an abnormally high release of calcitonin as a response, is not a suitable first test. It may well be considered if the patient is diagnosed with a phaeochromocytoma to rule out medullary thyroid cancer as part of MEN 2.

The patient does have a few features consistent with carcinoid syndrome, for example the episodic flushing and increased stool frequency. However carcinoid syndrome doesn't present with these symptoms until it has metastasised to the liver, and this patient has had a normal CT scan of his abdomen making this a less likely diagnosis.

MRI renal angiography is used to diagnose renal artery stenosis; this patient has far too many other endocrine features for this to be renal artery stenosis. In addition the fact his blood pressure has not got worse with angiotensin converting enzyme inhibitors makes the diagnosis less likely.

Testing for gut hormones would be done in a patient in whom you were considering a diagnosis of pancreatic neoplasm as part of MEN1.

This leaves the MIBG (metaiodobenzylguanidine) scan. The patient has raised levels of total urinary catecholamines, suggesting a diagnosis of phaeochromocytoma, however, his CT scan show's no evidence. As you may be aware there is a rule of 10 that is commonly used to describe phaeochromocytoma, in that:

10% are malignant

10% are bilateral

10% are familial

10% are in children

The other sites that phaeochromocytomas can present include: Anywhere alone the sympathetic chain which runs alongside the spinal cord, the distal aorta, ureters and urinary bladder.

The MIBG scan uses radioactive iodine as a tracer for phaeochromocytoma tumour cells, which is detected through the use of a gamma camera., making it the next test that should be used in diagnosing this patient's phaeochromocytoma, i.e. the cause of his hypertension.

Question 105 of 328

An 84-year-old male was admitted to the coronary care unit (CCU) after being admitted as a primary percutaneous coronary intervention (PPCI) call for sudden onset chest pain and ST elevation on anterior leads of his ECG. Angiography demonstrated a mid-left anterior descending artery acute occlusion likely secondary to plaque rupture that was stented with a drug-eluting stent. Two hours after being admitted to CCU, he complained of nausea and vomited twice, which settled after being prescribed cyclizine by a passing medical senior house officer. Thirty minutes afterwards, you are asked to see the patient as the patient has become increasingly short of breath. He complains of no new chest pain, nausea, vomiting, sweating or palpitations. Interrogation of his cardiac telemetry reveals a new sinus tachycardia only without other arrhythmias. On examination, the patient is in respiratory distress, with bibasal inspiratory crackles and raised jugular venous pulse.

His observations are as follows: blood pressure is 186/80 mmHg, heart rate 120 beats per minute and regular, Sats 92% on 3 litres, respiratory rate 33/min.

A repeat 12-lead ECG reveals no new ST elevation changes or other ischaemic changes. A portable chest radiograph demonstrates bibasal alveolar oedema and bilateral small pleural effusions.

What is the cause of the patient's deterioration?

A. In-stent thrombosis
B. Pulmonary embolus
C. New independent cardiac ischaemic occlusion
D. Cyclizine induced heart failure
E. Cardiac tamponade secondary to cardiac instrumentation

D. Cyclizine induced heart failure

This patient has entered acute pulmonary oedema, associated with systemic hypertension and tachycardia following administration of cyclizine in the context of an acute STEMI, typical of cyclizine-induced heart failure. Cyclizine is a H1 receptor antagonist and anticholinergic, resulting in systemic hypertension and inducing tachycardia. It can aggravate a fragile myocardium into acute failure and is hence not recommended by NICE in patients with ACS or severe heart failure1. In this patient, the lack of chest pain or ST elevation on ECG makes acute in-stent thrombosis or a further ACS episode unlikely. The patient is hypertensive instead of hypotensive, with no mention of low voltage on ECG, making cardiac tamponade an unlikely cause.

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A 63-year-old patient is called to his GP for a health check. He has a past medical history of asthma and a family history of hypertension and bowel cancer. He has a 10 pack-year history of smoking but quit with the help of the practice nurse 2 years ago. He drinks 24 units of alcohol per week, and his body mass index is 25 kg/m². He does not take any regular medication. His blood pressure is 155/95 mmHg. He is sent for ambulatory blood pressure monitoring (ABPM), which shows an average blood pressure of 145/90 mmHg. His 10-year cardiovascular risk is 8%.

Examination, including fundoscopy, is unremarkable. Bloods show:

Hb	132 g/l
Platelets	330 * 10 /l
WBC	7.3 * 10 /l
Na	142 mmol/l
K	3.9 mmol/l
Urea	5 mmol/l
Creatinine	80 µmol/l
eGFR	91 ml/min
Fasting plasma glucose	5mmol/L
Serum total cholesterol	4mmol/L
HDL cholesterol	1.8mmol/L
Urinary albumin:creatinine ratio	1.1 mg/mmol
Urine dip	negative for haematuria

An ECG shows sinus rhythm.

What is the first step in the management of this man's blood pressure?

A. Lisinopril

B. Lifestyle advice

C. Amlodipine

D. Spironolactone

E. Atenolol

B. Lifestyle advice

This patient has a persistently raised blood pressure. During ABPM, two measurements are taken every hour during waking hours and at least 14 measurements are used to confirm the average blood pressure. This man's average blood pressure is >135/85 mmHg on ABPM; he has stage 1 hypertension, which warrants intervention. Given that his 10-year cardiovascular risk is low, and there is no evidence of end-organ damage, the most appropriate option at this stage is lifestyle advice.

The patient should be advised regarding diet and exercise. He has already given up smoking, but he should cut down on caffeine, salt and alcohol. Relaxation therapies have been shown to produce a small reduction in blood pressure, but these are not routinely offered in primary care. Drug treatment should be initiated if the average blood pressure is >150/95 mmHg, if he has a 10-year cardiovascular risk >10%, or if he develops end-organ damage. The first drug of choice in a patient over 55 years would be a calcium-channel blocker, such as amlodipine.

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A 23-year-old rugby player has a blackout whilst playing a match. No head injury is noted. There was no warning and he recovered quickly within seconds. This has never happened before. There was no chest pain. His father died in his sleep aged 49 and was known to have Brugada syndrome. At presentation several hours later the patient's electrocardiogram (ECG) and routine blood profile including FBC, U&E, and glucose are normal. He is sent to cardiology clinic for further investigation. A diagnosis of Brugada syndrome is suspected.

Which agent may they elect to use there to provoke the down-going ST elevation in leads V1-V3 characteristically seen in the ECG of a patient with Brugada Syndrome?

A. S Digoxin

B. Sotalol

C. Nifedipine

D. Adenosine

E. Flecanide

E. Flecanide

Exertional syncope in a young person with a family history of sudden cardiac death or with an abnormal ECG at presentation should trigger alarm bells in your head this person may be at risk of sudden cardiac death (which you should note is a preventable death if the condition is recognised in time and an implantable cardioverter defibrillator (ICD) is put in.)

The condition alluded to here is Brugada Syndrome. This is an autosomal dominant condition resulting in loss of function of the cardiac sodium channel. The characteristic ECG change is down-going ST elevation in V1 to V3. However, this may not be present, especially so long after the syncope episode. The way to bring out the ECG change in cardiology clinic safely is to give a sodium channel blocker (usually flecainide), providing additional sodium channel blockade to that already pathologically present. Once a diagnosis is made, the life-saving treatment is an ICD device. Genetic family counselling and testing will be of additional importance (testing for a mutation in the SCN5A gene).

The substantial majority of the approximately 100,000 sudden cardiac deaths each year in the UK are caused by coronary heart disease. However, most sudden deaths in people under 30 years old are caused by inherited cardiomyopathies and arrhythmias. A sensible list of differentials to consider in a young-person sudden cardiac death case are hypertrophic obstructive cardiomyopathy (HOCM), long-QT syndrome, Brugada, and arrhythmogenic right ventricular cardiomyopathy (ARVC). These will each be covered elsewhere using different questions as triggers.

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Question 108 of 328

A 65-year-old lady with a history of diabetes and newly diagnosed atrial fibrillation (AF) was admitted to hospital with a moderate-large sized embolic stroke. Her blood pressure on admission was 165/90 mmHg with a heart rate of 95 beats per minute.

An MRI brain was performed 24 hours after admission which showed a moderate to large area of infarction involving the anterior 2/3 of the left middle cerebral artery territory without haemorrhagic transformation.

With regards to management of her AF and stroke prevention, the most appropriate decision would be to commence which of the following?

A. An adjusted dose intravenous heparin infusion

B. Therapeutic dose low molecular weight heparin (LMWH) andtransition to oral anticoagulation in 10-14 days

C. Aspirin and wait 10-14 days before commencing formalanticoagulation

D. Warfarin with LMWH bridging therapy

E. OR calculate the CHADS-vasc score and make a decision

C. Aspirin and wait 10-14 days before commencing formalanticoagulation

Moderate-large areas of cerebral infarction are subject to the risk of haemorrhagic transformation within the acute period - usually defined as 10-14 days. With this in mind, patients with AF should be cautiously commenced on therapeutic dose formal anticoagulation 10-14 days after stroke onset in the absence of spontaneous haemorrhagic transformation.

The use of aspirin and/or low dose heparin/LMWH for venous thromboembolism (VTE) prophylaxis can be considered within 24 hours of symptom onset.

CHADS2 or CHADS-Vasc scores are used to estimate the risk of embolic stroke in patients with atrial fibrillation and assist with deciding on whether or not to anti-coagulate a patient. This patient scores highly enough to be started on formal anticoagulation, but this is not the focus of the question in this case. The focus is on the timing of anticoagulation in this clinical setting.

Question 109 of 328

A 57-year-old man attends his GP surgery for review. For the past year, he has experienced exertional chest pain, which initially only came on whilst walking uphill, but has now started to affect him when walking on the flat. The pain is alleviated by the use of glyceryl trinitrate spray. He denies ever having had chest pain at rest and does not report any symptoms of postural hypotension. His other medications include aspirin, atorvastatin and atenolol, which has been maximally titrated.

What is the most appropriate next step in management for this patient?

A. Refer for assessment for percutaneous coronary intervention (PCI) or CABG
B. Start amlodipine
C. Start isosorbide mononitrate
D. Start ranolazine
E. Start verapamil

B. Start amlodipine

If angina is not controlled with a beta-blocker, a longer-acting dihydropyridine calcium channel blocker should be added

The correct answer is **start amlodipine**. This patient has a likely diagnosis of stable angina, as he suffers from exertional chest pain which is alleviated by the use of GTN spray. As he is currently taking a betablocker at the maximum permissible dose, the next most appropriate step in management is to add in a calcium channel blocker. In this scenario, dihydropyridine calcium channel blockers are preferred to nondihydropyridine calcium channel blockers, due to the increased risk of the latter causing complete heart block in combination with betablockers.

Refer for assessment for percutaneous coronary intervention (PCI) or CABG is incorrect. As the patient has stable angina, with no symptoms at rest, he does not warrant urgent referral for PCI. Non-urgent referral for PCI or CABG would be appropriate if his symptoms were not improved by the addition of amlodipine.

Start isosorbide mononitrate is incorrect. This would be an appropriate third-line management option for stable angina in this patient if amlodipine failed to alleviate his symptoms. However, it should only be started whilst awaiting assessment for revascularization with PCI or CABG.

Start ranolazine is incorrect. This would also be more appropriate as a third-line management option whilst awaiting specialist assessment for PCI or CABG.

Start verapamil is incorrect. Although it would be reasonable to start a calcium channel blocker in this patient, starting a non-dihydropyridine calcium channel blocker such as verapamil in a patient already on a betablocker would run the risk of precipitating complete heart block.

Question 110 of 328

A 63-year-old man presents for review at his GP surgery. He is known to have hypertension and was started on amlodipine five years ago. Due to persistent poor hypertensive control, he began taking enalapril two years ago but has continued to struggle to keep his blood pressure within the required range. He reports no symptoms of postural hypotension, and his latest ambulatory blood pressure readings show an average blood pressure of 161/93 mmHg.

His latest blood test results are shown below:

Na +	138 mmol/L	(135 - 145)
K +	4.3 mmol/L	(3.5 - 5.0)
Urea	4.5 mmol/L	(2.0 - 7.0)
Creatinine	84 µmol/L	(55 - 120)

What medication should be offered to this patient as the next step in managing his hypertension?

A. Bendroflumethiazide
B. Bisoprolol
C. Doxazosin
D. Indapamide
E. Spironolactone

D. Indapamide

orly controlled hypertension, already taking an ACE inhibitor and a calcium channel blocker - add a thiazide-like diuretic

The correct answer is **indapamide**. Current guidelines advise that in the absence of any clear contraindications, patients with poorly controlled hypertension already taking an ACE inhibitor and a calcium channel blocker should be offered a thiazide-like diuretic. Thiazide-like diuretics are sulfonamide-based diuretics that have similar pharmacological properties to thiazides but have been shown in recent trials to reduce systolic blood pressure more effectively than conventional thiazides, without a significant difference in side-effect profile. Indapamide is the only thiazide-like diuretic provided in the answer choices.

Bendroflumethiazide is incorrect as thiazide-like diuretics, rather than thiazide diuretics such as bendroflumethiazide now tend to be preferred as third-line antihypertensive therapy in most patients for the reasons outlined above.

Bisoprolol is incorrect. This would be an appropriate option for a patient with persistent hypertension already taking a calcium channel blocker, an ACE inhibitor and a thiazide-like diuretic with serum potassium > 4.5mmol/L.

Doxazosin is incorrect. Alpha blockade with doxazosin would be another option for a patient with persistent hypertension already taking a calcium channel blocker, an ACE inhibitor and a thiazide-like diuretic with serum potassium > 4.5mmol/L.

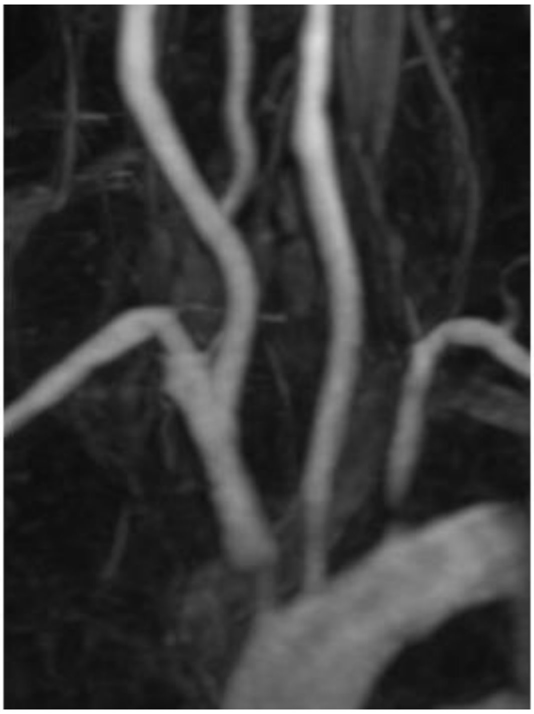
Spironolactone is incorrect. Aldosterone receptor antagonism with spironolactone may be offered to patients with persistent hypertension despite taking a calcium channel blocker, an ACE inhibitor and a thiazide-like diuretic with serum potassium < 4.5mmol/L, due to the propensity of spironolactone to cause hyperkalaemia.

Question 111 of 328

A 26-year-old woman is investigated for persistent malaise, fever, headaches and raised inflammatory markers. She was diagnosed with Raynaud's syndrome three years ago after complaining of cold hands. The most recent bloods in the referral letter show a CRP of 64 mg/l.

On examination blood pressure is 80/64 mmHg in the right arm and 72/54 mmHg in the left arm. The heart rate is 72/min and a diastolic murmur is noted on ascultation of the heart.

Magnetic resonance angiography is requested:



What is the most appropriate maangement?

A. Plasma exchange
B. Rituximab
C. Prednisolone
D. Cardiothoracic surgery referral
E. Intravenous immunoglobulin

C. Prednisolone

The magnetic resonance angiography (MRA) demonstrates stenoses of the supra-aortic arteries, especially the brachiocephalic trunk and the left subclavian artery with near-occlusion of the left vertebral artery. This findings are consistent with a diagnosis of Takayasu's arteritis.

Question 112 of 328

A 34-year-old gentleman with Marfan's syndrome and known mitral regurgitation presents to the emergency department with increasing shortness of breath for three days. He can currently walk 100 metres at a time before needing to stop. He feels restless at times and has noticed his heart is racing in his chest. There is no chest pain. He is under annual surveillance with echocardiography and is currently managed on furosemide and ramipril.

On examination, his pulse is irregularly irregular at 96 beats per minute. He is alert with a clear chest and no visible JVP. He has a soft S1 with a loud grade 4 pan-systolic murmur with radiation into the axilla. He is skinny but alert.

Na +	134 mmol/l
K +	4.2 mmol/l
Urea	4.3 mmol/l
Creatinine	89 µmol/l
ECG	no visible P-waves, irregular narrow complex rhythm at 84 per minute
CXR	cardiomegaly with no effusions or lung shadowing
BedsideECHO	No pericardial effusion, normal sized cardiac chambers, no regional wall motion abnormality

What is the most appropriate next action?

A. Increase furosemide
B. DC cardioversion
C. Bisoprolol
D. Refer to cardiothoracic surgery
E. Repeat Echocardiography

D. Refer to cardiothoracic surgery

New AF in mitral regurgitation -> refer for mitral valve replacement

This gentleman has mitral regurgitation and has developed atrial fibrillation which is a common complication following dilation of the mitral annulus. He is not in pulmonary oedema and there is no haemodynamic compromise so DC cardioversion would be inappropriate. He shows no signs of cardiac failure so diuresis with furosemide will not help. Although, he is in atrial fibrillation, his rate is slow and does not need reducing at present with a beta blocker. A repeat ECHO will be useful to confirm current heart structure. The development of atrial fibrillation is an indication to refer for mitral valve surgery as is an ejection fraction less than 60%, pulmonary hypertension or a dilated left ventricle.

Question 113 of 328

A 34-year-old male presents with increased shortness of breath on minimal exertion associated with reduced exercise tolerance. He was diagnosed with a 'heart condition' 4 years ago after three episodes of loss of consciousness. His only other past medical history is asthma, which is well controlled on a Seretide 250 inhaler. His last echocardiogram demonstrates asymmetric left ventricular and septal wall hypertrophy, associated with an increased left ventricular outflow tract gradient and systolic anterior wall motion. He was started on verapamil by his cardiologist 2 years ago but over the past 3 months, he reports a deterioration of his shortness of breath associated with increasing lower limb swelling.

Which treatment is most appropriate?

A. Propranolol
B. Disopyramide
C. Ramipril
D. DDDR pacemaker insertion
E. Cardiac myectomy

B. Disopyramide

The patient was diagnosed with echocardiogram findings associated with cardiogenic syncope in a young male, consistent with hypertrophic obstructive cardiomyopathy (HOCM). Medical treatment of HOCM consists of starting either a beta blocker, typically propranolol (but contraindicated in this case due to asthma) or a non-dihydropyridine calcium channel blocker, typically verapamil. In the case of persistent symptoms despite monotherapy, American Heart Association and European Society of Cardiology recommend consideration of disopyramide, a negative inotropic 1a anti-arrhythmic that demonstrated a significant decrease of LVOT gradients and mortality when compared to placebo. However, its significant cholinergic side effects and concerns of pro-arrhythmias, restricts its use to a second line agent. Surgical myectomy, DDDR pacemakers and alcohol ablation should be considered in patients who continue to be symptomatic despite 2 agents. ACEi should be avoided in HOCM patients with LVOT as reducing

afterload can worsen the LVOT gradient.

Question 114 of 328

A 43-year-old male presents with central chest pain that spread into his jaw and arm. He is nauseous and sweaty and arrives to the emergency department. He is immediately given aspirin and glyceryl trinitrate spray. On examination, he has normal heart sounds and a clear chest. However, he is pale and clammy

Hb	145 g/l
Platelets	400 * 10 /l9
WBC	8.6 * 10 /l9
Na +	135 mmol/l
K +	4.3 mmol/l
Urea	4.5 mmol/l
Creatinine	67 µmol/l
Troponin	957 ng/ml (normal range <20)
ECG	Biphasic T waves in V2-V3

What is the likely explanation of the ECG?

A. Normal variant
B. Brugada Syndrome
C. Wellens Syndrome
D. Hypertrophic cardiomyopathy
E. Takotsubo cardiomyopathy

C. Wellens Syndrome

Deeply inverted or biphasic T waves in V2-V3 suggests Wellen's syndrome and high risk of critical LAD stenosis

This gentleman has clear acute coronary syndrome. The ECG findings are unique to Wellens Syndrome in which there is a critical stenosis of the left anterior descending artery. These patients are at risk of imminent occlusion of this artery and should be admitted for coronary angiography and further intervention. Takotsubo cardiomyopathy has ST elevation in the apical leads (V5, V6). Hypertrophic cardiomyopathy typically has T wave inversion in the anteroseptal leads. Brugada has ST elevation and T wave inversion in V1-V2. Biphasic T-waves are not a normal variant.

Question 115 of 328

A 65-year-old man presents with loss of consciousness. He has a past medical history of two myocardial infarctions, one of which was four weeks ago. His wife states that he felt dizzy, complained of his vision going dark, and then fell to the floor.

An ECG shows ST elevation in V1-V4 without reciprocal depression.

What is the most likely diagnosis?

A. Thromboembolic stroke
B. Ventricular tachycardia
C. Anterior STEMI
D. Subarachnoid haemorrhage
E. Subdural haematomaubmit answer

B. Ventricular tachycardia

The presence of ST elevation without reciprocal depression shortly following a myocardial infarction is suggestive of a left ventricle aneurysm. Left ventricle aneurysms predispose to both ventricular arrhythmias and cardiac thrombo-embolisms.

The presence of ST elevation without reciprocal depression shortly following a myocardial infarction is very suggestive of a left ventricle aneurysm. Left ventricle aneurysms predispose to both ventricular arrhythmias and cardiac thromboembolisms, both of which are high up the differential in this case. The patient has presented with classic features of syncope, which makes an arrhythmia much more likely than an embolic stroke which rarely presents with syncope.

Subarachnoid haemorrhage can present with both loss of consciousness and ST elevation, and would be an important differential to consider, albeit less likely.

Question 116 of 328

A 36 year old woman presents to the medical assessment unit with a 2 day history of pleuritic right sided chest pain, and shortness of breath. She is 38 weeks pregnant with her first child, her pregnancy so far had been uncomplicated.

On examination, she is slightly tachypnoeic, with no added sounds on chest auscultation.

There is no clinical evidence of a deep vein thrombosis. Her oxygen saturations are 95% on room air, heart rate 98/min.

What is the next appropriate investigation?

A. CTPA
B. D Dimer
C. Chest x-ray
D. V/Q scan
E. Bilateral leg dopplers

C. Chest x-ray

Venous thromboembolic event (VTE) is an important cause of maternal death in the UK. Pregnant women are 10x more likely to have a VTE than non-pregnant women of the same age.

Where there is suspicion of a pulmonary embolism (PE) , a chest x-ray should be performed initially. If this does not explain the patient's symptoms, compression duplex dopplers should be performed. A diagnosis of deep vein thrombosis (DVT) can indirectly confirm a PE, reducing radiation doses for the mother.

If both tests are negative, and the clinical suspicion of PE remains high, further imaging should be organised; either CTPA or ventilationperfusion scan (V/Q scan). British thoracic society guidelines (BTS) recommend a CTPA in non-pregnant women.

The decision as to which scan to perform should be ideally taken with the input from the patient. V/Q scanning carries a slightly increased risk of childhood cancer compared with CTPA but carries a lower risk of maternal breast cancer. The ventilation component of the V/Q scan may be able to be omited, reducing the radiation dose.

A D Dimer would not assist in the diagnosis, as it may be raised anyway in pregnancy due to disturbances in the coagulation system.

Guidelines: RCOG- The acute management of thrombosis and embolism during pregnancy and the puerperium

Question 117 of 328

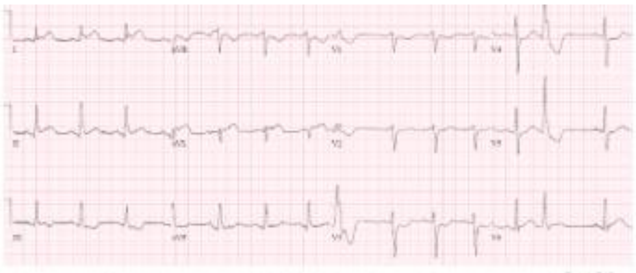
A 50-year-old woman with a past medical history of type 1 diabetes, angina, and hypertension presents with vomiting and epigastric discomfort that came on suddenly earlier this morning.

Her blood glucose level is 10.4 mmol/L and her urine dip is '+' for ketones.

Observations are as follows:

- Respiratory rate: 19/min
- Oxygen saturations: 96% on room air
- Heart rate 96bpm
- Blood pressure 105/67 mmHg

Her blood results are awaited, and her ECG is shown below.



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What diagnostic option is most likely to confirm the diagnosis?

A. Abdominal x-ray
B. CT scan of the abdomen
C. Repeat ECG with posterior chest leads
D. Serum cortisol
E. Venous blood gas

C. Repeat ECG with posterior chest leads

The ECG here shows sinus rhythm with a rate of roughly 85bpm. There is ST elevation in leads I, aVL, and v6. There is ST depression in lead v3 and v4. Conduction appears to be normal. There are two premature ventricular complexes (PVCs).

The above findings can be explained by an acute coronary syndrome involving the posterior and lateral coronary territories. Posterior chest leads would help to confirm a posterior myocardial infarction, so **repeat ECG with posterior chest leads** is the correct answer.

A **venous blood gas** would be the option of choice if aiming to confirm diabetic ketoacidosis, but given the blood glucose is <11.0 mmol/L and ketonuria is less than 2+, this diagnosis is less likely.

CT scan of the abdomen would be correct if her symptoms were thought to be coming from an intraabdominal cause, but given the ECG evidence of an acute coronary syndrome which would explain her epigastric discomfort and vomiting, this seems less likely.

Serum cortisol would be useful in confirming a diagnosis of adrenal crisis, which could present as epigastric discomfort, hypotension and vomiting. The ECG findings make the diagnosis of acute coronary syndrome more likely.

Abdominal x-ray would be useful in looking for evidence to suggest bowel obstruction or perforation. Obstruction would not explain the territorial ST-segment changes and so is not correct in this instance.

Question 118 of 328

A 60-year-old woman has a heart rate of 180bpm on the medical admission unit. An ECG performed demonstrates a regular narrowcomplex tachycardia. The patient complains of palpitations but no chest pain and blood pressure remains 140/95. Initial vagal maneuvers are performed including carotid massage with no success. Intravenous adenosine 6mg is given followed by a further two doses of adenosine 12mg. Unfortunately, this failed to cardiovert or reveal an interpretable underlying rhythm.

What is the next management step?

A. Further adenosine
B. Synchronised DC cardioversion
C. Verapamil
D. Digoxin
E. Amiodarone

C. Verapamil

The resuscitation council recommends:

Adenosine 6mg for supraventricular tachycardias followed by a further two doses of adenosine 12mg if no response. If adenosine is contraindicated, or fails to terminate a regular narrow-complex tachycardia without demonstrating atrial flutter, consider giving a calcium-channel blocker, for example intravenous verapamil 2.5 - 5mg over 2 min.

The combination of vagal maneuvers and 6mg/12mg/12mg will terminate most regular narrow-complex tachycardias. Further adenosine is not thought to be of benefit.

Digoxin and amiodarone are used in the treatment of atrial fibrillation but not indicated in regular narrow-complex tachycardias. However, remember to consider alternative diagnoses such as atrial flutter/fibrillation, especially when the above rhythm fails to terminate after adenosine.

The patient is stable; with no adverse features to suggest DC cardioversion is indicated.

Question 119 of 328

A 82-year-old man who is resident in a nursing home and has diabetes, hypertension and bilateral below knee amputations for peripheral vascular disease presents with a two month history of gradual deterioration. He is generally tired and has little appetite. He has lost 6 pounds since the start of the year and is not keen to leave his room anymore. He has spiked temperatures on numerous occasions and been given several course of oral antibiotics by the GP for possible urinary and lower respiratory tract infections.

On visiting him, you note a new pan systolic murmur which radiates into the axilla. He has several splinter haemorrhages and a palpable spleen. There are small haemorrhages and infarctions seen on fundoscopy. He is admitted to the emergency department?

CRP	72 mg/L
ESR	64 mm/hr
Blood cultures	Viridans streptococci
Transthoracic ECHO	11mm vegetation on the anterior cusp of the mitral valve

What is the likeliest cause behind this presentation?

A. Poor dentition
B. Former intravenous drug use
C. Childhood rheumatic fever
D. Senile calcification
E. Repeat courses of antibiotics

A. Poor dentition

Patients with very poor dental hygiene may develop endocarditis secondary to Viridans streptococci e.g. *Streptococcus sanguinis*

This is a case of infective endocarditis which can be acute or chronic. The chronic endocarditis tends to present over months with gradual deterioration and is most often caused by *Viridans streptococci*. This is a common oral commensal and observation of the oral cavity will likely reveal poor dental disease. This is common in people who are cared for where dental hygiene needs can be missed or difficult to achieve. Intravenous drug use is a cause commonly of *Staphylococcus aureus* endocarditis which is acute and rapidly fatal. Childhood rheumatic fever and senile calcification are rarer causes of endocarditis. Repeat courses of antibiotics causes *Clostridium difficile* colitis not endocarditis.

Question 120 of 328

A 78-year-old lady presents with syncope. She states that she has had several episodes of light headedness over the past couple of months. There are no obvious precipitants of the dizzy spells. She has a past medical history of angina, COPD and hypertension. Her regular medicines include GTN, aspirin, ramipril and furosemide.

ECG results are as follows:

Rate	68 beats per minute
Rhythm	Sinus rhythm
PR interval	220 ms
QRS duration	130 ms
QRS morphology	Right bundle branch block
Cardiac axis	Left axis deviation

What investigation will you order?

A. 7-day ECG Holter monitor
B. Transthoracic echocardiogram
C. Transesophageal echocardiogram
D. Brain natriuretic peptide (BNP)
E. Tilt-table test

A. 7-day ECG Holter monitor

Patients who present with syncope and have an ECG showing incomplete trifascicular block need a Holter test to assess for episodes of complete heart block

The right bundle branch block (RBBB), left axis deviation (LAD) and prolonged PR interval are suggestive of incomplete trifascicular block.

These patients require a 7-day ECG Holter monitor to assess for episodes of complete (third degree) hear block. It is likely that a permanent cardiac pacemaker will be required to prevent further syncopal episodes.

A tilt-table test is useful for investigating vasovagal syncope.

The patient may also have underlying structural heart disease so a transthoracic echo would also be prudent to perform. However, the most immediate concern is of arrhythmia, making a 7 day holter the better answer.

Question 121 of 328

An 18-year-old man attends the clinic with blurred vision. This has progressively worsened over the past few years. He has a past medical history of recurrent deep vein thromboses and mild learning difficulty. On examination, you note an increased arm span to body height ratio, and the presence of scoliosis. Ophthalmological assessment reveals a downward lens dislocation.

What is the most likely diagnosis?

A. answerEctopia lentis syndrome
B. Ehlers danlos syndrome
C. Homocystinuria
D. Marfan's syndrome
E. Multiple endocrine neoplasia type 2B

C. Homocystinuria

Tall, long fingered, downward lens dislocation, learning difficulties,

DVT - homocystinuria

The patient has several clinical features of a Marfanoid body habitus. The differential for a Marfanoid body habitus is wide and includes Marfan's syndrome and homocystinuria.

Homocystinuria is correct. The patient has several clinical features associated with homocystinuria (e.g. Marfanoid body habitus, recurrent DVTs, learning disability and ectopia lentis). It is important to remember that the ectopia lentis in homocystinuria is inferonasal, in contrast to the superior-temporally dislocation associated with Marfan's syndrome.

Ectopia lentis syndrome is incorrect. Ectopia lentis syndrome is an inherited connective tissue condition that shares some of the features of Marfan's syndrome - particularly lens dislocation of the eye. However, patients with ectopia lentis syndrome do not have any vascular complications associated with Marfan's syndrome. Patients with ectopia lentis syndrome do not have any of the other features demonstrated in this case.

Ehlers danlos syndrome is incorrect. The Ehlers-Danlos syndromes (EDS) are generally characterised by joint hypermobility, joint instability and dislocations, scoliosis, and other joint deformities. In the rarer types of EDS, there is also a weakness of specific tissues that can lead, for example, to major gum and dental disease, eye disease, cardiac valve and aortic root disorders, and life-threatening abdominal organ, uterine, or blood vessel rupture. Thrombotic complications and lens dislocation are rare in this condition making it a less likely diagnosis.

Marfan's syndrome is incorrect. Although the patient has a Marfanoid body habitus, the presence of downward lens dislocation, learning difficulties, and recurrent DVTs favour the diagnosis of homocystinuria.

Multiple endocrine neoplasia type 2B is incorrect. This is a rare, genetic disorder that affects the endocrine glands and causes medullary thyroid cancer, pheochromocytoma, and parathyroid gland cancer. Although it can be associated with a Marfanoid body habitus, there are no other clinical features to suggest that this is the diagnosis.

Question 122 of 328

A 67-year-old with a history of ischaemic heart disease (primary percutaneous intervention for a STEMI three years ago) is admitted with a pyrexia of unknown origin. On examination his pulse is 96/min, temperature 38.2ºC and blood pressure 104/66 mmHg. A systolic murmur is noted but auscultation of the chest is unremarkable. His postmyocardial infarction echocardiogram three years ago showed no valvular disease. Chest x-ray is normal and urine dipstick shows blood ++. A petechial rash is noted on his hands and legs. A presumptive diagnosis of infective endocarditis is made and empirical treatment with IV amoxicillin and gentamicin given. Two days later blood cultures show a coagulase-negative staphylococcus.

What is the most appropriate action with respect to antibiotic therapy?

A. Switch to flucloxacillin
B. Switch to flucloxacillin + vancomycin + rifampicin
C. Make no changes to treatment
D. Switch to flucloxacillin + vancomycin
E. Switch to flucloxacillin + rifampicin

A. Switch to flucloxacillin

The BNF now recommend flucloxacillin monotherapy for native-valve endocarditis caused by staphylococci.

Question 123 of 328

A 49-year-old Afro-Carribean man is diagnosed with hypertension. He has no significant past medical history and remains asymptomatic. His 24 hour blood pressure monitoring shows an average of 165/97mm Hg.

Which of the following drugs should be used initially to lower his blood pressure?

A. Ramipril
B. Bisoprolol
C. Amlodipine
D. Indapamide
E. Furosemide

C. Amlodipine

Newly diagnosed patient of black African or African-Caribbean origin with hypertension - add a calcium channel blocker

This patient has stage two hypertension. Because he is of Afro-Caribbean origin, the first line drug is a calcium channel blocker, not an angiotensin converting enzyme inhibitor. Diuretics and beta-blockers are not first line management for hypertension.

Question 124 of 328

A 73-year-old man with a history of severe biventricular heart failure comes to the clinic for review. He can only walk around 100 metres to the bus stop and climbs the stairs once per day only. Medication includes ramipril 10mg, furosemide 80mg, bisoprolol 10mg, spironolactone 25mg, aspirin and atorvastatin. On examination his blood pressure is 123/82 mmHg, pulse is 85 beats per minute at rest. He has bilateral basal crackles on auscultation of the chest and mild pitting oedema of both ankles.

Investigations

Hb	122 g/l	Na +	134 mmol/l	Bilirubin	11 µmol/l
Platelets	190 * 10 /l 9	K +	5.0 mmol/l	ALP	105 u/l
WBC	7.4 * 10 /l 9	Urea	11.2 mmol/l	ALT	33 u/l
Neuts	4.5 * 10 /l 9	Creatinine	142 µmol/l	γGT	56 u/l
Lymphs	1.2 * 10 /l 9	Albumin	38 g/l		
Eosin	0.5 * 10 /l9				

Chest x-ray: Cardiomegaly, bilateral upper lobe diversion ECG: Left bundle branch block, ventricular rate 85 ECHO: Ejection fraction 31%

Which of the following is the most appropriate next step with regards to his heart failure medication?

A. Add digoxin
B. Add valsartan

C. Add ivabradine
D. Increase furosemide by 40mg per day
E. Increase spironolactone to 100mg per day

C. Add ivabradine

The correct answer is add ivabradine. This patient has uncontrolled symptoms of heart failure despite near maximal medical therapy. His heart rate is greater than 75 beats per minute at rest, according to NICE guidelines this means ivabradine is indicated as a potential intervention. The ivabradine outcome study in heart failure indicated an 18% relative risk reduction in cardiac events (mainly driven by heart failure admissions and heart failure deaths), for the intervention added in to standard of care versus standard of care alone.

A large meta-analysis of use of digoxin in heart failure suggested a 14% increase in mortality associated with its' use. This has led to a reduction in use of digoxin for heart failure in recent years. With respect to increasing spironolactone or adding valsartan, these options are likely to significantly increase risk of hyperkalaemia with limited benefit in improving symptoms. Although increasing furosemide may reduce symptoms of heart failure, it may also precipitate a large rise in serum creatinine.

Question 125 of 328

A 77-year-old female has been admitted electively for investigation of raised pulmonary arterial pressures (50 mmHg) detected on a transthoracic echocardiogram, which had otherwise demonstrated normal left ventricular function and chamber sizes. No valve abnormalities were identified. Her right heart catheter saturations are shown as below:

SVC	76%
IVC	74%
Right atrium (high)	73.5%
Right atrium (mid)	73%
Right atrium (low)	72.7%
Right ventricle	72%
Pulmonary artery	70.8%
Pulmonary capillary wedge	95%

What is the most likely diagnosis?

A. Normal study
B. Patent ductus arteriosus
C. Anomalous pulmonary venous drainage to SVC
D. Patent foramen ovale
E. Tricuspid regurgitation

C. Anomalous pulmonary venous drainage to SVC

This is a very subtle question. When interpreting right heart catheter data, remember the saturation should decrease gradually as the venous blood reaches the pulmonary capillary wedge saturation, which should be equal to arterial blood. The second consideration for normal venous return is that oxygenation in the superior vena cava should always be lower than the inferior vena cava, due to the high oxygen demands from the brain. There are no sudden increases in oxygen saturations in this study, suggestive of a left to right shunt but the SVC saturations is markedly higher than the IVC, allowing a diagnosis of anomalous pulmonary venous drainage of more highly oxygenated blood into the SVC.

Question 126 of 328

A 72-year-old gentleman is admitted with syncope and palpitations. He is known to have atrial fibrillation and is on flecainide and warfarin. His past medical history also includes benign prostatic hypertrophy and essential thrombocytosis.

His other medications include tiotropium 18 micrograms OD, tamsulosin 400 micrograms OD, ramipril 2.5mg and simvastatin 40mg. He has recently stopped a tablet due to shortness of breath but cannot remember what it was.

On arrival, he is tachycardic with a heart rate of 215 /min. He is alert. He denies chest pain but is aware of some shortness of breath. He is attached to a 3-lead cardiac monitor which shows a narrow complex tachycardia of 210-220 beats per minute. It is difficult to tell on the monitor however it appears regular. A 12-lead ECG confirms a regular narrow complex tachycardia with a ventricular rate of 215/min.

What is the most likely diagnosis?

A. Atrial fibrillation
B. Atrial flutter with 1:1 conduction
C. Atrial flutter with variable conduction
D. Atrio-ventricular re-entry tachycardia (AVRT)
E. Atrio-ventricular nodal re-entry tachycardia (AVNRT)

B. Atrial flutter with 1:1 conduction

Flecainide is a pure sodium channel blocker which prolongs the cardiac action potential to slow conduction. It is recognised to increase the risk of AF transforming into flutter. Flutter with 1:1 conduction would result in a ventricular rate of 300 /min which is impossible to conduct via the AV node. However, because flecainide slows the rate of cardiac conduction it means that if a patient develops atrial flutter the atrial rate will not be 300 /min, but more likely around 200 /min which then could conduct via the AV node at a 1:1 ratio. For this reason, patients should always be co-prescribed a beta blocker to reduce risk if this happens, and you wonder if this was the tablet that had recently been stopped.

The key to this question is recognising the increased risk of atrial fibrillation transforming into atrial flutter in patients on flecainide. The regular rhythm on ECG makes atrial fibrillation and atrial flutter with variable conduction less likely. He has no specific risk factors for AVRT or AVNRT, however, his flecainide use increases the chance of atrial flutter and therefore this is the correct answer.

Question 127 of 328

An 87-year-old gentleman is reviewed in the cardiology clinic. He has unstable angina with multiple confluent lesions in all three main coronary vessels and in major tributaries on angiography. The lesions are not amenable to stenting or bypass. On assessment in the clinic, the blood pressure is 104/43mmHg, heart rate is 57bpm. His ECG confirms atrial fibrillation but no new ischaemic changes. There is no clinical sign of heart failure. He is currently taking aspirin, bisoprolol, ramipril, isosorbide mononitrate, nicorandil and simvastatin. His main symptom is chest pain on minimal exertion.

Which of the following is the most appropriate drug to control this patients angina?

A. Diltiazem
B. Digoxin
C. Ivabradine
D. Omega 3
E. Ranolazine

E. Ranolazine

National guidelines were published by the Scottish Intercollegiate Guideline Network (SIGN) on the management of stable angina in 2007 with updates by NICE in 2011. Stable angina is defined as chest pain of cardiac aetiology which is precipitated by exertion with a demonstrable flow limiting lesion on angiography. Many patients who experience angina will ultimately go on to have revascularisation procedures to improve or restore coronary flow which will improve their symptoms of angina, however while waiting for interventional procedures optimisation of drug therapy is the aim. Drug therapy also is considered first-line treatment for non-critical lesions.

First line therapy in the treatment of angina is beta-blockade. Betablockers reduce myocardial oxygen consumption by acting as a negative inotrope and also by reducing heart rate. Some patients are unable to take beta-blockers such as asthmatics, or find that side effects such as tiredness, eczema or sexual dysfunction mean they are not tolerable. In these instances, use of calcium channel blockers such as diltiazem are recommended as first-line treatment. Since both of these classes of drug cause a reduction in heart rate and blood pressure their use must be monitored carefully, especially in the elderly. Concomitant use of betablockers and calcium channel blockers should be avoided where possible due to the risk of high degree conduction block and cardiac arrest.

Long-acting nitrates, such as isosorbide mononitrate or dinitrate are considered second-line therapy in stable angina. They can be used as monotherapy or as an adjunct to beta-blocker or calcium channel blocker therapy. They work by causing vasodilatation; both drugs possess a nitric oxide moiety which causes endothelial relaxation in blood vessels. This causes coronary vasodilation and improved blood flow to the myocardium with improved symptoms. A consequence of vasodilatation is hypotension and this should be considered when prescribing this drug to patients.

Another second-line option drug for stable angina in those patients who cannot tolerate nitrates or they are ineffective is nicorandil. This drug has a similar ultimate effect in that it causes dilatation of peripheral and coronary vessels to reduce symptoms of angina. It works by activating potassium channels on endovascular smooth muscle cells reducing intracellular calcium and hence relaxing the blood vessel. It too can cause profound hypotension and severe headaches.

Ivabradine is a drug which can be used for both angina and heart failure. It works by reducing heart rate to reduce myocardial oxygen demand and improve diastolic function. In this case, the heart rate is adequately controlled so reduction is not necessary. Ivabradine also works by inhibiting the hearts natural pacemaker potential in the sinoatrial node, hence it only works when the patient is in sinus rhythm, unlike here.

Ranolazine is a viable option in the management of angina where other avenues have been exhausted. It works by inhibiting the delayed sodium influx channel in the myocardium, reducing the intracellular calcium concentrations in the heart muscle. This, in turn, leads to a negative inotropic effect and reduction in symptoms of angina. Ranolazine does not cause a profound hypotensive effect and is the best choice in this scenario.

Digoxin is not commented upon as a specific treatment for heart failure In NICE guidance although it does have a modest positive inotropic effect. The relative bradycardia of the patient in the vignette makes this an unwise choice here.

Omega 3 and other fatty fish oils are not recommended to be prescribed to heart failure patients in NICE guidance due to lack of evidence of any effectiveness.

Question 128 of 328

A 32-year-old primigravida at 37 weeks attends the antenatal unit complaining of abdominal pain which is worse on the right side. She has also been vomiting. Her blood pressure is 148/97 mmHg. She denies any abnormal discharge and reports that fetal movements are still present. Her blood results are shown below.

Hb	93 g/l
Platelets	89 * 10 /l9
WBC	9.0 * 10 /l9
Urate	0.49 mmol/l
Bilirubin	32 µmol/l
ALP	203 u/l
ALT	190 u/l
AST	233 u/l

You are phoned for advice on the results. What is the most likely diagnosis?

A. HELLP syndrome
B. Obstetric cholestasis
C. Acute fatty liver
D. Hyperemesis gravidarum
E. Gout

A. HELLP syndrome

The most likely diagnosis here is HELLP syndrome (haemolysis, elevated liver enzymes and low platelets), a serious manifestation of preeclampsia. The clinical features of hypertension, vomiting and abdominal pain support the diagnosis but are not pre-requisites. The abdominal pain here may be a sign of liver inflammation and resulting stretch of the liver capsule.

Obstetric cholestasis is associated with intense pruritus and the most sensitive marker is a rise in serum bile acids. Acute fatty liver is another serious condition, which also has associations with pre-eclampsia. It would typically cause greater elevations in liver enzymes and a deep jaundice. Hyperuricaemia may be a useful marker of pre-eclampsia and does not necessarily indicate an attack of gout. Urate is thought to rise due to diminished kidney function and reduced clearance. Hyperemesis gravidarum should be a diagnosis of exclusion and would be unlikely to present for the first time this late into pregnancy

Question 129 of 328

A 76-year-old female presents to the clinic for evaluation in the company of her daughter. She has a history of ischaemic cardiomyopathy with a recent echocardiogram showing an ejection fraction of 30%, increased filling pressures, moderate aortic stenosis, moderate mitral regurgitation, and pulmonary arterial systolic pressures of 32 mmHg. At present, she can walk approximately 100 yards before she gets significantly short of breath, and this has been the case since her last hospitalisation 4 months ago. Otherwise, she feels reasonably well and has no new complaints today. She monitors her weight regularly at home and says that these have been stable.

Her medications include aspirin, amlodipine, metoprolol, lisinopril, furosemide, gabapentin, as required paracetamol, and timolol and brinzolamide eye drops. Physical examination shows a heart rate of 63 beats per minute and blood pressure of 129/76 mmHg. Her JVP is about 4cm above the angle of Louis, her apical impulse is laterally displaced, and heart sounds are audible with a holosystolic murmur heard loudest at the apex. Auscultation of her chest is clear and she has trace peripheral oedema.

What is the next best step in the management of this patient?

A. Discontinue amlodipine
B. Add spironolactone
C. Undertaken ECG to assess her QRS interval
D. Up-titrate the dose of furosemide
E. Go down on the dose of her metoprolol

B. Add spironolactone

Patients with heart failure and an ejection fraction < 35% who are euvolaemic and are already taking an ACE-inhibitor and a betablocker should have an aldosterone antagonist added to their regimen as a next step

This patient has congestive heart failure related to her history of ischaemic cardiomyopathy. She seems to be doing relatively well, and her symptoms are stable. In addition, she has been monitoring her weight at home which has also been stable, and she does not have signs of volume overload on physical exam. Her heart rate and blood pressure are also in acceptable limits. Of note, her recent echocardiogram shows an ejection fraction of 30%. She, therefore, qualifies for the addition of an aldosterone antagonist to her drug regimen. Individuals with congestive heart failure who already on an ACE-inhibitor and betablocker, and to have an ejection fraction of less than 35% who are started on an aldosterone antagonist have been shown to have low mortality compared to those who do not have an aldosterone antagonist added to their medications.

Her blood pressure is acceptable at the moment and so she does not need to discontinue amlodipine.

Cardiac resynchronization therapy is indicated in heart failure patients with NYHA functional class III and IV patients who remain symptomatic despite stable, optimal heart failure medical therapy and who have an LVEF ≤ 35% and a prolonged QRS duration on an ECG. This patient is not yet on optimal medical therapy and should be started on an aldosterone antagonist before she can be considered for this approach.

She does not have signs of volume overload and therefore does not require changes to the dose of her furosemide today.

Her heart rate is in acceptable limits today and she does not have symptoms to suggest that the dose of her beta-blocker needs to be decreased.

Question 130 of 328

An 84-year-old presents to the emergency department with and acute deterioration in shortness of breath on a background of 9 months of progressively reduced exercise tolerance. Her past medical history includes hypertension, previous myocardial infarctions 7 months ago, chronic kidney disease and COPD. She has a 50 pack year smoking history giving up 2 years ago. Her exercise tolerance is 150 yards, limited by shortness of breath.

On examination, an ejection systolic murmur can be heard in the aortic area with bibasal crackles on chest auscultation. An admission chest xray is consistent with pulmonary oedema and intravenous diuresis is commenced. A transthoracic echocardiogram demonstrates ejection fraction of 33% with impaired left ventricular function, no vegetations, a bicuspid heavily calcified aortic valve with an area of 0.7cm2 and a peak gradient of 32mmHg. Angiography demonstrates non-flow limiting stenosis of 65% in the left anterior descending artery but no lesions requiring revascularisation. Lung function testing following successful diuresis reveals forced vital capacity at 55% of predicted and forced expiratory volume in 1 second at 48% of predicted.

The patient is keen for a definitive intervention if appropriate.

What is the appropriate next action?

A. answerNo action
B. Balloon aortic valvuloplasty
C. Aortic valve replacement
D. Transcatheter aortic valve implantation
E. Coronary artery bypass graft and aortic valve replacement

D. Transcatheter aortic valve implantation

The patient has presented with pulmonary oedema on a background of severe aortic stenosis, secondary to calcifications and a bicuspid valve. Interestingly, this is a patient who presents with low-gradient low-area aortic stenosis not uncommonly encountered in more elderly patients. In the context of poor LV function, the aortic valve gradient may be normal or only mildly raised in the presence of a severely narrowed aortic valve area.

A second consideration is the suitability of this patient for surgery. She has underlying lung disease secondary to COPD and functional impairment on pulmonary testing, renal impairment and recent myocardial infarction. She is a high-risk surgical candidate and unlikely suitable for aortic valve replacement. Inaction is not an option, the patient has presented with pulmonary oedema and underlying exerciseinduced dyspnoea. Balloon aortic valvuloplasty is a palliative procedure prone to restenosis for patients unsuitable for other interventions. She does not require a CABG for her coronary artery disease, which worryingly seems inconsistent with the degree of left ventricular impairment, suggesting aortic stenosis to likely be responsible for the majority of symptoms. A transcatheter aortic valve implant (TAVI) is the appropriate management in this situation. The catheter-delivered device produces similar one-year survival as aortic valve replacement but a higher risk of stroke, TIAs and vascular complications. Compared to medical management alone, one-year survival is reduced with TAVI from 50.7% to 30.7%

Question 131 of 328

A 53-year-old gentleman is reviewed in clinic due to abnormal blood tests. He has a past medical history of hypertension, type two diabetes, obesity, and depression. He started taking atorvastatin two months ago due to a routine assessment of QRISK and elevated cholesterol levels. He had a blood test as requested at three months following the start of treatment.

These shows that his alanine aminotransferase have increased from 28iU/L to 94iU/L. Other blood tests have remained within normal ranges apart from cholesterol which as improved from 5.4mmol/L to 4.9mmol/L.

How should his atorvastatin treatment be managed?

A. Stop atorvastatin and repeat LFT within 4-6 weeks
B. Continue atorvastatin and repeat LFT within 4-6 weeks
C. Change atorvastatin to simvastatin
D. Investigate for elevated creatinine kinase
E. Stop atorvastatin and arrange for an urgent abdominalultrasound

B. Continue atorvastatin and repeat LFT within 4-6 weeks

The correct answer is to continue atorvastatin and repeat LFT within 4-6 weeks. This patient has developed elevated transaminases following starting a statin, but these are within three times the upper range of normal. Please note that this is three times the maximum normal range, not three times above the patient's baseline result. Therefore it is acceptable to continue atorvastatin but monitor liver function with a repeat test in 4-6 weeks. If the level had been greater than three times the normal upper range then stopping the atorvastatin and repeating LFT would have been appropriate. There is no need to arrange for an ultrasound at the moment as there is a clear explanation, and there is no evidence of new muscle pain to justify testing for creatinine kinase. Changing statin can play a role if the statin is not tolerated, but that is not the case so far.

Question 132 of 328

A 28 year-old man with a background of asthma presents to the emergency department with palpitations.

He reports a 2 day history of coryzal symptoms and wheeze. He has been using his salbutamol inhaler several times per day due to these symptoms.

On examination the patient appears alert and comfortable but complains of a fluttering sensation in his chest. The pulse rate is 180bpm and the blood pressure is 135/90mmHg. Oxygen saturations are 96% on room air.

There is equal bilateral air entry with mild polyphonic wheeze throughout on chest auscultation.

A 12-lead ECG reveals a narrow-complex regular tachycardia at 180bpm with pseudo r' waves after each QRS complex in lead V1.

What is the most appropriate initial management?

A. Adenosine 6mg IV
B. Metoprolol 5mg IV
C. Synchronised DC shock
D. Vagal manoeuvres
E. Amiodarone 300mg IV

D. Vagal manoeuvres

The first-line management of SVT is vagal manoeuvres: e.g. Valsalva manoeuvre or carotid sinus massage

This scenario describes a supraventricular tachycardia, most likely to be atrioventricular nodal reentrant tachycardia (AVNRT). AV nodal reentrant tachycardia is the most common regular supraventricular tachycardia.

Often a retrograde p-wave is visible in continuity with the QRS complex, appearing as a 'pseudo r'' wave in lead V1 or a 'pseudo s'' wave in the inferior leads.

According to the Resuscitation council guidelines, a regular narrowcomplex tachycardia in the absence of adverse features (shock, syncope, myocardial ischaemia or heart failure) should be treated initial with vagal manoeuvres. Adenosine and metoprolol may worsen bronchospasm so would not be appropriate in this setting. IV Verapamil or synchronised DC cardioversion could be tried if vagal manoeuvres are unsuccessful.

Question 133 of 328

A 45-year-old male is referred to the rapid access chest pain clinic with symptoms typical of angina. He is a non-smoker and has no past medical history. The pain is located in the centre of his chest and made worse by exertion.

According to the NICE guidelines, which is the most appropriate investigation?

A. Treadmill ECG
B. Trans-thoracic echocardiogram
C. CT-coronary angiography
D. Nuclear perfusion stress test
E. Diagnostic coronary angiography

C. CT-coronary angiography

The correct answer is **CT-coronary angiography**. According to the NICE guidelines (CG95), a CT coronary angiogram should be offered as the first-line investigation for patients with typical or atypical angina symptoms and who are in the intermediate risk group (10-29% 10-year risk of developing cardiovascular disease). This patient fits into this category due to his age, sex, and lack of other risk factors. The CT coronary angiogram will help visualise the coronary arteries and identify any significant stenosis.

Treadmill ECG was once a common initial test for angina but it has now been superseded by more accurate tests such as CT coronary angiography. Treadmill ECG can have false positives and negatives, hence it is not recommended as a first-line investigation by NICE.

A **Trans-thoracic echocardiogram** is useful in assessing cardiac function but it does not directly visualise the coronary arteries so it would not be appropriate in this case. It may be used in conjunction with other tests if there are signs of heart failure or valvular heart disease.

Nuclear perfusion stress test involves injecting a radioactive tracer and then imaging the heart under stress and at rest. While this test can identify areas of myocardium that are under-perfused due to significant stenosis, it exposes patients to radiation, is more expensive than CT angiography and does not directly image the coronaries. Hence, it is usually reserved for cases where other investigations are contraindicated or inconclusive.

Finally, **Diagnostic coronary angiography**, while considered the gold standard for diagnosing coronary artery disease, is invasive and carries risks such as bleeding, stroke or kidney damage from contrast dye. As per NICE guidelines, it should only be used when non-invasive tests are inconclusive or contraindicated or when immediate revascularisation is being considered.

Question 134 of 328

A 68-year-old male is admitted with confusion and an ataxic gait. His past medical history includes heart failure and atrial fibrillation. His drug history includes dabigatran, bisoprolol and ramipril.

Investigation results are as follows:

Hb	110 g/l	Na +	139 mmol/l
Platelets	140 * 10 /l 9	K +	3.8 mmol/l
WBC	10.2 * 10 /l 9	Urea	6.5 mmol/l
Neuts	8.2 * 10 /l 9	Creatinine	42 µmol/l
Lymphs	1.4 * 10 /l 9	CRP	12 mg/l
PT ratio	1.6 * 10 /l 9	aPTT	50 seconds
CT head	Large acute right-sided subdural haematoma with ventricular effacement and midline shift		

How will you manage this patient?

A. Platelet transfusion
B. Fresh frozen plasma
C. Red cell transfusion
D. Tranexamic acid
E. Idarucizumab

E. Idarucizumab

Bleeding on dabigatran? Can use idarucizumab to reverse

Dabigatran is an oral anticoagulant acting by direct inhibition of thrombin. In active bleeding, or life-threatening haemorrhage, idarucizumab should be used. This is a specific antidote which reverses the effect of dabigatran.

The PT ratio and INR is not a reliable marker following dabigatran ingestion, and should not be used to monitor the anticoagulant effects. The aPTT and, if available, the thrombin time (TT) should be used to measure the anticoagulant effect of dabigatran,

If idarucizumab is unavailable, and particularly if there is evidence of acquired coagulopathy, consider administration of red cell concentrate, fresh frozen plasma, and/or platelet transfusion.

Question 135 of 328

An 18-year-old male presents with shortness of breath. He states that it has gradually worsened over the past year. He denies cough, wheeze or chest pain. He has no past medical history of note. On examination you note a loud second heart sound. An ECG shows right bundle branch block (RBBB) with left axis deviation (LAD).

What is the most likely diagnosis?

A. Coronary sinus atrial septal defect

B. Ventricular septal defect

C. Idiopathic pulmonary hypertension

D. Chronic pulmonary emboli

E. Ostium primum atrial septal defect

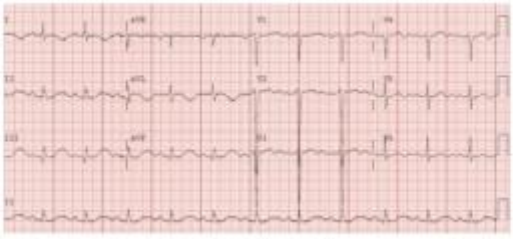
Both primum and secondum atrial septal defects result in RBBB. The axis can be of use in distinguishing between ostium primum ASDs (usually have a LAD) and ostium secondums (usually have RAD).

The patient has presented with features of pulmonary hypertension, namely the progressive shortness of breath and loud second heart sound. The ECG is very suggestive of an atrial septal defect (ASD). Atrial septal defects result in RBBB. The axis can be of use in distinguishing between ostium primum ASDs (usually have a LAD) and ostium secondums (usually have RAD). The ECG features in this case are therefore more suggestive of an ostium primum.

Furthermore, although ostium secondums are more common overall, they do not usually present as early as this. In contrast, ostium primum defects are usually much lower down in the septum and often involve the atrioventricular valves which accelerates the pathogenesis resulting in presentation at a younger age.

Question 136 of 328

A 23-year-old man with known ulcerative colitis presents for the fourth time in 6 months with a severe flare. On examination, he has a distended and generally tender abdomen. He was treated with intravenous hydrocortisone, standard 0.9% saline intravenous fluids and thromboprophylaxis. Although an abdominal film demonstrated dilated large bowel loops, no operation was deemed necessary after surgical review. The same treatment was continued for 3 days. On day 3 of his admission, he reports chest pain with palpations. A 12 lead ECG was taken:



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What is the most likely underlying diagnosis?

A. Hyperkalaemia
B. Hypokalaemia
C. Hypocalcaemia
D. Intravascular depletion → sinus tachycardia
E. Atrial fibrillation

B. Hypokalaemia

Hypokalaemia is a common consequence following extensive GI losses, in addition to bicarbonate and chloride losses. In this case, lack of potassium replacement resulted in tachyarrhythmia with U waves (see extra complex after the T wave). The patient may well be intravenously dry as well but while this may result in tachycardia, it does not explain U waves.

Question 137 of 328

A 78-year-old woman presents to the emergency department with complaints of palpitations. Her episodes of palpitations occur roughly once a month and are not associated with any chest pain or shortness of breath. She has a past medical history of ischaemic heart disease, type 2 diabetes, depression, hypertension and migraines. She takes aspirin, metformin, simvastatin and uses paracetamol when she needs them for headaches. She lives alone with a twice daily package of care.

An ECG demonstrates an irregularly irregular rhythm with no p-waves. She is offered bisoprolol to control her symptoms but does not want to take it regularly. She asks if she could take the bisoprolol only when she has palpitations as they occur so rarely.

What would make a 'pill-in-thepocket'□ strategy inappropriate for her?

A. Hypertension
B. History of ischaemic heart disease
C. Age
D. Living alone
E. Palpitations too frequent

B. History of ischaemic heart disease

The correct answer is a history of ischaemic heart disease. This patient has paroxysmal AF. NICE recommends that when people have infrequent paroxysms and few symptoms a 'pill-in-the-pocket' strategy can be offered. This means giving rate-control medications for as needed use, rather than regular use. It can be considered in those with paroxysmal AF who:

- Have no history of left ventricular dysfunction, valvular or ischaemic heart disease
- Have infrequent episodes
- Systolic blood pressure >100mmHg and heart rate >70bpm
- Are able to understand when to take the tablet

This patient could not be offered this strategy as she has a history of ischaemic heart disease.

Question 138 of 328

A 73-year-old gentleman was shown to have left ventricular dysfunction and hypertrophy on an echocardiogram post acute myocardial infarction (MI). Prior to discharge, he was started on ramipril 2.5mg, bisoprolol 2.5mg, atorvastatin 10mg and aspirin 75mg. His medical history included osteoporosis for which he took Calcichew D3 forte and alendronic acid. He did not smoke or drink alcohol. On admission his kidney function showed the following:

Sodium	136 mmol/L
Potassium	3.7 mmol/L
Urea	7.0 mmol/L
Creatinine	120 micromol/L
eGFR	64 ml/min/1.73m²

His GP checked his bloods again ten days after the initiation of his new medication to titrate the dose of ramipril as requested on the discharge letter from the cardiology team. The results are below:

Sodium	134 mmol/L
Potassium	4.2 mmol/L
Urea	8.0 mmol/L
Creatinine	156 micromol/L
eGFR	50 ml/min/1.73m²

How would you manage this gentleman's deteriorating renal function?

A. Continue the ramipril and repeat the bloods in one week
B. Decrease the dose of ramipril to 1.25mg daily
C. Stop the ramipril
D. Switch the ramipril to losartan
E. Request a renal ultrasound scan

A. Continue the ramipril and repeat the bloods in one week

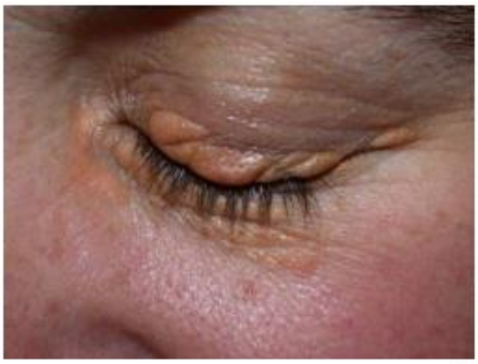
NICE guidelines are useful for everyday practice like managing chronic kidney disease (CKD). Knowing the major points about recent guidelines will also help you in your revision for exams. Guideline 73 is about the diagnosis and management of CKD, the link is below.

It is useful for staging CKD and guiding typical frequency of blood monitoring. In our gentleman's case, his renal function deteriorates due to starting ramipril. NICE guidance says it is acceptable to have a 25% reduction in eGFR OR a 30% rise in creatinine after starting an ACE inhibitor or angiotensin receptor blocker.

Question 139 of 328

A 56-year-old woman presents to her GP with discolouration around her eyes which has been present for some months. She feels very conscious about its appearance but denies any pain, itch or discomfort.

On examination, she appears well. The area in question appears as follows:



Given the likely diagnosis, which of the following would be an appropriate management option?

A. Topical emollient
B. Topical hydrocortisone
C. Topical ketoconazole
D. Topical trichloroacetic acid
E. Urgent referral to secondary care

Not answered

Question 140 of 328

A 38-year-old woman is referred to the cardiology outpatient department with exertional shortness of breath. She has no past medical history.

On examination, the jugular venous pulse is elevated and there is a loud P2 component of the second heart sound.

A transthoracic echocardiogram reveals an elevated mean pulmonary artery pressure (mPAP) of 54 mmHg, which is confirmed with a right heart catheterization (RHC). Acute vasodilator testing at the time of RHC demonstrates a decrease in mPAP to 28 mmHg.

A set of comprehensive investigations does not reveal an identifiable cause of the abnormalities.

Given her diagnosis and subsequent investigations, what class of medications are appropriate to treat her illness?

A. Calcium channel blockers
B. Endothelin receptor antagonists
C. Phosphodiesterase inhibitors
D. Prostacyclin analogues
E. Vitamin K antagonists

A. Calcium channel blockers

Pulmonary arterial hypertension patients with positive response to vasodilator testing should be treated with calcium channel blockers

Calcium channel blockers is the correct answer. The patient has pulmonary hypertension. The gold standard investigation for the investigation of pulmonary hypertension is right heart catheterization. A mean pulmonary artery pressure >= 25 mmHg at rest defines pulmonary hypertension. Patients with pHTN should undergo vasodilator testing. A positive response to vasodilator testing as evidenced by a decline in mPAP suggests the patient may benefit from a calcium channel blocker. An inability to find a secondary cause of her disease suggests she has the idiopathic form of the illness.

Endothelin receptor antagonists is incorrect. An example of this type of medication is bosentan. This class of medication is used in those patients with negative acute vasodilator testing.

Phosphodiesterase inhibitors is not the right answer. An example of this class of medication is sildenafil. It can be used orally. This class of medication is used in those patients with negative acute vasodilator testing. Examples of vasodilators include IV epoprostenol, IV adenosine, or inhaled nitric oxide.

Prostacyclin analogues is incorrect. An example of this class of medication is iloprost. This is given as an infusion. It is used in those patients with negative acute vasodilator testing.

Vitamin K antagonists is not the right answer. An example of this class of medication is warfarin. Chronic thromboembolic disease is a cause of pulmonary hypertension but we are told a comprehensive set of investigations did not reveal a cause of pulmonary hypertension and therefore anticoagulation is not indicated.

Question 141 of 328

A 68-year-old Afro-Caribbean gentleman is short of breath on dressing, washing and climbing the stairs. He has a past medical history of agerelated macular degeneration, systolic heart failure (ejection fraction on echocardiogram = 43%), osteoporosis and myelodysplasia. He is currently taking ramipril, bisoprolol and spironolactone, at the maximum tolerated doses, as well as alendronate once weekly.

Which medical therapy could further improve his prognosis?

A. Erythropoietin
B. Blood transfusion
C. Isosorbide mononitrate and hydralazine
D. Folic acid
E. Furosemide

C. Isosorbide mononitrate and hydralazine

Hydrazine and nitrate should be considered for Afro-Caribbean patients with heart failure who are not responding to ACE-inhibitor, beta-blocker and aldosterone antagonist therapy

This gentleman is symptomatic (New York Heart Association Stage III). He is fully optimised on conventional medical treatment, but NICE recommends that Afro-Caribbean patients are additionally treated with hydralazine and nitrate, to improve prognosis. Furosemide may improve his symptoms, but does not change his prognosis. This gentleman may also be symptomatic from anaemia, due to myelodysplasia, for which folic acid, erythropoietin and blood transfusion may improve his symptoms, but not his prognosis. The management of myelodysplasia in elderly patients with multiple co-morbidities is usually supportive care.

Question 142 of 328

A 56-year-old Caucasian male patient with no history of ischaemic heart disease had a non-ST elevation myocardial infarction 20 days ago and was started on aspirin, atorvastatin, bisoprolol and enalapril as an inpatient. He has been discharged to your care in the cardiology outpatients department. He presents for review 1 month later saying he remains breathless on minimal exertion (walking 50 metres) with no chest pains. There is no evidence of pulmonary oedema on examination. A recent echocardiogram shows a left ventricular ejection fraction of 40%. His beta natriuretic peptide level is 436 pg/mL (normal <100 pg/mL).

What would be most appropriate to add to his oral therapy?

A. Furosemide
B. Spironolactone
C. Hydralazine and isosorbide dinitrate (modified release)
D. Irbesartan
E. Ivabradine

NICE guidelines regarding chronic heart failure with reduced left ventricular ejection fraction:

A. Start an ACE-inhibitor and β blocker in increasing doses to maximum tolerated.

B. Second line treatment most appropriate for this Caucasian patient is spironolactone because of the recent history of myocardial infarction.

C. Furosemide has no prognostic benefit and is not indicated if there are no signs of fluid overload on examination.

D. Eplerenone is a mineralocorticoid antagonist that is specifically marketed for post-myocardial infarction patients. However, it is not available off-patent in the UK and therefore rarely first line on hospital formularies

Question 143 of 328

A 44-year-old man attends with a 2-day history of fevers on rigours. On examination, you note splinter haemorrhages and a pan systolic murmur.

Blood results are as follows:

Hb	130 g/L	Male: (135-180)Female: (115 - 160)
Platelets	380 * 10 /L 9	(150 - 400)
WBC	18.2 * 10 /L 9	(4.0 - 11.0)
Na +	126 mmol/L	(135 - 145)
K +	4.4 mmol/L	(3.5 - 5.0)
Urea	7.2 mmol/L	(2.0 - 7.0)
Creatinine	115 µmol/L	(55 - 120)
CRP	82 mg/L	(< 5)

Three sets of blood cultures are positive for the following organism:

Blood cultures	Gram +ve coagulase positive cocci

What treatment is indicated?

A. Benzylpenicillin and low-dose gentamicin

B. Flucloxacillin

C. Flucloxacillin, rifampicin and low-dose gentamicin

D. Vancomycin and low-dose gentamicin

E. Vancomycin and rifampicin

B. Flucloxacillin

IV flucloxacillin is the treatment of choice for native valve endocarditis caused by staphylococci

Flucloxacillin is correct. The positive cultures are highly suggestive of *S.aureus* infection due to the positive coagulase test result. *Streptococci* are not coagulase producing. Native valve endocarditis caused by *S.aureus* is treated with high-dose intravenous flucloxacillin.

Benzylpenicillin and low-dose gentamicin is incorrect. This combination of drugs is used to treat endocarditis caused by less sensitive *Streptococci*.

Flucloxacillin, rifampicin and low-dose gentamicin is incorrect. This combination of drugs is used to treat prosthetic valve endocarditis caused by *S.aureus*.

Vancomycin and low-dose gentamicin is incorrect. This combination of antibiotics can be used for initial blind therapy in patients with a penicillin allergy, a history of methicillin-resistant *Staphylococcus aureus* (MRSA) or severe sepsis.

Vancomycin and rifampicin is incorrect. This combination of antibiotics can be used for native valve endocarditis caused by *S.aureus* if they have a penicillin allergy or a history of MRSA.

Question 144 of 328

A 27-year-old man presents to the emergency department. He was recently discharged following an internal fixation of his left tibia. The fracture occurred due to a fall whilst intoxicated and he broke his leg as he landed on the pavement.

He has returned because he experienced a sudden onset of shortness of breath and became concerned. He has no cough or chest pain. He has no previous medical problems. His medications include codeine and paracetamol; post-operative analgesia that he was discharged on. He denies any allergies.

Observations demonstrate hypoxia with saturations at 92% on room air and are otherwise unremarkable. Blood tests, including a FBC, U&E and CRP are all within normal ranges. Clinical examination reveals a few leftsided crepitations and there is swelling and tenderness of his left calf. This was the side of his operation. A chest X-ray demonstrates a small left-sided pleural effusion causing blunting of his left costophrenic angle. No CT or US scanning is available as it is 3 AM.

What is the most appropriate next step?

A. Treatment dose apixaban
B. Wait to morning then order CTPA
C. Immediate drainage of effusion
D. Wait to morning and then US guided drainage of effusion
E. Wait to morning then order VQ scan

A. Treatment dose apixaban

Strong suspicion of PE but a delay in the scan: start on treatment dose anticoagulant meanwhile

The correct answer is treatment dose apixaban. This is a young and fit patient who has presented with sudden onset of shortness of breath with a swollen calf following immobilisation and surgery. The most likely diagnosis is a PE from a DVT. The clinical likelihood is very high and therefore immediate treatment is appropriate as the patient is hypoxic. Waiting to confirm the diagnosis should be done but it is more appropriate to start treatment first. The effusion is most likely secondary to an embolus and unlikely to be responsible for the level of hypoxia as it is small on the X-ray.

Question 145 of 328

A 68-year-old male is admitted with crushing chest pain radiating to the left arm and an ECG suggestive of anterior ST-elevation myocardial infarction. He is treated under the local primary percutaneous cardiac intervention protocol and a drug-eluting stent is successfully deployed in his left anterior descending artery. A left ventriculogram is performed during the procedure indicating severely reduced systolic function. An echocardiogram confirms this with an ejection fraction calculated at 32%.

Which rate control medication would be the most appropriate first choice in treating this gentleman?

A. Carvedilol
B. Ivabradine
C. Metoprolol
D. Atenolol
E. Diltiazem

A. Carvedilol

Carvedilol, bisoprolol and nebivolol are the B-blockers that are licensed in the United Kingdom for the treatment of heart failure

This is a patient that has recently suffered a myocardial infarction with residual reduced ejection fraction secondary to that. The beta-blockers currently licensed in the treatment of heart failure and as secondary prevention of ischaemic cardiac disease include bisoprolol, carvedilol and nebivolol.

Ivabradine has a role in the management of angina and rate-control in patients with heart failure however it does not appear to be involved in the first line management and secondary prevention of ischaemic heart disease.

Diltiazem is a rate-limiting calcium channel blocker; all calcium channel blockers (rate limiting) are contraindicated in moderate to severe heart failure.

Metoprolol has previously shown increased rate of adverse events in early use in coronary artery disease (STEMI) while atenolol is not licensed in the UK for heart failure management.

Question 146 of 328

A 62-year-old man sees his GP with complaints of leg pain. Over the last few months, he notices a crampy uncomfortable feeling in the back of both of his calves when he walks to the shops. The discomfort can be so bad that he has to stop and rest for a few minutes after which he notices that his symptoms are improved. On occasions, he also gets the symptoms when he is shopping in the supermarket. He attributes this to overexerting himself and tries to rest on his shopping trolley while walking but does not seem to help. He has a 40-pack year smoking history and takes amlodipine for his blood pressure, and paracetamol and ibuprofen for lower back pain that has troubled him for years.

Physical examination reveals mild atrophy of his thigh and calf muscles bilaterally, in addition to shiny pale skin with significant hair loss throughout his lower limbs. His pedal pulses are bilaterally impalpable, and popliteal pulses are faint. Power in both lower limbs is normal throughout all movements, and he has normal patellar reflexes bilaterally and absent ankle reflexes. His Babinski reflex is downgoing on the left side and equivocal on the right side. A recently obtained ankle brachial pressure index test yielded a result of 0.70 on the right side and 0.95 on the left side. X-rays of his lumbar spine show evidence of joint space narrowing and osteophytes.

Which one of the following is the next best step in the management of this patient?

A. MRI scan of lumbar spine
B. Check his HbA1c to screen for diabetes
C. Refer to vascular surgery for consideration of peripheral arterialstenting or bypass surgery
D. Starting him on duloxetine to manage his pain
E. Screen for coronary artery disease with ECG stress testing

C. Refer to vascular surgery for consideration of peripheral arterialstenting or bypass surgery

In patients with cardiovascular risk factors and symptoms suggestive of claudication with an equivocal/borderline ankle brachial pressure index study result, the next best study is an ankle brachial pressure index after exercise

This gentleman has symptoms of claudication, in particular, vascular claudication. Although he does have a history of chronic back pain for which he takes analgesia, he is less likely to have neurogenic claudication compared to vascular claudication. An important discriminating factor between neurogenic claudication and vascular claudication is that patients with neurogenic claudication have symptoms on exertion that improve with manoeuvers such as leaning forward, and sitting down, whereas vascular claudication does not change with these manoeuvers and only improves with rest. In addition, his physical exam shows evidence of peripheral arterial disease characterised with muscle atrophy, shiny skin with hair loss and impalpable pedal pulses. Absent ankle jerk reflexes and equivocal Babinski reflexes can be normal variants and do not always indicate neurological pathology. This patient has rightly already undergone an ankle brachial pressure index assessment, and the results indicate a diagnosis of peripheral arterial disease. He is functionally impaired by his symptoms and so the next best step in his management would be to refer him to vascular surgery for consideration of treatment strategies which may include percutaneous interventions with stenting and/or surgical bypass.

Although this patient has evidence of degenerative joint disease on his lumbar spine x-ray, his clinical presentation is not consistent with neurogenic claudication and so an MRI scan of his lumbar spine is not indicated.

Although it is important to screen for and aggressively manage cardiovascular risk factors in patients with peripheral arterial disease, this in itself would not address the patient's symptoms or the disease course.

This patient's pain is most consistent with vascular claudication as opposed to neuropathic pain, for which an agent like duloxetine would be appropriate.

Although individuals with peripheral arterial disease have a significant likelihood of having concomitant coronary disease, this patient is not complaining of angina or symptoms suggestive of coronary disease and so screening for this is not indicated. The next best step in managing this patient is to address and treat his symptoms related to his peripheral arterial disease.

Question 147 of 328

A 43-year-old man presents with palpitations to the emergency department. His ECG demonstrates a broad complex tachycardia at 180 beats/min, suggestive of ventricular tachycardia. His blood pressure is 134/80 mmHg, he is uncomfortable but alert. The emergency department doctor has administered a 300mg bolus of amiodarone and the consultant subsequently inserted a central venous line to continue amiodarone treatment with a 900mg infusion over 24 hours. He transiently settles but on the second day of admission, his cardiac monitor demonstrates persistent VT at 180 beats per minute again. His blood pressure is 140/75 mmHg.

What is your treatment?

A. answerNo treatment
B. Repeat bolus of amiodarone 300mg
C. DC cardioversion
D. Intravenous lidocaine infusion
E. Emergency VT ablation

D. Intravenous lidocaine infusion

The patient has entered refractory VT, continuing despite a therapeutic dose of amiodarone and sufficient loading. It is unlikely further boluses of amiodarone will produce additional therapeutic effects. Similarly, no treatment is not an option, his myocardium is unlikely to continue tolerating such a persistent tachycardia without soon reducing left ventricular output and arrest. He is not haemodynamically unstable and hence does not justify DC cardioversion as an emergency procedure. Similarly, emergency VT ablation is not appropriate after only 1st line treatment. Intravenous lidocaine is the second line treatment: first as a bolus, followed by an infusion regime over 6.5 h

Question 148 of 328

A 25-year-old female presents with palpations and dizziness. She states that she has had a few episodes similar to this in the past and each time they have spontaneously resolved. On examination she has a blood pressure of 85/65 mmHg and a heart rate of 180bpm. An ECG shows an irregularly irregular rhythm, no identifiable P waves, and a QRS of 135ms.

Your senior decides to electrically cardiovert her. While the cardioversion is being prepared you instruct the patient to perform a vagal manoeuvre. Shortly after this she loses consciousness and goes into cardiac arrest.

What is the most likely underlying disorder?

A. Torsades de Pointes
B. Atrial fibrillation
C. Atrioventricular nodal re-entry tachycardia (AVNRT)
D. Monomorphic ventricular tachycardia
E. Wolff-Parkinson-White syndrome (WPW)

E. Wolff-Parkinson-White syndrome (WPW)

AV blocking drugs and vagal manoeuvres are absolutely contraindicated in patients with AF and pre-excitation

There are few arrhythmias, which can cause an irregularly irregular broad complex tachycardia. These include irregular atrial arrhythmias with bundle branch block (e.g. atrial fibrillation), and polymorphic ventricular tachycardia (including Torsades de Pointes). Another important group of conditions to be aware of include the pre-excitation syndromes.

Pre-excitation refers to early activation of the ventricles due to impulses bypassing the AV node via an accessory pathway. This results in a broad QRS complex e.g. the slurred upstroke of QRS (delta wave) as is seen in WPW. When present with atrial fibrillation, the accessory pathway allows for rapid conduction directly to the ventricles bypassing the AV node. This results in an irregularly irregular broad complex tachycardia.

The Valsalva manoeuvre increases the vagal tone resulting in AV node block. This promotes conduction of the atrial signals through the accessory pathway with a resultant increase in ventricular rate and possible degeneration into VT or VF, as is seen in this case.

AV blocking drugs and vagal manoeuvres are absolutely contraindicated in patients with AF and pre-excitation. In a haemodynamically unstable patient, urgent synchronised DC cardioversion is required. Medical treatment options in a stable patient include procainamide, although DC cardioversion may be preferred. Procainamide is a class 1 anti-arrhythmic which preferentially blocks the accessory pathway.

Question 149 of 328

A 67-year-old woman is reviewed in cardiology clinic following a GP referral. A recent transthoracic echocardiogram demonstrated severe aortic stenosis with an aortic valve at 0.7cm2 and a mean pressure gradient of 62mmHg and a left ventricular ejection fraction of 43%. Pulmonary arterial pressure is normal. She has a chest X-ray two months ago which was normal. She denies any symptoms of shortness of breath, chest pain or lightheadedness.

She is normally fit and well with a past medical history of hypothyroidism, two caesarian sections, osteoarthritis and migraines. She is a non-smoker.

What is the most appropriate management plan?

A. Review in six months
B. Cardiac MRI
C. Exercise test
D. Aortic valve replacement
E. Transcatheter aortic valve implantation (TAVI)

D. Aortic valve replacement

The correct answer is aortic valve replacement. This is an asymptomatic patient with severe stenosis but she has an ejection fraction of less than 50%. This means that she should be referred for aortic valve replacement or TAVI if unsuitable. Exercise testing would be recommended if her ejection fraction was greater than 50%. Cardiac MRI would not be helpful in this instance. If she had an ejection fraction greater than 50% and passed exercise testing then she could be reviewed in six months

Question 150 of 328

A 62-year-old woman is reviewed prior to discharge by the medical team. She was admitted with a lower respiratory tract infection. She now feels well and is ready for discharge. She has a background of type two diabetes mellitus, hypertension, hypothyroidism, osteoarthritis and dementia. Her medication history includes metformin 500mg BD, ramipril 2.5mg OD, levothyroxine 75 mcg OD and paracetamol 500mg QDS PRN. On the first two days in hospital, her metformin was stopped, but her other medications continued as in the community during her admission.

It is noted that she has persistently high blood pressure despite being confirmed as taking ramipril.

What is the most appropriate plan to control her hypertension?

A. Increase the dose of ramipril and check U&Es within 1-2 weeks
B. Increase the dose of ramipril and check U&Es within 3-4 weeks
C. Add amlodipine, increase the dose of ramipril and check U&Eswithin 3-4 weeks
D. Add on amlodipine at a low dose
E. Stop ramipril and start amlodipine instead

A. Increase the dose of ramipril and check U&Es within 1-2 weeks

The correct answer is to increase the dose of ramipril and to check U&Es within 1-2 weeks. The patient is on a low dose of ramipril and there is room to increase it, and this should be done prior to adding on a second anti-hypertensive. It is important to test renal function within two weeks of either starting or increasing the dose of an ACE inhibitor to exclude deteriorating renal function, which would need to be investigated promptly. Amlodipine would have been a more appropriate antihypertensive choice if she did not have diabetes.

Question 151 of 328

A 56-year-old gentleman is admitted to Emergency Department with back pain. His past medical history included type 2 diabetes and hypertension. Initial observations are as follows: heart rate 112 beats per minute, blood pressure 155/82 mmHg, respiratory rate 26/min, oxygen saturations 95% on 2 litres oxygen via nasal cannula, temperature 37.2ºC.

Examination demonstrates muffled heart sounds I and II. JVP is not elevated and there is no peripheral oedema. Auscultation of the chest is clear with no crackles or wheeze and good air entry bilaterally. An ECG confirms sinus tachycardia with a heart rate of 102 beats per minute and 2mm inferior ST depression. Portable chest x-ray shows poor inspiratory effort with cardiomegaly and clear lung fields.

What single investigation would you arrange next?

A. CT aortogram
B. CT pulmonary angiogram (CTPA)
C. Ventilation/Perfusion scan (V/Q scan)
D. Echocardiogram
E. 12 hour troponin

A. CT aortogram

This patient has clinical features suggestive of type A aortic dissection, and an urgent CT aortogram would be required to exclude this diagnosis. Patients often describe back pain as the aorta is a retroperitoneal structure. A major risk factor is hypertension. The Stanford classification outlines type A dissections which involves the ascending aorta and type B dissections which originate in the descending aorta. Proximal extension of type A dissections can result in rupture of the coronary sinus and secondary ischaemia. They may be associated with pericardial effusions, which often manifests as cardiomegaly on a plain chest x-ray and results in muffled heart sounds. Management of type A dissections involves urgent referral to a cardiothoracic surgical unit

Question 152 of 328

A 65-year-old man is reviewed in the outpatient cardiology clinic and reports palpitations and fatigue when carrying out normal household tasks. He has a history of heart failure (with an ejection fraction of 41%) and chronic obstructive pulmonary disease. He takes bisoprolol 10mg once daily, ramipril 10mg once daily and uses a tiotropium bromide inhaler.

Observations:

- Heart rate 88 beats per minute
- Blood pressure 145/82 mmHg
- Respiratory rate 18/minute
- Oxygen saturations 96% on room air (95% on exertion)

On examination, the pulse is regular.

What is the most appropriate next step in the patient's management?

A. Add a long-acting beta agonist and inhaled corticosteroid
B. Add digoxin
C. Add ivabradine
D. Add spironolactone
E. Increase the dose of bisoprolol

D. Add spironolactone

Offer a mineralcorticoid receptor antagonist, in addition to an ACE inhibitor (or ARB) and beta-blocker, to people who have heart failure with reduced ejection fraction if they continue to have symptoms of heart failure

Add spironolactone is correct. The patient reports palpitations and fatigue on minimal exertion. These symptoms suggest symptomatic heart failure rather than worsening COPD. This is reaffirmed by the normal SpO2 results following exertion. In a patient that is on maximally tolerated doses of beta-blocker and an ACE inhibitor, the correct next step is to add spironolactone.

Add a long-acting beta-agonist and inhaled corticosteroid is incorrect. Symptoms of palpitations and fatigue are more suggestive of symptomatic heart failure rather than COPD where wheezing and shortness of breath would be more typical. Therefore adjusting his COPD inhaler regimen is incorrect.

Add digoxin is incorrect. Digoxin is indicated in the management of symptomatic heart failure (especially in the context of co-existent atrial fibrillation) but only after other medications such as spironolactone have been initially trialled. This patient is not in AF.

Add ivabradine is incorrect. This is a 3rd line option for those patients with an ejection fraction < 35% and a heart rate > 75.

Increase the dose of bisoprolol is incorrect. 10mg is the maximum dose of bisoprolol that is recommended for heart failure.

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A 24-year-old female is brought into the emergency department by ambulance. She has a family history of Wolff-Parkinson-White syndrome and was binge drinking last night. She is complaining of palpitations which she says started suddenly 1 hour ago.

Her observations are as follows

Heart rate 180 bpm

Blood pressure 100/60 mmHg Saturations 98% on air Respiratory rate 26/min Temperature 36.8ºC

An ECG (electrocardiogram) is done which shows pre-excited atrial fibrillation (AF)

Which of the following is an appropriate treatment?

A. Adenosine
B. Atenolol
C. Flecainide
D. Diltiazem
E. Digoxin

C. Flecainide

In pre-excited AF don't give anything that blocks conduction at AV node (including calcium channel blockers, adenosine or digoxin) as this can cause ventricular tachycardia

Flecainide is the only appropriate treatment on this list

All of the other options are drugs which block transmission through the

AV (atrioventricular) node. Giving these drugs in pre-excited AF (atrial fibrillation) can precipitate VT (ventricular tachycardia) or VF (ventricular fibrillation).

Note that flecainide is contraindicated in patients with structural heart abnormalities. In this situation then amiodarone can be given instead.

If any of the following are present: hypotension, signs of shock, altered mental status, chest pain or acute heart failure then synchronised cardioversion should be used rather than medication.

Question 154 of 328

A 48-year-old female presents with chest pain. She describes the pain as central, dull and tight. She has associated nausea. She has no past medical history and takes no regular medicines.

An ECG is performed:

P waves	Normal morphology
PR interval	130ms
QRS	115ms
ST segments	ST elevation in V1-V3

Blood results are as follows:

Troponin (on admission)	< 0.015 µg/l (normal < 0.015)
Troponin (6 hours later)	< 0.015 µg/l (normal < 0.015)

What is the most likely diagnosis?

A. answerAcute STEMI
B. Acute NSTEMI
C. Unstable angina
D. Stable angina
E. Prinzmetal's angina (variant angina)

E. Prinzmetal's angina (variant angina)

In coronary vasospasm (Prinzmetal's angina, or variant angina), the ECG shows ST elevation that is very similar to an acute STEMI. However, unlike acute STEMI the ECG changes are transient, reversible with vasodilators and not associated with myocardial necrosis

The presence of ST elevation and chest pain should alert you to the diagnosis of acute STEMI. However importantly the serial troponins are negative effectively ruling out myocardial infarction. In coronary vasospasm (Prinzmetal's angina, or variant angina), the ECG shows ST elevation that is very similar to an acute STEMI. However, unlike acute STEMI the ECG changes are transient, reversible with vasodilators and not associated with myocardial necrosis. Prinzmetal's angina is therefore the most likely diagnosis.

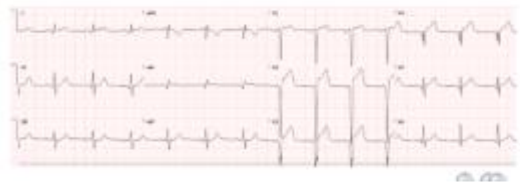
Acute NSTEMI is excluded by the normal serial troponins.

Angina pectoris (stable or unstable) is of course a possibility. However the presence of ST elevation, the relatively young age of the patient, and the absence of cardiovascular risk factors, makes Prinzmetal's angina the more likely diagnosis.

The patient will need coronary angiography. The gold standard test for diagnosing variant angina is to visualise the coronary arteries by angiography before and after injection of a provocative agent such as acetylcholine to precipitate an attack of vasospasm.

Question 155 of 328

A 72-year-old man arrives at the emergency room complaining of chest pain for six hours. It is a crushing pain at the centre of his chest, which radiates to the neck. He was diagnosed with Grave's disease previously, which was treated with radioiodine ablation, following which he developed hypothyroidism. He suffered an ischemic stroke two months ago. His medications include aspirin, atorvastatin and thyroxine. His heart rate is 83 beats/min, BP 130/80 mmHg, respiratory rate 20 breaths/min, and oxygen saturation is 98%. An ECG is taken:



What is the most appropriate next step in managing this patient?

A. Give aspirin and arrange for an emergency CRP, troponin level and ECHO
B. Give aspirin and arrange for an emergency troponin level andECHO
C. Give aspirin and glyceryl trinitrate and arrange for an emergency troponin level
D. Give aspirin and glyceryl trinitrate, and prepare for urgent PCI
E. Give aspirin and glyceryl trinitrate, and prepare for urgentfibrinolysis

D. Give aspirin and glyceryl trinitrate, and prepare for urgent PCI

The most appropriate next step is to **give aspirin and glyceryl trinitrate and plan for PCI**. The ECG in this scenario indicates a STEMI, with ST elevation observable in leads V1 to V5, which typically indicates an occlusion of the LAD. It is important to note that ECG manifestations of STEMI can vary depending on the timing post-ischemia onset. Initially, hyperacute T waves may be observed, followed by ST-segment elevation. As the condition evolves over several hours to days, T wave inversions and Q waves tend to appear within the same leads. Prompt primary PCI is essential for this patient in order to achieve reperfusion as soon as possible. Detailed management guidelines for acute coronary syndromes such as STEMI, NSTEMI, and unstable angina are outlined in the accompanying notes.

Give aspirin and arrange for an emergency CRP, troponin level, and ECHO is incorrect. Acute pericarditis is a close mimic of STEMI. However, it can be differentiated based on clinical features and ECG findings. Clinically, patients with pericarditis present with a sudden onset of a sharp pain over the anterior chest wall, similar to pleuritic pain, which increases on inspiration and decreases on leaning forwards. ECG findings include global ST elevations and PR-segment depressions in most limb leads (I, II, III, aVL, aVF) and precordial leads (V2 - V6). Initial investigations for pericarditis include CRP to check for infection, an ECHO scan to look for associated pericardial effusion and a troponin level. However, the history and ECG in this case do not suggest pericarditis.

Give aspirin and arrange for an emergency troponin level and ECHO

is not appropriate in this scenario. This patient has typical features of STEMI in the ECG and should be taken up for PCI immediately. Troponin level and ECHO will be requested in all cases of myocardial infarction, but they will take time. Given that the patient has a STEMI, preparing for PCI is the most appropriate strategy here.

Giving aspirin and glyceryl trinitrate and arranging for an emergency troponin level would be misguided. In instances where STEMI has been identified by ECG findings'as it has been in this case'immediate reperfusion strategies such as PCI or fibrinolysis are warranted without delay. Troponin levels might still fall within normal ranges early after an acute coronary syndrome has occurred; therefore, they should not be solely relied upon for diagnosing STEMI.

Giving aspirin and glyceryl trinitrate and preparing for urgent fibrinolysis would be contraindicated due to the patient's recent history of ischemic stroke within the past three months. Fibrinolysis could potentially be considered for patients with no contraindications when they present with STEMI within 12 hours from symptom onset; however, it is only recommended if primary PCI is not accessible at their presenting hospital or if transfer to another facility offering PCI services cannot occur within 120 minutes.

Question 156 of 328

You review a 65 year-old male who was admitted yesterday. He is a diabetic gentleman who was admitted with a non-ST elevation myocardial infarction and subsequent flash pulmonary oedema. He has been treated with aspirin, clopidogrel, fondaparinux and intravenous furosemide. He is achieving a good diuresis - producing 100mls of urine per hour.

You have been asked to see him as he continues to have significant breathlessness but no ongoing chest pain. His blood pressure is 92/87mmHg and oxygen saturations are 83% on 65% humidified oxygen. He has bibasal crepitations and JVP is raised. His ABG results are as follows:

pH	7.32
pCO2	4.6kPa
pO2	7.9kPa

What would be your next clinical intervention?

A. Start non-invasive ventilation
B. Start continuous positive airway pressure ventilation (CPAP)
C. Refer for angiography and primary coronary intervention
D. Start a tirofiban infusion
E. Start a GTN infusion

B. Start continuous positive airway pressure ventilation (CPAP)

Acute heart failure not responding to treatment - consider CPAP

This gentleman has had a non-ST elevation myocardial infarction (NSTEMI) and is now struggling with hypoxia secondary to pulmonary oedema. He has been treated optimally for his NSTEMI and is now pain free. He is diuresing well but continues to be hypoxic with signs of ongoing pulmonary oedema. The next logical intervention would be to try CPAP to improve his oxygenation. GTN, tirofiban and angiography would all look to improve coronary perfusion, however the gentleman is pain free suggesting this is not a priority.

Question 157 of 328

A 72-year-old man attends cardiology clinic for routine follow-up of ischaemic heart disease. The patient had suffered a non-ST elevation myocardial infarction the previous year treated with a drug eluting stent (see below angiogram report). The patient reported being generally well over previous months although did experience retro-sternal chest pain on exertion, typically on walking up hills or climbing stairs. The patient denied any episodes of pain at rest and did not feel his symptoms were worsening. The patients regular medications were aspirin 75 mg daily, clopidogrel 75 mg daily, atorvastatin 80 mg daily and ramipril 5 mg daily. As required medications were sildenafil and a nitrate spray (which he reported as giving effective relief from his chest pain episodes).

Clinical examination did not demonstrate any evidence of cardiac failure. Blood pressure in clinic was 125 / 75 mmHg.

Results of investigations conducted 1 year previously following presentation with NSTEMI are included below.

Angiography: 90 % stenosis to left anterior descending artery treated with drug-eluting stent; 60 % stenosis to mid-right coronary artery; minor disease to other vessels.

Transthoracic echocardiogram: valvular function unremarkable; mild hypokinesis to anterior myocardium; overall ejection fraction approximately 40-45 %.

Electrocardiogram: sinus rhythm at 75 beats per minute; normal axis; normal QRS; no significant ST changes; inverted T waves in V3-V5.

What is most appropriate next line therapy for the patients chest pain?

A. Amlodipine
B. Bisoprolol
C. Percutaneous coronary intervention
D. Long-acting isosorbide mononitrate
E. Nicorandil

B. Bisoprolol

The patients reported symptoms are consistent with a diagnosis of stable angina secondary to known ischaemic heart disease. Medical treatment for symptomatic relief is the appropriate next line therapy. In this patient with a history of NSTEMI and controlled blood pressure a beta-blocker is preferred over a calcium-channel blocker such as amlodipine.

Both ISMN and nicorandil are second-line options for medical therapy after intolerance or failure of beta-blocker and calcium-channel blocker therapy. Such both medications induce systemic vasodilatation they would be contraindicated in this patient while he still was taking sildenafil.

Revascularisation techniques such as PCI have not been show to reduce mortality or rate of MI in stable coronary artery disease. Therefore, medical therapy should be attempted before consideration of invasive treatment.

Question 158 of 328

A 74-year-old man presents to the emergency department with a collapse. He has a past medical history of hypertension and atrial fibrillation. His medications include amlodipine and apixaban. A recent 24-hour tape arranged because of episodes of pre-syncope demonstrates sustained atrial fibrillation with episodes of bradycardia (minimum heart rate 20 beats per minute) associated with symptoms of pre-syncope.

The admission ECG demonstrates slow atrial fibrillation with a heart rate of 24 beats per minute.

The patient has recovered by the time he has been assessed by the medical team and his Glasgow coma scale is 15/15 and his heart rate is 82 beats per minute.

A decision is made to insert a permanent pacemaker.

What is the appropriate mode to programme the pacing system?

A. AAI
B. AOO
C. OOO
D. VAT
E. VVI

E. VVI

VVI or VVR pacemakers are useful for pure sustained AF. Since the atria are not functioning they do not require sensing or pacing

VVI is the correct pacing mode. In this mode, the ventricle is sensed and the ventricle is paced. If a QRS complex is detected, it doesn't fire. If it is not detected i.e. there has been no ventricular electrical activity within the interval specified by its set rate, then it will fire. This is suitable for atrial fibrillation as the atria are not working correctly and there will be no detectable P wave and therefore the atria do not need to be sensed or paced.

AAI is not the correct pacing mode to choose. AAI pacing mode means atrial paced, atrial sensed, and inhibition of pacing output in response to an atrial sensed event (P wave). Since the atria are not functioning in AF, they do not require sensing or pacing. AAI pacing has better hemodynamic features than dual-chamber pacing and is the best mode for patients with sick sinus syndrome without a significant AV conduction disorder. This mode essentially relies on the AV node to fire and guarantees a certain atrial rate.

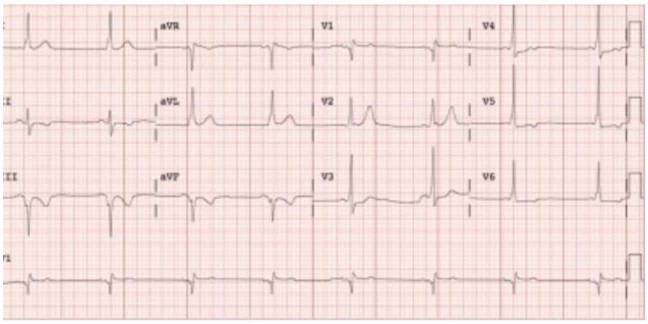
AOO is incorrect. This is asynchronous pacing. This refers to the continuous pacing of the atria irrespective of the presence or absence of an intrinsic rhythm. This pacing mode is limited to rare cases, such as when pacemaker-dependent patients (who do not have a ventricular paced setting) are subject to artefact (e.g., electrocautery during surgery), which could result in asystole due to oversensing and pacing inhibition if the pacemaker has been set to a non-asynchronous mode. It is never a permanent mode.

OOO is incorrect. This essentially means there is no sensing, no pacing and no response to sensing. It means the pacemaker is turned off.

VAT is incorrect. This mode means the atria are sensed and the ventricles are paced. This is suitable for a patient with a dysfunctional AV node but working atria. For example, in someone with a complete heart block and a normal atrial rate, a VAT pacemaker will sense the atrium and pace the ventricle in response.

Question 159 of 328

A 35-year-old man is investigated for recurrent palpitations associated with pre-syncopal symptoms and dyspnoea. Blood tests are unremarkable and a resting 12 lead ECG is shown below:



Given the likely diagnosis it is decided that radiofrequency ablation is the most appropriate treatment. What part of the heart should be ablated?

A. Sinoatrial node
B. Atrioventricular node
C. Junction of right atrium and right ventricle
D. Left atrial appendage
E. Junction of left atrium and left ventricle

C. Junction of right atrium and right ventricle

The ECG shows a short PR interval associated with a slurred upstroke (delta wave). Note the non-specific ST-T changes which are common in WPW and may be mistaken for ischaemia. The left axis deviation means that this is type B WPW, implying a right-sided pathway.

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A 34-year-old male presents to the emergency department with chest tightness worse on inspiration. He informs you that over the past 3 days he has had a low-grade temperature and a degree of myalgia. Prior to all this he had no limitations to his activity and never experienced any chest tightness. He was given glyceryl trinitrate spray and aspirin in the ambulance, neither of which have made any difference.

Examination is unremarkable, however you note that his temperature is 37.4ºC. He is a smoker and he informs you that his father, mother and paternal uncle all have had heart attacks around the age of 60. His bloods are the following:

Hb	165 g/L	Male: (135-180)Female: (115 - 160)
Platelets	537 * 10 /L 9	(150 - 400)
WBC	12.3 * 10 /L 9	(4.0 - 11.0)
Na +	138 mmol/L	(135 - 145)
K +	4.3 mmol/L	(3.5 - 5.0)
Urea	4.6 mmol/L	(2.0 - 7.0)
Creatinine	93 μmol/L	(55 - 120)
CRP	34 mg/L	(< 5)
Troponin T (0hrs)	586 ng/L	(<14)
Troponin T (3hrs)	615 ng/L	(<14)
D-dimer	Negative	

An ECG is performed indicating sinus rhythm, no intraventricular delay and widespread ST elevations; you also notice was subtle PR depression.

What treatment should be initiated in the case of this gentleman?

A. Arrange urgent angioplasty and potential revascularisation
B. Load patient with aspirin and clopidogrel
C. Load patient with aspirin and ticagrelor
D. Prescribe ibuprofen TDS
E. Prescribe ibuprofen TDS and colchicine BD

E. Prescribe ibuprofen TDS and colchicine BD

First line management of acute pericarditis involves combination of NSAID and colchicine

The history, examination and blood investigations are consistent with acute pericarditis/myopericarditis. The high troponin results indicate myocardial injury which in this case can be attributed to myopericarditis. Troponin is a marker of myocardial injury which can result due to primary ischaemia (plaque rupture or intraluminal coronary artery thrombus), ischaemia secondary to misbalance of supply and demand (tachy/bradyarrhythmias, aortic dissection or severe aortic valve disease, sepsis, severe anaemia or respiratory failure etc.) and myocardial injury not necessarily associated to ischaemia or multifactorial (such as myopericarditis, severe pulmonary embolism with right heart strain, cardiac stunning, cardiac surgery or intervention etc.) There is no strong, dynamic rise in the already high troponin level.

The fact that he is a smoker undeniably puts this gentleman at a higher risk of coronary event, however his blood tests, ECG and history point further to the direction of pericarditis. Given this, options 1, 2 and 3 which are associated with the management of acute coronary syndrome (ACS) are wrong.

The current guidelines as per the European Society of cardiology state that the treatment of choice in such patients is a combination of nonsteroidal anti-inflammatory drugs (e.g. ibuprofen) and colchicine (option 5) and do not advocate the use of a single agent, such as ibuprofen (option 4)

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A 55-year-old woman presents to the emergency department with chest pain. She is currently pain free, but has had two similar episodes recently lasting approximately 2-3 minutes. She describes the pain as squeezing and central and feels that the pain is brought on by exertion and relieved within a few minutes by resting. Her father died of a heart attack at 72, she has never smoked and has no significant medical history.

On examination, she is slim, her blood pressure is 125/75 mmHg and heart rate is 85 beats per minute, no murmurs are heard and chest sounds are normal. A 12-lead ECG shows normal sinus rhythm.

serum cholesterol	3.2 mmol/L (<5.2)
serum LDL cholesterol	2.0 mmol/L (<3.36)
serum HDL cholesterol	2.65 mmol/L (>1.55)
fasting serum triglycerides	1.0 mmol/L (0.45-1.69)
haemoglobin	125 g/L (115-165)

What is the next most appropriate investigation?

A. Invasive coronary angiography
B. CT coronary angiography
C. Exercise tolerance test
D. Myocardial perfusion scintigraphy with single photon emissioncomputed tomography
E. Stress echocardiogram

B. CT coronary angiography

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A 65-year-old man presents to his general practitioner with exertional chest tightness. This occurs stereotypically after walking approximately 100 meters. It stops when he rests. He has a past medical history of hypertension and asthma. His medications include aspirin, atorvastatin, amlodipine, and a regular inhaler.

The examination is unremarkable. His blood pressure is 154/42 mmHg.

What is the most appropriate choice of medication to control his symptoms?

A. Atenolol
B. Bisoprolol
C. Diltiazem
D. Ramipril
E. Ranolazine

E. Ranolazine

For a patient with symptomatic stable angina on a calcium channel blocker but with a contraindication to a beta-blocker, the next line treatment should be long-acting nitrate, ivabradine, nicorandil or ranolazine

Ranolazine is correct. The patient has symptomatic stable angina. A beta blocker would be an obvious choice to add at this point but is contraindicated in this patient due to their history of asthma. It may precipitate bronchospasm due to the effect on beta-adrenoreceptors. The mechanism of action of ranolazine is largely unknown. Ranolazine may have antianginal effects by inhibition of the late sodium current in cardiac myocytes.

Atenolol is incorrect. Beta-blockers are contraindicated in this patient due to the history of asthma.

Bisoprolol is incorrect. This is another beta-blocker. It is more cardioselective than atenolol but still would not be a prudent choice for a patient with asthma when there are alternatives available.

Diltiazem is incorrect. This is another calcium-channel blocker. It should not be used together with amlodipine, which is also a calcium channel blocker. Diltiazem is a non-dihydropyridine calcium channel blocker while amlodipine is a dihydropyridine calcium channel blocker. Dihydropyridine calcium channel antagonists cause predominantly peripheral vasodilatory actions. Nondihydropyridine CCBs have sinoatrial (SA) and AV node depressant effects with lesser amounts of peripheral vasodilation.

Ramipril is incorrect. This has predominantly anti-hypertensive rather than anti-anginal effects. The blood pressure in this patient is elevated but remains at stage 1 grade. Additionally, we were asked to choose a medication to help his *symptoms* rather than control his blood pressure.

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A 16-year-old female presents with a swollen knee. She states that she has had several similar episodes previously. She also complains of excessive bleeding after dental work. She states that her father has a bleeding disorder. On examination her right knee is hot and swollen. A knee aspirate reveals a bloody effusion.

Blood results are as follows:

Hb	110 g/l
Platelets	682 * 10 /l9
Prothrombin time (PT)	12 s (normal 10-13)
activated partial thromboplastin time (aPTT)	54 s (normal 25-36)
Factor VIII level	2% of normal (very low)
Factor IX level	Normal
von Willebrand Factor level	Normal

What disorder does she most likely have?

A. Down syndrome
B. Edwards syndrome
C. Marfan syndrome
D. Ehlers-Danlos syndrome
E. Turner's syndrome

E. Turner's syndrome

Females with Turner's syndrome have just one X chromosome. Therefore they have the same probability of being affected by an X linked recessive disease as males

The patient clearly has haemophilia A as indicated by the prolonged aPTT and severely reduced factor VIII levels. Rare cases of females with severe haemophilia can occur due to extreme lyonization, homozygosity, mosaicism, or Turner syndrome.

Females with Turner's syndrome have just one X chromosome. Thus they have the same probability of being affected by an X linked recessive disease as males. For the aforementioned reason, Turner's syndrome is the most likely underlying diagnosis out of the given options.

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A 68-year-old man is referred to the cardiology outpatient department with shortness of breath on exertion. He becomes breathless when ascending stairs. There is no chest pain. He has a past medical history of ischaemic heart disease and heart failure with reduced ejection fraction (30%). He is on aspirin, bisoprolol, ramipril, spironolactone, atorvastatin and lansoprazole. He does not smoke or drink alcohol.

His observations are heart rate 83 beats per minute, blood pressure 110/85 mmHg, respiratory rate 18/minute, oxygen saturations 97% on room air and temperature 37.1ºC.

On examination, he is euvolemic. His heart sounds are normal and there is no peripheral oedema. Chest auscultation is normal. The pulse is regular.

From the options listed, what is the best choice of medication to improve his symptoms?

A. Flecainide
B. Ivabradine
C. Nicorandil
D. Ranolazine
E. Verapamil

B. Ivabradine

Ivabradine should be considered in heart failure if the patient has sinus rhythm > 75/min and a LVEF < 35% and have not responded to to ACE-inhibitor, beta-blocker and aldosterone antagonist therapy

Ivabradine is the correct answer. This man has NYHA class II/III heart failure despite treatment with an ace inhibitor, beta-blocker and mineralocorticoid receptor antagonist. Ivabradine is an option in heart failure for those patients who have not responded to the previously described combination of medications and have a heart rate > 75 beats per minute and an LVEF < 35%.

Nicorandil and ranolazine are incorrect. These medications are options for the treatment of angina, not heart failure.

Flecainide is incorrect. This is a medication used to chemically cardiovert atrial fibrillation. This patient is in sinus rhythm.

Verapamil is incorrect. Negatively inotropic calcium channel blockers should be avoided in heart failure, especially in the context of current beta blocker treatment

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A 102-year-old man with a previous history of heart failure, amyloid disease and a permanent pacemaker attends the emergency department due to a syncopal episode. He recalls passing out in his chair whilst watching TV and was attended to by nursing home staff who were able to rouse him after 2 minutes of loss of consciousness. This is the third episode in the past week.

On examination, his heart rate is 32 beats per minute and irregular. He has crepitations in his chest and a raised JVP but no leg oedema. He is alert but cool peripherally. He has no pain at present.

Na +	135 mmol/l
K +	4.5 mmol/l
Urea	5.6 mmol/l
Creatinine	76 µmol/l
ECG	p-waves present with no proceeding QRS, widened QRS complexes seen
Chest xray	chronic lower zone changes, pacemaker leads have a necked appearance

What is the likely diagnosis?

A. Lead fracture
B. Lead displacement
C. Twiddlers syndrome
D. Silent myocardial infarction
E. Progression of cardiomyopathy

A. Lead fracture

Early signs of pacemaker lead failure are a 'necked' appeared on imaging

This gentleman has complete heart block despite a permanent pacemaker in situ. Cardiomyopathy progression and silent myocardial infarction would be detected by progression of congestive cardiac failure with worsening pulmonary or peripheral oedema. The presence of complete heart block suggests that the pacemaker may not be functioning correctly. It is noted that the leads have a necked appearance and this occurs after repeat bending of the leads that causes them to weaken and eventually fracture. This is a rare complication.

Despite being intact on chest x-ray. the necking suggests that the wires are starting to fray and function can be lost prior to a complete fracture of the lead is seen. Twiddling of the wires or lead displacement would be seen on chest x-ray.

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A 78-year-old gentleman is seen in clinic with long-standing heart failure with reduced ejection fraction (28%). He has had numerous admissions this year with heart failure decompensation and is wondering if there is anything else that you can do for him. You review his ECG, which is in sinus rhythm with a heart rate of 58/min with a QRS of 98 msec and no signs of intraventricular delay. His blood pressure is: 98/55 mmHg. You also review his bloods:

Hb	129 g/L	Male: (135-180)Female: (115 - 160)
Platelets	335 * 10 /L	(150 - 400)
WBC	6.8* 10 /L	(4.0 - 11.0)
Na	137 mmol/L	(135 - 145)
K	4.7 mmol/L	(3.5 - 5.0)
Urea	5.4 mmol/L	(2.0 - 7.0)
Creatinine	125 µmol/L	(55 - 120)

He is currently on carvedilol 25 mg BD, enalapril 10 mg BD, bumetanide 2 mg BD, aspirin 75 mg, ivabradine 2.5 mg BD, spironolactone 25 mg OD.

What alteration to this gentleman's medications could potentially decrease this gentleman's probability of being re-admitted?

A. Increase ivabradine
B. Add sacubitril/valsartan
C. Increase spironolactone
D. Stop enalapril - initiate sacubitril/valsartan
E. Increase bumetanide

B. Add sacubitril/valsartan

Sacubitril-valsartan should be initiated following ACEi or ARB washout period

This gentleman poses the issue of long-standing management of heartfailure. The ESC guidelines advice strictly on the uptitration of medication up to their maximal desired effect taking in consideration the patient's status - the frontline medications include ACE inhibitors, b-blockers and mineralocorticoid receptor antagonists. The patient is on all three. If the patient remains symptomatic then a good choice would be dependable on their QRS morphology with a CRT-P device being appropriate in certain cases (e.g. QRS >130 msec) or ivabradine in someone with a heart rate of more than 70/min. The option of digoxin is also discussed in the ESC guidelines.

However the patient above is already bradycardic and hypotensive with a slightly raised potassium therefore making the possibility of increasing any of his current medications less appealing. In these patients the possibility of introducing sacubitril/valsartan is appropriate, however it is important for the clinician to be aware of the need for a wash-out period between the ACEi and the initiation of sacubitril/valsartan. The wash-out period advised is that of 36 hours.

The increase in diuretic might appear appealing, there is no evidence that diuretics improve mortality rates in such patients. The PARADIGMHF trial that was comparing sacubitril/valsartan and enalapril indicated that sacubitril/valsartan was superior to enalapril in reducing cardiovascular mortality, heart failure hospitalisation and 30-day hospital readmission in heart failure patients with reduced ejection fraction.

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An 80-year-old man with a history of atrial fibrillation is admitted following an acute coronary syndrome and is treated with percutaneous coronary intervention. His medication on admission was warfarin, cocodamol and allopurinol.

With regards to antithrombotic therapy, what should he be prescribed in the immediate aftermath of the event?

A. Continue warfarin with the addition of 2 antiplatelets
B. Continue warfarin with the addition of 1 antiplatelet
C. Continue warfarin monotherapy
D. Stop warfarin, start 2 antiplatelets
E. Stop warfarin, start 3 antiplatelets

A. Continue warfarin with the addition of 2 antiplatelets

In the initial phase following an ACS/PCI patients who also have AF are generally given 2 antiplatelets + 1 anticoagulant

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You are working in the general medical clinic where a 42 year old woman comes for review following a recent, short admission to hospital where she was treated for a paracetamol overdose. She has a past history of depression but denies any other previous problems.

During the review, she is found to have a manual blood pressure reading of 165/85 mmHg. Clinical examination of cardiovascular and respiratory systems are normal, as is urine dip and fundoscopy.

Given this information what should be your next course of management in relation to her blood pressure.

A. Start ramipril
B. Offer ambulatory blood pressure monitoring
C. Arrange to check blood pressure again following a two week interval
D. Start amlodipine
E. Screen for causes of secondary hypertension

B. Offer ambulatory blood pressure monitoring

In 2011 the National Institute for Clinical Excellence updated its 2006 guideline for the management of hypertension (see the link below for the quick reference guide). Within this guideline, the first line use of ambulatory blood pressure monitoring (ABPM) to confirm hypertension in those found to have an elevated clinic reading (> 140/90 mmHg) is emphasised. When using ABPM to confirm a diagnosis of hypertension, two measurements per hour are taken during the persons waking hours. The average value of at least 14 measurements are then used to confirm a diagnosis of hypertension.

Generally speaking, secondary causes of hypertension should be sought in; patients under 40 who lack traditional risk factors for essential hypertension, patients with other sings and/or symptoms of secondary causes, and patients with resistant hypertension. Although in reality the most common cause of secondary hypertension is hyperaldosteroneism,

and as such a trial of an aldosterone antagonist such as spironolactone is often employed as both a therapeutic and diagnostic measure.

Drug treatment of essential hypertension can be summarised as follows, but for a more detailed explanation see the link below;

- Step 1; Age <55 - ACE inhibitor. Age >55 or of black African or\Caribbean origin - calcium channel blocker
- Step 2; ACE inhibitor + calcium channel blocker
- Step 3; ACE inhibitor + calcium channel blocker + thiazide-like diuretic
- Step 4; consider further diuretic or beta-blockade or alpha blocker and seeking expert advice

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A 24-year-old primigravida presents to the emergency department with a 72-hour history of nausea and vomiting with associated right upper quadrant pain. She as no past medical history of note and takes only pregnancy vitamins. During her 32-week midwife appointment 1 week ago, it was noted that she had gained weight and had borderline hypertension. The decision at that appointment was to monitor.

On examination, she is clinically dehydrated and appears unwell. Respiratory and cardiovascular examinations are unremarkable and abdominal examination demonstrates a gravid uterus and right upper quadrant tenderness. There is pitting oedema to the mid-shin. Observations are all within normal limits aside from a blood pressure of 145/90mmHg. She is alert and orientated, reports no headaches and has no rashes.

Blood tests are performed, results are as demonstrated:

Hb	100 g/L	Male: (135-180)Female: (115 - 160)
Platelets	97 * 10 /L	(150 - 400)
WBC	7.3 * 10 /L	(4.0 - 11.0)
PT	13.0 seconds	(9.5-13.5)
APTT	39.0 seconds	(30-40)
Na	132 mmol/L	(135 - 145)
K	3.4 mmol/L	(3.5 - 5.0)
Bicarbonate	22 mmol/L	(22 - 29)
Urea	7.5 mmol/L	(2.0 - 7.0)
Creatinine	100 µmol/L	(55 - 120)
Bilirubin	45 µmol/L	(3 - 17)
ALP	150 u/L	(30 - 100)
ALT	350 u/L	(3 - 40)
Albumin	34 g/L	(35 - 50)

An ultrasound abdomen shows patchy areas of hepatic enhanced echogenicity. Fetal monitoring is satisfactory, but the baby is in the breech position.

Given the likely diagnosis, what is the most appropriate management?

A. Commence IV antibiotics
B. Give steroids and organise an induction of labour
C. Organise a plasma exchange with fresh frozen plasma
D. Rehydrate with IV fluids and monitor
E. Give steroids and organise a caesarean section within 48 hours

A. Give steroids and organise a caesarean section within 48 hours

Delivery of the baby is the treatment of HELLP syndrome

This patient has HELLP syndrome as shown by her anaemia (secondary to haemolysis), thrombocytopenia and deranged LFTs. She is 33 weeks pregnant and thus steroids would be recommended prior to delivery if possible to aid the maturation of her child's lungs. She does not have any evidence of a liver haematoma, disseminated intravascular coagulation or haemodynamic instability and thus she does not need an immediate delivery at this stage. Induction of labour would not be appropriate as her child is breech.

Differentials would include cholecystitis and thrombotic thrombocytopenic purpura (TTP).

IV antibiotics would be appropriate in the treatment of cholecystitis, but this patient has no features of infection, going against a diagnosis of cholecystitis.

Plasma exchange with fresh frozen plasma would be the recommended treatment for TTP, however, TTP would typically present with neurological symptoms such as confusion, headaches, and seizures in severe cases, which our patient does not have.

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A 24-year-old female presents to clinic complaining of several episodes of heart palpitations. She states that during the attacks it feels like her heart skips a bit. She denies pre-syncope or syncope. She states that she has one episode every few days. She drinks approximately 28 units of alcohol per week and several cups of coffee per day.

You arrange a 7 day cardiac holter monitor. The results are as follows:

PR interval	100ms
QRS duration	110ms
Events	24 single ectopics with normal P wave morphology and a QRS of110ms

What is the most appropriate initial management plan?

A. Bisoprolol
B. Ablation
C. Reassurance and lifestyle modifications
D. Implantable Cardioverter Defibrillator (ICD)
E. Verapamil

C. Reassurance and lifestyle modifications

Patients with ventricular ectopics can usually be managed by reassurance and lifestyle modifications. If pharmacological management is required, then beta blockers are first line

The ectopics captured have normal P wave and QRS duration suggestive of supraventricular ectopics of sinus or atrial origin. The first line management of supraventricular ectopics is generally reassurance and lifestyle modifications. The patient should be advised to reduce alcohol and caffeine intake. If symptoms persisted then a beta blocker would be first line.

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A 74-year-old man attends the emergency department with epistaxis. He denies any other bleeding. He has a past medical history of atrial fibrillation and severe mitral stenosis for which he takes warfarin. Observations are as follows: heart rate 85 beats per minute, blood pressure 135/75 mmHg, respiratory rate 16 breaths per minute, SpO2 95% (on air), and temperature 37.5ºC.

Blood results are as follows:

Hb	140 g/L	Male: (135-180)Female: (115 - 160)
Platelets	88 * 10 /L 9	(150 - 400)
WBC	6.2 * 10 /L 9	(4.0 - 11.0)
Prothrombin time (PT)	96 secs	(10-14 secs)
INR	6.8	(<1.1)
Activated partial thromboplastin time (APTT)	44 secs	(25-35 secs)
Fibrinogen	3.2 g/L	(2 - 4)

You withhold the patient's warfarin. What further measures are required?

A. Fresh frozen plasma
B. Intravenous 10mg vitamin K
C. Intravenous 3mg vitamin K
D. Platelet transfusion
E. Prothrombin complex concentrate

C. Intravenous 3mg vitamin K

NR 5.0-8.0 (minor bleeding) - stop warfarin, give intravenous vitamin K 1-3mg, restart when INR < 5.0

The results of the coagulation screen suggest warfarin toxicity. The INR (derived from the PT) is used to monitor warfarin since it is the most representative, however, it is important to remember that warfarin can also prolong the APTT.

Intravenous 3mg vitamin K is correct. The patient has a minor bleed with an INR between 5.0 - 8.0. Thus the warfarin should be withheld and the patient should receive intravenous vitamin K 1-3mg. The warfarin should be restarted when the INR < 5.0

Fresh frozen plasma is incorrect. Fresh frozen plasma (FFP) is not recommended for use as a warfarin reversal agent as it can take hours to work due to the volume required. In major haemorrhages, FFP should only be given if prothrombin complex concentrate is not available.

Intravenous 10mg vitamin K is incorrect. For minor haemorrhage, the dose of intravenous vitamin K recommended is 1-3mg.

Platelet transfusion is incorrect. In the context of haemorrhage, a platelet level of <50 is the standard trigger, with the exclusion of CNS or posterior eye bleeds in which the transfusion trigger is <100.

Prothrombin complex concentrate is incorrect. Prothrombin complex concentrate (PCC) is recommended only for major bleeding. PCC will reverse anticoagulation within minutes of administration however administration of vitamin K is also required to counteract the long halflife of warfarin.

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You are asked to chase up the INR result for a patient. The patient is currently admitted and being treated for a lower respiratory tract infection with clarithromycin. They take warfarin due to atrial fibrillation. The INR result is returned as below:

INR	6.8	(<1.5)

You review the patient who looks well, though they report persistent oozing of blood from where their cannula had been removed earlier that day. The gauze dressing has required changing 4 times.

What is the most appropriate course of action?

A. Hold warfarin and administer IV vitamin K

B. Hold warfarin and administer IV vitamin K and humanprothrombin complex
C. Hold warfarin and administer oral vitamin K
D. Hold warfarin and repeat INR the next day
E. Re-prescribe warfarin at a lower dose

A. Hold warfarin and administer IV vitamin K

INR 5.0-8.0 (minor bleeding) - stop warfarin, give intravenous vitamin K 1-3mg, restart when INR < 5.0

In this case, we have a patient with a raised INR which is likely secondary to clarithromycin use. The patient also has some minor bleeding as indicated by persistent ooze from a wound site.

Hold warfarin and administer IV vitamin K is correct. 1-3mg of IV vitamin K should be administered to this patient with minor bleeding and an INR of 5.0-8.0 as per NICE guidelines.

Hold warfarin and administer IV vitamin K and human prothrombin complex is incorrect. Human prothrombin complex should be administered in the case of major bleeding, fresh frozen plasma can be used as an alternative if human prothrombin complex is not available. As this patient is only having minimal bleeding, the use of human prothrombin complex is not recommended.

Hold warfarin and administer oral vitamin K is incorrect. This would be appropriate if the patient had no evidence of bleeding at all with an INR of >8.0. In this case, the INR is between 5.0 and 8.0 and associated with minor bleeding, intravenous vitamin K would therefore be more appropriate.

Hold warfarin and repeat INR the next day is incorrect. This would be appropriate if the INR was between 5.0 and 8.0 without any bleeding, you would then also want to reduce the maintenance dose. In this case, because there is evidence of bleeding, a dose of IV vitamin K is recommended.

Re-prescribe warfarin at a lower dose is not correct. This patient has a raised INR in the context of minor bleeding, the warfarin needs to be held and reversed in view of this.

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A 26-year-old patient comes into the Emergency Department in cardiac arrest. The paramedics tell you that she was pulled out of a lake by friends after she had suddenly become unresponsive while swimming. Her friends started cardiopulmonary resuscitation (CPR) at the scene. On arrival, the paramedics noted that the patient was in Ventricular Fibrillation (VF). The patient was defibrillated, given IV amiodarone and adrenaline. Her friends tell you she has no significant past medical history and was always fit and well until this point.

Despite continued attempts to resuscitate the patient, she dies in the Emergency Department. A post-mortem is carried out which is normal.

What is the most likely diagnosis?

A. Hypertrophic Obstructive Cardiomyopathy (HOCM)
B. Long QT1 Syndrome
C. Long QT3 Syndrome
D. Right Arrhythmogenic Ventricular Dysplasia (RAVD)
E. Wolf-Parkinson White Syndrome

B. Long QT1 Syndrome

All of these differentials can result in sudden cardiac death in a young patient. However, you have been told here that the patient's postmortem was normal which makes HOCM and RAVD unlikely.

This leaves Long QT1 Syndrome, Long QT3 Syndrome and WolfParkinson-White as possible differentials. The big clue here is that the patient became unresponsive when swimming which is typical of Long QT1 Syndrome.

Long QT syndromes can result in sudden cardiac death when patients are exposed to an adrenergic surge which can put the patient into VF or VT.

Long QT1: adrenergic surge due to intense physical activity such as swimming

Long QT2: adrenergic surge due to intense emotion such as excitement or fear

Long QT3: death during sleep

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You are asked to review a 22-year-old female with a heart murmur. Her height is 4ft 7 inches and she reports having always been short for her year. She has been diagnosed with autoimmune hypothyroidism, for which she is taking levothyroxine replacement and is also taking 'hormone replacement to protect her bones' after her GP noted she had not started her periods by 19 years old.

On examination, you note short 4th metacarpals on both hands and lymphoedematous in her hands and feet. Auscultation of her precordium reveals an ejection systolic murmur at the left infraclavicular region. Her body mass index is 22.4 kg/m². The patient's blood tests are as follows:

Hb	123 g/l
Platelets	380 * 10 /l9
WBC	6.5 * 10 /l9
Na +	144 mmol/l
K +	4.8 mmol/l
Urea	7.2 mmol/l
Creatinine	110 µmol/l
Adjusted calcium	2.40 mmol/l
Phosphate	1.1 mmol/l
PTH	9 mg/dl (8.5-10.2mg/dl)

What is the underlying unifying diagnosis?

A. Turner's syndrome
B. Pseudohypoparathyroidism
C. Pseudopseudohypoparathyroidism
D. Noonan syndrome
E. Down's syndrome

A. Turner's syndrome

A syndromic presentation appears most likely in this patient. An acute infective endocarditis is unlikely for someone who is systemically well, a regurgitant murmur would also be more likely. The symptoms cannot be attributed to delayed puberty, defined as onset of puberty outside 95% of the normal population and typically before 18 years old. The combination of primary amenorrhoea, short stature, lymphoedema, shortened 4th metacarpal, autoimmune predisposition and an ejection systolic murmur suggestive of aortic coarctation should infer a diagnosis of Turner's syndrome, a disorder caused by loss of an X chromosome.

Noonan syndrome, an autosomal dominant disorder, is a reasonable differential in a patient with short stature, lymphoedema and cardiac problems. However, patients with Noonan syndrome typically presents with learning difficulties, facial dysmorphisms and webbed neck. While valvular abnormalities are less common, hypertrophic cardiomyopathy is more commonly present.

Similar to Noonan syndrome, Down's syndrome, caused by trisomy 21, also classically presents with facial dysmorphisms such as upslanting palpebral fissures, low-set ears and epicanthic folds. Cognitive impairment and learning difficulties are also common. However, similar to our patient, short stature, thyroid dysfunction and cardiac problems, predominantly ASD, VSD and PDA are frequently associated.

Pseudohypoparathyroidism is caused by normal parathyroid hormone release but end organ resistance to hormonal effects, resulting classically in round facies and shortened fourth and fifth metacarpals. The patient is commonly obese with possible learning difficulties.

Pseudopseudohypoparathyroidism presents with the phenotype of pseudohypoparathyroidism but with normal calcium chemistry.

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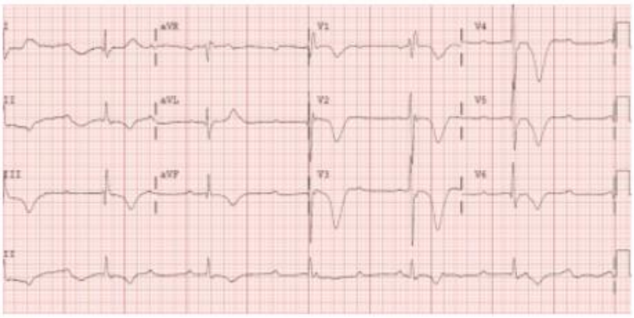
A 56-year-old man is brought to the Emergency Department. He works as a window cleaner. His colleague describes him suddenly falling from the ladder without warning. On admission he is conscious with a bradycardia of around 36/min. His blood pressure is 110/62 mmHg and he is well perfused with no signs of heart failure. An ECG is taken on account of his bradycardia:



A previous ECG taken four months ago was completely normal.

What is the most likely diagnosis?

A. Sick sinus syndrome
B. Stokes-Adams attack with associated complete heart block
C. Myocardial infarction with new left bundle branch block
D. Second-degree atrioventricular block (Mobitz II)
E. Second-degree atrioventricular block (Mobitz I / Wenckebach)

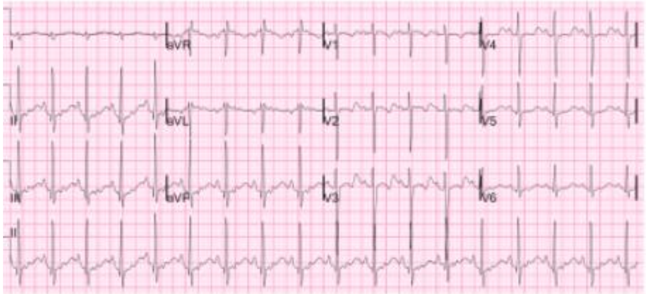
B. Stokes-Adams attack with associated complete heart block

There are regular p waves at a rate of around 90 / min but they do not conduct. The ventricular escape rhythm has a rate of about 36 / min and has a right bundle branch block (RBBB) like morphology. The bizarre, wide, inverted T-waves can be seen in Stokes-Adams attacks and do not necessarily imply new ischaemia.

Question 176 of 328

A 22-year-old woman presents to the emergency department with syncope. She also states that he has had profuse vomiting and diarrhoea for the last 3 days. Observations are as follows: heart rate 100 bpm, blood pressure 100/60 mmHg, respiratory rate 18 breaths per minute, SpO2 98% on air, temperature 37.2 ºC.

An ECG is performed in the department:



What is the likely cause of this ECG abnormality?

A. Arrhythmogenic right ventricular dysplasia (ARVD)
B. Hyperkalaemia
C. Hypokalaemia
D. Hypothermia
E. Wolff-Parkinson-White syndrome

C. Hypokalaemia

The ECG demonstrates sinus rhythm with a rate of approximately 100 beats per minute. There are prominent U waves following the T wave.

Hypokalaemia is correct. The electrocardiographic features are most in keeping with hypokalaemia. U waves are present and classic of hypokalemia, however, it is important to remember that they can be present in other conditions (e.g. bradycardia, hypocalcaemia, hypomagnesaemia, hypothermia and raised intracranial pressure). Other ECG features of hypokalaemia include small or absent T waves (occasionally inversion), prolonged PR interval, ST depression, and long QTc.

Arrhythmogenic right ventricular dysplasia (ARVD) is incorrect. ARVD is characterised by an epsilon wave which is a small deflection buried at the end of the QRS complex usually best seen in the ST segment of V1 and V2.

Hyperkalaemia is incorrect. ECG features of hyperkalaemia include peaked T waves, P wave widening/flattening, PR prolongation, bradyarrhythmias, and QRS widening with bizarre QRS morphology.

Hypothermia is incorrect. Hypothermia classically presents with sinus bradycardia and Osborn waves (J wave) defined as positive deflection seen at the J point in precordial and true limb leads. Osborn waves are characteristically seen in hypothermia, however, they are not pathognomonic and can be seen in other conditions such as hypercalcaemia and myocarditis.

Wolff-Parkinson-White syndrome is incorrect. This condition is associated with a short PR interval (< 120ms), broad QRS (> 100ms), and a slurred upstroke to the QRS complex (the delta wave).

Question 177 of 328

A 19-year-old woman presents to the acute medical take with three days of acute shortness of breath. She initially had a dry cough and sore throat but became increasingly lethargic. She is usually fit and well and is fully vaccinated. She is taking the combined oral contraceptive pill. On examination, she had bibasal crepitations with dullness to percussion at the bases. Her observations show a respiratory rate of 25 breaths per minute, heart rate 105 beats per minute and saturations of 96% maintained on 60% venturi mask. Her JVP is seen at 5 cm above her sternal angle and there are no murmurs.

Na +	130 mmol/l
K +	4.9 mmol/l
Urea	5.6 mmol/l
Creatinine	90 µmol/l
Troponin	20,000 ng/ml (normal range <20)
ECG	ST elevation seen in V1-V4

What is the most likely diagnosis?

A. Myocarditis
B. Myxoedema coma
C. Nephrotic syndrome
D. Myocardial infarction
E. Takotsubo cardiomyopathy

A. Myocarditis

Myocarditis presents with ST elevation and acute pulmonary oedema in a young patient with a recent flu-like illness

Bibasal crepitations and a raised JVP show acute heart failure. She has no chest pain making an acute myocardial infarction unlikely and there is no recent emotional distress as would be in Takotsubo cardiomyopathy. Given her recent viral illness, this is the most likely cause of her sudden deterioration in someone who was formerly well. Nephrotic syndrome and myxoedema coma would not have ST elevation.

Question 178 of 328

A 58-year-old woman was admitted with a stroke following a month's history of recurrent fevers, anorexia and weight loss. On examination, she had a left-sided hemiparesis and facial droop. Cardiovascular examination revealed splinter haemorrhages in 5 of her fingers across both hands, and a soft diastolic murmur heard loudest in expiration over the aortic area.

A trans-thoracic echocardiogram showed an oscillating vegetation on an aortic leaflet, in the path of regurgitant jets. Two blood cultures were positive for Streptococci spp. She was diagnosed with infective endocarditis and started on intravenous benzylpenicillin 1.2g every 4 hours and gentamicin 1mg/kg twice daily therapy.

She was reviewed after 5 days on antibiotic therapy with the following results.

Na+	139 mmol/l
K+	4.7 mmol/l
Urea	14.2 mmol/l
Creatinine	178 µmol/l
Serum bilirubin	16 µmol/l
Serum alkaline phosphatase	115 IU/l
Serum aspartate aminotransferase	18 IU/l
C Reactive protein	89 mg/l
Haemoglobin	138 g/l
White cell count	13.6 x 10^9/L
INR	1.1
Blood cultures	Streptococcus Bovis
Penicillin Minimum Inhibitory Concentration (MIC)	0.6 mg/L (high)
ECG	Prolonged PR interval (not present on admission ECG)

What is the next most important management?

A. Increase antibiotics to intravenous benzylpenicillin 2.4g every 4 hours and gentamicin 1mg/kg twice daily
B. Organise an urgent colonoscopy
C. Switch antibiotics to ceftriaxone 2g once daily
D. Refer to cardiothoracic surgeons
E. Organise urgent trans-oesophageal echocardiogram

E. Refer to cardiothoracic surgeons

This patient has infective endocarditis, as diagnosed by two positive major criteria from the Duke's criteria for infective endocarditis. In addition, she has a number of minor criteria, including fever, vascular events (stroke), immunological events (splinter haemorrhages, renal impairment secondary to glomerulonephritis). Despite starting on appropriate empirical treatment she appears to have an ongoing infection from her blood tests, and her ECG demonstrates a prolonged PR interval which may be reflective of an enlarging aortic abscess disrupting the atrioventricular node, which is an indication for referral for cardiothoracic surgery in infective endocarditis.

Other surgical indications include

- Heart failure: valve obstruction resulting in pulmonary oedema or shock, severe acute regurgitation
- Uncontrolled infection: abscess, false aneurysm, persisting fever and positive blood cultures for greater than 10 days despite appropriate antibiotics, multiresistant microorganisms
- Prevention of embolism: large vegetations resulting in one or more embolic episodes despite appropriate antibiotic therapy, or other predictors of complications eg. heart failure.

She has a penicillin-resistant streptococci, according to the British Society of Antimicrobial Chemotherapy guidelines, should be treated with vancomycin and gentamicin, so options (a) and (c) are incorrect. *Streptococcus Bovis* bacteraemia is associated with bowel malignancy, and a colonoscopy should be arranged to rule out malignancy, however, the worsening PR prolongation takes priority and should be managed first.

An urgent trans-oesophageal echocardiogram may be useful to evaluate the size of the aortic abscess, however, it would not change management as this patient needs to be referred to the cardiothoracic surgeons as there is already evidence of enlarging vegetation.

Question 179 of 328

A 75-year-old man is an inpatient on the orthopaedic ward recovering from an elective knee replacement performed three days previously. The patient's immediate post-operative recovery had been unremarkable and he had begun to mobilise with the ward physiotherapist. Routine observations recorded on the ward had been unremarkable during the previous 24 hours.

During her drug round, the patient's nurse saw the patient suddenly become unable to breathe and clutch at his chest. After calling for help the nurse went to her patient and found him to be in cardiac arrest. Resuscitations attempts were initiated following advanced life-support protocol. Please see the below table for a summary of the patient's electrical rhythm and treatments administered during the initial phases of the resuscitation attempt.

The patient was noted to be in good physical health with his only comorbidity being hypertension, well controlled with medication. The patient's admission clerking recorded that he was a retired schoolteacher who lived independently at home with his wife. A review of the patient's drug chart indicated that he had been receiving subcutaneous enoxaparin as prophylaxis against venous thromboembolism, but that the patient had refused to wear compression stockings during his admission as he found them uncomfortable.

Number of rhythm check	Result of rhythm check	Treatment administered
1	Pulseless electrical activity	IV adrenaline 1 mg
2	Ventricular fibrillation (coarse)	DC shock 150 J
3	Ventricular fibrillation	???

In addition to a further DC shock, what is the appropriate choice of IV drug treatment following the third rhythm check?

F. IV adrenaline 1 mg
G. IV atropine 400 micrograms & IV amiodarone 300 mg
H. No IV drug treatment indicated
I. IV adrenaline 1 mg & IV amiodarone 300 mg
J. IV atropine 400 micrograms

A. IV adrenaline 1 mg

The advanced life support algorithm for pulseless electrical activity (PEA) and asystole requires adrenaline to be given immediately and then continued every 3 to 5 minutes (in practice, after every second rhythm check). By contrast, the algorithm for ventricular fibrillation and ventricular tachycardia (VF / VT) requires IV adrenaline to not be given until after the third shock and then continued every 3 to 5 minutes.

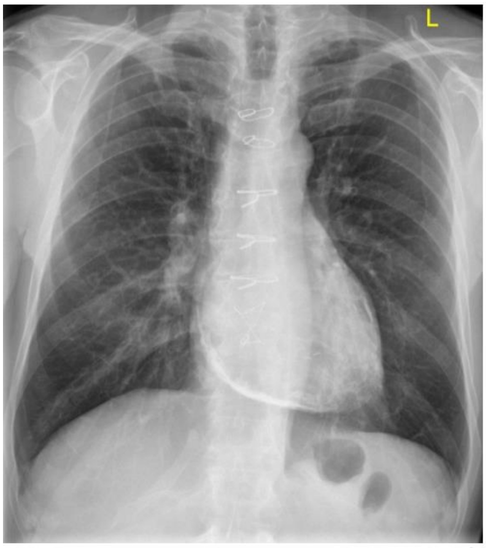
However, once adrenaline has been given during a resuscitation attempt it should be given every 3-5 minutes even if the type of rhythm changes from PEA/asystole to VF / VT, even if this is only the second cycle of the rhythm being VF / VT (as in the example in the question).

As part of the VF / VT algorithm, IV amiodarone 300 mg should be given after three defibrillation attempts. A further dose of IV amiodarone 150 mg should be considered after a total of five defibrillation attempts. Atropine is not used as part of the advanced life-support algorithm.

Question 180 of 328

A 60-year-old man is admitted to the Emergency Department with acute dyspnoea. He is unable to give a full history and his notes are not yet available.

His chest x-ray is shown below:



What is the most likely explanation for these changes seen over the heart?

A. Left ventricular aneurysm
B. Sarcoidosis
C. Atrial myxoma
D. Primary hyperparathyroidism
E. Previous episodes of uraemia

E. Previous episodes of uraemia

Pericardial calcification most commonly develops following repeated episodes of acute pericarditis. Calcification of the pericardium often results in constrictive pericarditis.

Question 181 of 328

A 19-year-old female presents to the medical outpatient with a history of palpitations. She suffers from bouts of anxiety and dizziness associated with these palpitations and has had one episode of syncope. She has had bouts of atrial fibrillation in the past, although documentary evidence is not available.

On examination, her blood pressure is 125/85 mmHg and her pulse is 140 bpm.

Her ECG reveals a broad complex regular tachycardia with a short PR interval and a slurred upstroke of the QRS complex. There is additionally a tall R wave in V1.

Which of the following would be the most appropriate initial step in medical management?

A. IV adenosine
B. IV verapamil
C. IV digoxin
D. IV propranolol
E. IV procainamide

E. IV procainamide

The description of the ECG given is that of WPW syndrome, a form of Atrio-Ventricular Reciprocating Tachycardia that results from conduction over an accessory pathway.

There are two types of WPW syndrome, Type A and Type B.

In case of type A (left atrioventricular connections), a positive R wave will be seen in V1 ('positive delta') on the precordial leads of the electrocardiogram,

In type B (right atrioventricular connections), a predominantly negative

delta wave will be seen in lead V1 ('negative delta').

The treatment of choice in the long term management is radiofrequency catheter ablation of the accessory pathway.

Care must be taken in the use of AV nodal blocking agents, especially when the tachycardia is broad complex, since these may paradoxically increase the conduction over the accessory pathway and lead to a 1:1 atrioventricular conduction which may result in ventricular fibrillation.

A simple mnemonic to remember for drugs to avoid in WPW syndrome is ABCD (Adenosine, -Blockers, Calcium Channel Blockers, Digoxin).

Question 182 of 328

A 58-year-old man is admitted with chest pain and shortness of breath. His breathlessness is worsening and he has a new oxygen requirement. His past medical history includes metastatic melanoma, hypertension and diabetes mellitus. He had surgical removal of the original melanoma lesion but now has extensive metastases. He has been seen regularly by the palliative care team as his pain has been very difficult to manage.

His pulse is regular on examination and chest sounds clear on auscultation. His ECG shows sinus rhythm with a rate of 61bpm and QTC is 455ms. His chest x-ray demonstrates clear lung fields. The results of his blood tests are shown below:

Hb	115 g/L	(135-180)
Platelets	451 * 10 /L 9	(150 - 400)
WBC	7.2 * 10 /L 9	(4.0 - 11.0)
Na +	142 mmol/L	(135 - 145)
K +	3.7 mmol/L	(3.5 - 5.0)
Urea	9.6 mmol/L	(2.0 - 7.0)
Creatinine	133 µmol/L	(55 - 120)
CRP	9 mg/L	(< 5)

Which of the following may account for his ECG findings?

A. Digoxin
B. Cyclizine
C. Bisoprolol
D. Amlodipine
E. Methadone

E. Methadone

Methadone is a common cause of QT prolongation

This man's ECG shows a prolonged QT interval at 455ms (normal for a male is <430ms).

Methadone is not a drug commonly used but can be used by the palliative care team for patients who are intolerant of opioids and their pain is unmanageable. One of the side effects of methadone is that it prolongs the QT interval and so is the correct answer.

Digoxin is used in the management of atrial fibrillation. Digoxin toxicity is associated with the 'reverse tick' ST morphology change.

Anti-emetics such as ondansetron, not cyclizine are associated with a prolonged QT interval.

Bisoprolol can be associated with bradycardia and atrioventricular block.

Amlodipine may cause palpitations and tachycardia but has no effect on the QT interval.

Question 183 of 328

A 67-year-old male presents with increasing chest pain on exertion over the past 3 weeks, stopping him from going to work. His past medical history includes hypertension, type 2 diabetes mellitus, sick sinus syndrome, recent abstinence from alcohol after a history of excess (with an episode of acute liver decompensation 9 months ago) and stable angina. He previously tried isosorbide mononitrate but reported significant headaches and facial flushing. He has not previously suffered myocardial infarctions, with a recent echo demonstrating 70% ejection fraction. An ECG demonstrates first-degree heart block with normal QRS complexes at 50 beats/ minute, blood pressure is 140/76 mmHg. He currently takes bisoprolol 5mg OD alone and has been using his GTN spray with increasing frequency without effect.

What is the most appropriate next step in management?

A. Reperfusion therapy with coronary artery bypass graft or percutaneous coronary intervention
B. Ranolazine
C. Ivabradine
D. Diltiazem
E. Nicorandil

Not answered

Question 184 of 328

A 73-year-old man presents to the medical take with a 3-month history of thoracic back pain. Over the last week, he had been having episodes of sweats and shivers, particularly at night.

He was admitted and blood tests were taken.

Haemoglobin	87 g/L
White cells	11.6x10^9/L
Platelets	214 x10^9/L
MCV	70 fl
MCH	20 pg
Blood cultures	Streptococcus gallolyticus

MRI showed a discitis at thoracic disks 8/9. ECHO: No vegetation seen

What is the next investigation for this patient?

A. Colonoscopy
B. CT chest/abdomen/pelvis
C. Ultrasound abdomen/pelvis
D. Repeat blood cultures

E. PET scan

A. Colonoscopy

Streptococcus bovis endocarditis is associated with colorectal cancer

Streptococcus gallolyticus is a subtype of *Streptococcus bovis*. *Streptococcus bovis* bacteraemia is associated with underlying colonic malignancies in approximately 10 to 25 percent of patients, therefore, all patients presenting with this should be investigated for rectal cancer. The gold standard investigation is a colonoscopy.

If a malignancy was found on colonoscopy then a CT chest/abdomen/pelvis would be warranted to look for metastases, but it would not be as sensitive as a colonoscopy to look for colonic malignancies. An ultrasound of the abdomen would also be less sensitive to diagnose colonic malignancies than a colonoscopy.

A CT-PET scan may well show a malignancy in the colon, but the patient must be exposed to radioactive dye and radiation, therefore is not the first line investigation.

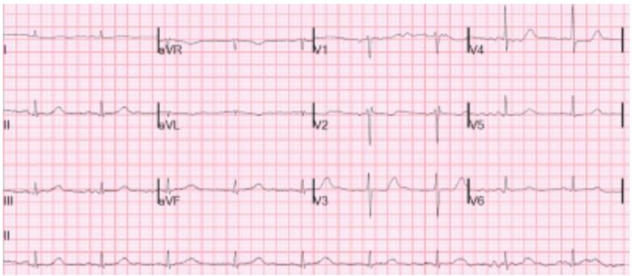
Repeat blood cultures would be useful when determining the length of treatment needed for this patient, however, they would not aid in the discovery of the underlying diagnosis.

Question 185 of 328

A 26-year-old woman presents to the emergency department with generalised weakness. She has a past medical history of type 1 diabetes mellitus and reports having been vomiting repeatedly over the past 4 days and cannot keep any food or liquids down. There is no history of fever or diarrhoea.

On examination, she appears lethargic with dry mucous membranes. Chest sounds are clear and heart sounds are normal. Her abdomen is soft with generalised tenderness mostly within the epigastric region.

Her blood glucose is recorded as 7.4 mmol/L. An ECG is taken on arrival at the emergency department, as shown below:



What is the recommended initial management for this patient?

A. Intravenous 0.9% sodium chloride with 40 mmol potassium
B. Intravenous 1.26% sodium bicarbonate
C. Intravenous calcium gluconate
D. Intravenous magnesium sulphate
E. Warmed intravenous 0.9% sodium chloride

A. Intravenous 0.9% sodium chloride with 40 mmol potassium

This patient's ECG shows U waves that are most noticeable in leads V2 and V3. U waves are small positive deflections that appear shortly after a T wave and frequently represent hypokalaemia. There is also a prolonged QTc interval (> 440ms). Other features of severe hypokalaemia include increased P wave amplitude, PR interval prolongation and T wave flattening.

Intravenous 0.9% sodium chloride with 40 mmol potassium is correct. There are several causes of U waves including hypothermia, bradycardia, hypocalcaemia, hypomagnesaemia and severe hypokalaemia. There are also multiple causes of a prolonged QTc interval including hypokalaemia, hypomagnesaemia, hypocalcaemia and hypothermia. Given the presentation of prolonged vomiting in a patient with type 1 diabetes (likely representing gastroparesis), this patient is at high risk for having hypokalaemia. The clinical history and ECG are suggestive of hypokalaemia and therefore intravenous potassium replacement is necessary.

Intravenous 1.26% sodium bicarbonate is incorrect. Sodium bicarbonate 1.26% can be administered in instances of metabolic acidosis and certain arrhythmias (eg broad complex tachycardia associated with salicylate overdose). It is not indicated for this patient.

Intravenous calcium gluconate is indicated for severe hypocalcaemia and hyperkalaemia with ECG changes such as tall tented T waves, QRS widening and flattened P waves. Although hypocalcaemia can cause a prolonged QTc interval which is seen here, it is a less common cause of U waves making calcium gluconate a less appropriate drug to administer.

Intravenous magnesium sulphate is given in the management of acute arrhythmias including atrial fibrillation and Torsades de Pointes (a monomorphic ventricular tachycardia associated with QTc prolongation). Although hypomagnesaemia can cause QTc prolongation, it is a less common cause of U waves in comparison to hypokalaemia, making potassium replacement the most appropriate initial management. However, it is important to remember that a normal magnesium level is required for the maintenance of a potassium level within the normal range and, therefore, replacement may also be necessary.

Warmed intravenous 0.9% sodium chloride is incorrect. Hypothermia is a less common cause of U waves and QTc prolongation and more commonly produces J waves which are not seen here. Secondly, there is no indication from the clinical presentation that this patient is hypothermic.

Question 186 of 328

A 67-year-old gentleman is brought to the resuscitation area of the Emergency Department after an out of hospital cardiac arrest. The paramedics are performing CPR and a laryngeal mask airway in place for ventilation. They inform you that the patient is in asystole and they have already administered IV adrenaline. On the next rhythm check you notice some P-waves on the monitor.

Which of the following is the best management option?

A. answer300mg IV amiodarone
B. IV fluid bolus
C. External pacing
D. Defibrillation
E. 3mg IV atropine

C. External pacing

This patient is in P-wave systole, a special case which does not follow the main cardiac arrest algorithm. In this scenario, external pacing can result in a return of spontaneous circulation.

Atropine is no longer used in any rhythm associated with cardiac arrest, while amiodarone and defibrillation are part of the shockable rhythm protocol. IV fluid bolus is recommended in cardiac arrest where hypovolaemia is suspected as an underlying cause, however, in this scenario, external pacing is more likely to result in the return of spontaneous circulation.

Question 187 of 328

A 80-year-old man with a past medical history of gout, reflux and ischaemic heart disease is admitted to the emergency department with a atrial fibrillation with fast ventricular response. He is managed according to ALS protocol and is stabilised.

A full set of bloods are sent and are displayed below:

Hb	135 g/l
Platelets	260 * 10 /l9
WBC	6 * 10 /l9
Mg	0.34 µmol/l
Ca (adj)	2.1 u/l
PO4	0.8 u/l

This is discussed with the cardiology registrar, who advises correction of the magnesium.

What medication is the most likely cause of hypomagnesaemia in this case?

A. Aspirin
B. Omeprazole
C. Ranitidine
D. Colchicine
E. Ramipril

B. Omeprazole

Careful electrolyte balance is important in the management of arrhythmias. During the generation of the action potential in cardiac pacemaker cells, phase 4 (inflow of potassium) is dependant on magnesium channels, and, although the exact effect in vivo of magnesium administration is unclear, restoring normomagnesaemia is important in patient presenting with dysrhythmias.

PPI use is associated with hypomagnesaemia - the exact mechanism of this is not known, but may be related to poorer intestinal absorption from dietary sources in patients on PPIs.

Question 188 of 328

A 66-year-old man was admitted from the community with an anterior STEMI to the local district general hospital. He complained of severe chest pain before his wife rang for help. She noted he was clammy and sweaty. About five minutes after being transferred into the emergency department a nurse observed that the patient looked unwell, appeared grey and poorly responsive, . The nurse put out a peri-arrest call.The cardiac monitor showed a broad complex tachycardia. His femoral pulse was faintly palpable and his blood pressure was 76/36 mmHg.

What should be the carried out immediately ?

A. Start CPR
B. IV fluids
C. DC cardioversion
D. IV amiodarone
E. Transfer to tertiary center for immediate PCI

C. DC cardioversion

DC cardioversion should be given to a patient presenting with ventricular tachycardia with haemodynamic compromise

The answer is immediate DC cardioversion for a patient with ventricular tachycardia presenting with haemodynamic compromise. If the patient was stable, amiodarone and magnesium could be considered. It is important to check the electrolytes. Ideally, the patient needs PCI but must be stable before transfer can take place. There is no role for CPR as this is not a cardiac arrest situation.

Question 189 of 328

A 51-year-old man is referred to the cardiology clinic by his GP.

He complains of a 1-year history of exertional breathlessness. He also feels breathless when lying flat.

He has a past medical history of hypertension, chronic kidney disease, and atrial fibrillation and is prescribed ramipril, bisoprolol, and apixaban.

On examination, his pulse is 70bpm and his blood pressure is 134/92mmHg. He has coarse crackles at both lung bases.

His blood tests are shown below:

Hb	141 g/L	Male: (135-180)Female: (115 - 160)
Platelets	177* 10 /L 9	(150 - 400)
WBC	6.7 * 10 /L 9	(4.0 - 11.0)
Na +	138 mmol/L	(135 - 145)
K +	5.5 mmol/L	(3.5 - 5.0)
Urea	12.9 mmol/L	(2.0 - 7.0)
Creatinine	188 µmol/L	(55 - 120)

The echocardiogram shows:

Summary: Left ventricular systolic function is moderately impaired. The ejection fraction is visually estimated at ~40%.

Regular furosemide is started.

What additional medication should be prescribed?

A. Amlodipine
B. Dapagliflozin
C. Ivabradine
D. Sacubitril and valsartan
E. Spironolactone

B. Dapagliflozin

SGLT-2 inhibitors should be considered as second-line therapy in patients with HFrEF in addition to optimised standard care

Dapagliflozin is correct. This patient has evidence of heart failure with reduced ejection fraction on his echocardiogram. The first-line treatment would be an ACE inhibitor and a beta blocker, but this patient already takes both of these agents. The second-line treatment is usually an aldosterone antagonist such as spironolactone, which can then be complemented with an SGLT2 inhibitor such as dapagliflozin to reduce mortality further. Given that this patient is already hyperkalaemic, spironolactone would be an inappropriate choice, and an SGLT2 inhibitor would be preferable.

Amlodipine is incorrect. Amlodipine would be indicated if the patient required an additional antihypertensive agent, but in this case, the blood pressure is within acceptable parameters, and amlodipine is not otherwise indicated in the management of heart failure. It may even worsen symptoms due to its vasodilatory effect, leading to fluid retention

Ivabradine is incorrect. Ivabradine is an inhibitor of the funny current in cardiac pacemaker cells. It is used in the management of angina and heart failure. In heart failure, however, it is only recommended as thirdline therapy and only in those in sinus rhythm with a ventricular rate over 75 bpm AND an ejection fraction of less than 35%. If ivabradine is used in patients with a resting heart rate below 75 bpm, the risk of bradycardia increases significantly. This patient is not yet on second-line therapy and meets neither of the above criteria, so this answer is incorrect.

Sacubitril and valsartan is incorrect. The combination of sacubitril and valsartan is recommended as a third-line therapy in the management of chronic heart failure, but only in those with a left ventricular fraction of less than 35%. This combination requires initiation by a specialist after an ACE inhibitor or ARB wash-out period. This patient has not yet been tried on second-line therapy; however, aldosterone antagonists +/—SGLT2 inhibitors would be considered first.

Spironolactone is incorrect. Second-line therapy in the management of stable heart failure is an aldosterone antagonist such as spironolactone. SGLT2 inhibitors may also be added at this stage. Given that this patient is already hyperkalaemic, an aldosterone antagonist would be contraindicated, making an SGLT2 inhibitor the correct answer in this case.

Question 190 of 328

A 52-year-old gentleman works as a lorry driver. His past medical history includes hypertension and raised BMI. He is admitted to the Emergency Department with chest pain. An ECG confirms ST depression in V1 to V4, with no pathological Q waves. Subsequent blood tests reveal a troponin of 10,000 consistent with a non-ST-elevation myocardial infarction (NSTEMI). He is commenced on appropriate secondary prevention, including aspirin 75mg OD, clopidogrel 75mg OD and fondaparinux 2.5mg SC. The patient is transferred to the cardiology unit and awaits inpatient angiography.

You are asked to review him urgently as he describes acute breathlessness. He denies chest pain. Observations are as follows: heart rate 130 beats per minute, blood pressure 95/62 mmHg, temperature 36.1ºC, respiratory rate 28/min, saturations 94% on 10 litres oxygen. On examination, heart sounds are normal with no added sounds. JVP is mildly elevated. There is no evidence of ascites or peripheral oedema. A repeat ECG confirms sinus tachycardia (heart rate 130bpm) but no dynamic changes. A portable chest x-ray reveals cardiomegaly with upper lobe diversion and peri-hilar shadowing. An urgent bedside echocardiogram demonstrates significant LV systolic dysfunction with a 2cm of pericardial effusion but no features of tamponade.

What is the single next best management step?

A. Continuous positive airway pressure (CPAP)
B. Ultrasound-guided pericardiocentesis
C. Urgent angiography
D. IV fluids
E. Inotrope and IV diuretics

E. Inotrope and IV diuretics

This patient is demonstrating features of cardiogenic shock, secondary to anterior wall myocardial infarction. With anterior infarcts, the mainstay of treatment includes diuretics to reduce pulmonary congestion and inotropic support to enhance cardiac output and perfusion.

Echocardiography demonstrates a reasonable-sized pericardial effusion but there is no indication for pericardiocentesis in the absence of features consistent with cardiac tamponade. Intravenous fluids would exacerbate pulmonary oedema in the context of anterior infarcts and are relatively contraindicated for this reason. They are of relevance, however, in inferior infarcts where preload needs to be maintained. This patient is preserving oxygen saturations via Venturi mask and therefore, CPAP is not currently indicated. Angiography is required to clarify the extent of coronary disease in due course, but the patient requires stabilisation before this can occur.

Question 191 of 328

A 35-year-old man presents to the emergency department with palpitations that came on suddenly earlier that afternoon. He has had symptoms like this on 3 previous occasions which resolved by themselves after a few minutes. He smokes a pack of cigarettes a day and drinks 5-6 pints of beer during the weekends.

He works as a builder on a construction site. He has no significant medical conditions and does not take any regular medication. In the emergency department, his heart rate is 180 beats per minute and his blood pressure 134/62 mmHg. He has no shortness of breath or chest pain but continues to have significant palpitations. His ECG shows a rapid regular rhythm with a QRS interval of 89 ms. On physical examination, he is comfortable at rest, and peripherally warm and well perfused. His JVP is not visible, and heart sounds are normal without any added sounds or murmurs, his chest is clear to auscultation, his abdomen is soft and nontender, and he has no swelling of his lower limbs. He is attached to a cardiac monitor and is asked to blow forcefully into a syringe on 2 separate occasions which does not improve his symptoms.

What is the next step in management?

A. Perform carotid sinus massage at the bedside
B. Administer 5 mg of IV verapamil
C. Administer 6 mg of IV adenosine
D. Administer 12 mg of IV adenosine
E. Administer sedation and prepare for direct current cardioversion

C. Administer 6 mg of IV adenosine

Patients with SVT who are haemodynamically stable and who do not respond to vagal manoeuvres, the next step is treating with adenosine

This young man presents with a likely supraventricular tachycardia. He has symptoms with palpitations, but does not have hemodynamic compromise, nor does he have signs or symptoms of cardiac ischemia or acute heart failure. He, therefore, does not need to be managed with direct current cardioversion. He has already tried vagal manoeuvres, by blowing forcefully into a syringe. The next step in his management is administrating a bolus of IV adenosine at a dose of 6 mg initially, in an attempt to slow down his heart rate and reveal the underlying rhythm.

He has already tried vagal manoeuvres on 2 occasions unsuccessfully and it would be inappropriate to try vagal manoeuvres again while forgoing giving adenosine which would be the next step in his management.

If the initial dose of 6 mg of IV adenosine is unsuccessful then he can be trialled with further adenosine at a higher dose of 12 mg. If these higher doses of adenosine are unsuccessful and he remains hemodynamically stable he could be treated with IV verapamil.

This patient is hemodynamically stable and does not have clinical features to suggest cardiac ischemia or acute heart failure and so does not require direct current cardioversion at present.

Question 192 of 328

A 13 year-old girl presents with chest pain and fever.

On examination the temperature is 38.1ºC, heart rate is 90 beats/minute and respiratory rate is 18 breaths/minute. The chest is clear to auscultation. There is an early diastolic murmur at the left sternal edge.

ECG reveals sinus rhythm, PR interval 210ms. Blood tests reveal:

Hb	121 g/l
Platelets	420 * 10 /l
WBC	9.3 * 10 /l
Na	136 mmol/l
K	3.7 mmol/l
Urea	3.8 mmol/l
Creatinine	72 µmol/l
Bilirubin	22 µmol/l
ALP	110 u/l
ALT	53 u/l
Albumin	36 g/l
C-reactive protein	36
Antistreptolysin O antibody titre	320 units/ml

Which other sign may also be present in this patient?

A. Dermatitis herpetiformis
B. Erythema marginatum
C. Pyoderma gangrenosum
D. Vitiligo
E. Livedo reticularis

B. Erythema marginatum

Acute rheumatic fever (ARF) is caused by a reaction to a bacterial infection with particular strains of group A streptococcus.

Symptoms of ARF generally develop several weeks after an episode of streptococcal pharyngitis

ARF causes a variety of characteristic clinical features:

- Polyarthritis
- Carditis
- Sydenham's chorea
- Erythema marginatum (A characteristic skin rash that occurs in about 10% of first attacks of ARF in children. it is rare in adults). The rash appears as pink or red macules (flat spots) or papules (small lumps), which spread outwards in a circular shape. As the lesions advance, the edges become raised and red, and the centre clears.

Question 193 of 328

A 55-year-old managing director presents with sudden onset retrosternal chest pain associated with light-headedness. She is a nonsmoker and has no other past medical history, except undergoing the menopause 6 years ago and taking a short course of hormone replacement therapy. On examination, she has cool peripheries and normal heart sounds. Her chest is clear with no peripheral oedema. Her calves are soft and non-tender. Her ECG demonstrates ST elevation in V2-V4, troponin 0.8 (normal range <0.03). Percutaneous coronary intervention was performed overnight, demonstrating no occlusions in her coronary arteries. However, ballooning of her left ventricular midcavity and apex was noted, in addition to left ventricular hypokinesia.

What is the most likely diagnosis?

A. Inferior non-ST elevation myocardial infarction (NSTEMI)
B. Hypertrophic obstructive cardiomyopathy
C. Prinzmetal angina
D. Pulmonary embolus
E. Takotsubo cardiomyopathy

E. Takotsubo cardiomyopathy

This is a patient with cardiac sounding chest pain with a troponin positive event, in the absence of vascular risk factors, and in the context of normal coronary vessels. The diagnosis is made on the LV description: apical ballooning is pathognomonic of Takotsubo cardiomyopathy, with the shape of the heart named after the Japanese octopus trap. It classically presents in post-menopausal women and is particularly triggered by anxiety and emotional stress. Treatment is supportive as per heart failure, possibly requiring inotropes if there is no LV outflow tract obstruction and with beta blockade, if there is LV outflow tract obstruction. Patients generally recover their LV ejection fraction within 4 weeks.

Question 194 of 328

A 69-year-old gentleman presents to the emergency department with shortness of breath. He has been experiencing worsening palpitations since he was diagnosed with a chest infection two days ago by his GP and started on oral antibiotics. He is now feeling very unwell and severely short of breath. He has a past medical history of myocardial infarction, AF and type 2 diabetes mellitus. On examination he has a heart rate of 167bpm, blood pressure of

87/50mmHg, oxygen saturations of 93% on 5 litres of oxygen and a temperature of 36.5ºC. He appears very unwell and distressed with bilateral crepitations to his midzones and peripheral oedema to his thighs. His ECG shows atrial fibrillations.

What is the most appropriate immediate management?

- A. IV fluids
- B. Synchronised DC cardioversion
- C. Unsynchronised DC cardioversion
- D. IV amiodarone
- E. IV adenosine

B. Synchronised DC cardioversion

In AF with decompensation the correct management is synchronised DC cardioversion

The patient should have a synchronised DC cardioversion attempted immediately. This patient has a tachyarrhythmia which has clearly decompensated him with evidence of acute heart failure and shock. This may have occurred due to underlying infection. The shock must be synchronised to deliver it during an R wave. This means that the defibrillator will detect R waves and when the shock is administered the machine will pause until it identifies an R wave and then deliver the shock. The purpose of this is primarily to avoid shocking during a T wave which could cause asystole. A shock without synchronisation is used in the resuscitation algorithm during VF and pulseless VT. Amiodarone should be used if three shocks fail to restore rhythm and adenosine is used in haemodynamically stable narrow complex tachycardia. Fluids may worsen pulmonary oedema in this setting

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A 53-year-old woman is found to have a systolic murmur on a routine check-up. A transthoracic echocardiogram reveals severe primary mitral regurgitation and a left ventricular ejection fraction of 55%, with other valves showing no significant pathology. A routine ECG demonstrates sinus rhythm. On questioning, she has no attributable symptoms such as shortness of breath, reduced exercise tolerance or chest pain. She is normally very active and works in an insurance firm. She has a past medical history of well-controlled asthma, post-partum depression and a tibial fracture one year ago. She has a salbutamol inhaler but no other regular medications.

What is the most appropriate management plan?

- A. Follow-up in six months
- B. Start nitrites
- C. Stop salbutamol
- D. Exercise testing
- E. Referral for mitral valve surgery

E. Referral for mitral valve surgery

The correct answer is a referral for mitral valve surgery. This asymptomatic patient with mitral regurgitation who has an ejection fraction of 55%. As this is below 60% the patient should be referred for surgery. Other surgical indications include new onset of AF or high likelihood of durable repair with low surgical risk and absence of risk factors. Follow-up in six months would be appropriate if there were no indications for surgery and medical management, such as nitrites, would be indicated if the patient was symptomatic and at too high risk for surgery. Stopping the salbutamol is unlikely to make any significant difference.

Question 196 of 328

A 55-year-old man with type 2 diabetes comes to the Emergency department with symptoms of worsening cardiac failure. Current medication includes metformin and empagliflozin for blood glucose control, ramipril, doxazosin, furosemide, aspirin and atorvastatin. His blood pressure is 112/70 mmHg, his pulse is 80 beats per minute and regular. There are bilateral crackles to the mid zones on auscultation of the chest, and he has pitting oedema to the mid-shins bilaterally.

Investigations

Na +	138 mmol/l
K +	4.5 mmol/l
Urea	6.2 mmol/l
Creatinine	112 µmol/l

Which of the following medications would you discontinue?

- A. Atorvastatin
- B. Doxazosin
- C. Empagliflozin
- D. Metformin
- E. Ramipril

B. Doxazosin

In this situation of worsening heart failure, discontinuation of the alphablocker doxazosin may well improve symptoms. The ALLHAT trial suggested that alpha blockade versus other antihypertensive options was associated with a 25% greater risk of congestive cardiac failure. Guidelines recommend that heart failure is best managed with ACE inhibition, a cardioselective beta blocker rather than an alpha blocker, and loop diuresis if required for fluid overload.

Although atorvastatin may be associated with myositis, it isn't thought to exacerbate heart failure. Evidence for empagliflozin, as one of the SGLT2 inhibitor class, is that it promotes sodium excretion and has a thiazide diuretic like effect with some benefit in patients with incipient cardiac failure. Metformin has no negative impact on heart failure. Metformin is only really contraindicated in a period of acute hypotension, not the chronic heart failure setting seen here.

Question 197 of 328

An 89-year-old woman presents to the emergency room with increased shortness of breath and decreased mobility of 1-week duration. Her shortness of breath is worse at night and she sometimes wakes up gasping for breath. She has fallen over twice in the last week which isn't normal for her. She has a past medical history of diabetes mellitus type 2 and hypertension. She has had no previous surgeries. She lives independently with her husband and her daughter lives close by and helps with the shopping.

On examination, she is found to have coarse crackles bi-basally on auscultation of her chest. She has a regular heart rate with a pan-systolic murmur loudest over the apex.

Investigations show:

Haemoglobin	11g/dl
WCC	6 x 10^9/l
Platelets	178 x 10^9/l
Sodium	139 mmol/l
Potassium	4.2 mmol/l
Urea	8 mmol/l
Creatinine	92 µmol/l
Blood cultures	Methicillin-sensitive Staphylococcus aureus

Echocardiogram severe mitral regurgitation with large mobile structure on valve leaflet

Chest X-ray: bilateral blunting of the costophrenic angles and upper lobe diversion

What is the best treatment for this lady?

A. Flucloxacillin orally
B. Flucloxacillin intravenously
C. Amoxicillin orally and vancomycin intravenously
D. Ceftriaxone intravenously
E. Amoxicillin intravenously and vancomycin intravenously

B. Flucloxacillin intravenously

This lady has a native mitral valve endocarditis with a bacteraemia caused by a methicillin-sensitive *Staphylococcus aureus*. Once cultures have grown the causative bacteria and it is found to be sensitive to methicillin guidelines suggest flucloxacillin intravenously to be the treatment of choice. Flucloxacillin orally would not provide sufficient antimicrobial cover nor will ceftriaxone, amoxicillin and vancomycin.

There is some debate about the optimal length of treatment, but 6 weeks of intravenous therapy is generally accepted as the length of treatment needed. Shorter therapeutic regimens may be effective in selected patients with right-sided endocarditis and with endocarditis due to highly susceptible *Streptococcus viridans* treated with synergistic antimicrobials.

Question 198 of 328

An 82-year-old presents with suprapubic discomfort. He reports urgency but has been unable to pass any urine for over 24 hours. Three months ago, he underwent a third failed transurethral resection of the prostate for benign prostatic hypertrophy (PSA was normal). His past medical history includes hypertension, type 2 diabetes mellitus, chronic kidney disease (baseline creatinine 150 µmol/l) previous MI in 2007 and 2010. He remains relatively active, with an exercise tolerance of 500 yards. On examination, his mucous membranes are moist, his peripheries are warm and his JVP at 3 cm above the angle of Louis. His heart sounds and chest are unremarkable. His abdomen is tender and distended in the suprapubic region. On insertion of a urethral catheter, 900mls of residual urine is noted. His blood tests are as follows:

Hb	115 g/l
Platelets	282 * 10 /l9
WBC	6.8 * 10 /l9
Na +	144 mmol/l
K +	5.8 mmol/l
Urea	12.1 mmol/l
Creatinine	201 µmol/l
Troponin T	0.08 (normal <0.03)

His ECG demonstrates left bundle branch block (old) and first-degree heart block at a rate of 49 beats/ minute and regular.

What is the appropriate management?

A. Treat as acute coronary syndrome only
B. Intravenous fluids only
C. Monitor renal function and consider long term catheter only
D. Treat as acute coronary syndrome, monitor renal function and consider long term catheter
E. Treat as acute coronary syndrome, intravenous fluids, monitorrenal function and consider long term catheter

C. Monitor renal function and consider long term catheter only

This patient is a typical elderly admission with multiple co-morbidities involving multiple organ systems. The key to this question is recognising which aspects are important and which are not. The patient is clearly euvolaemic and intravenous fluids at this stage are not required. However, it would be prudent to monitor urine output and if significant amounts are produced, to replace output with oral intake or intravenous input. Troponin is raised but this is in the context of an acute on chronic kidney injury. In the context of no reported chest pain and no new dynamic ECG changes, treatment as an acute coronary syndrome is unnecessary. The patient is at greater risk of post-renal injury and his renal function tests should instead be monitored. A long-term catheter is a reasonable suggestion considering the number of failed TURPs.

Question 199 of 328

A 59 year-old man with history of ischaemic heart disease and type 2 diabetes mellitus is eight hours post right curative hemicolectomy for bowel malignancy. In the surgical high-dependency unit he is noted to be tachycardic on the monitor

On examination the patient appears comfortable. The pulse rate is 200bpm and the blood pressure is 148/79mmHg. Oxygen saturations are

98% on 2L/min nasal oxygen. Capillary refill is 2 seconds. The chest is clear to auscultation.

A 12-lead ECG reveals a regular broad complex tachycardia with a monomorphic waveform at a rate of 200bpm.

Post-operative blood tests reveal:

Hb	131 g/l
Platelets	563 * 10 /l9
WBC	13.4 * 10 /l9
Na +	141 mmol/l
K +	4.1 mmol/l
Mg ++	0.87 mmol/l
Urea	4.2 mmol/l
Creatinine	121 µmol/l
Bilirubin	23 µmol/l
ALP	109 u/l
ALT	34 u/l
Albumin	33 g/l

What is the most appropriate initial management?

A. Magnesium sulphate 2g IV
B. Amiodarone 300mg IV
C. Synchronised DC shock

D. Metoprolol 5mg IV
E. Adenosine 6mg IV

B. Amiodarone 300mg IV

This case outlines a description of ventricular tachycardia.

Following the resuscitation council guidelines, in the absence of shock, syncope, myocardial ischaemia or heart failure (which would necessitate DC cardioversion), the most appropriate initial management is 300mg amiodarone intravenously.

Question 200 of 328

A 65-year-old obese female with known type 2 diabetes mellitus presents with one-week history of unstable angina. Her troponin was negative and she presented with no dynamic ECG changes. She undergoes an angiogram and a drug-eluting stent was inserted into a 90% stenosis of his LAD.

On discharge, what should her antithrombotic regime be?

A. Aspirin and prasugrel for 1 month, followed by lifelong aspirin
B. Aspirin and prasugrel for 1 month, followed by lifelong prasugrel
C. Aspirin and prasugrel for 1 year, followed by lifelong aspirin
D. Aspirin only, lifelong
E. Prasugrel only, lifelong

C. Aspirin and prasugrel for 1 year, followed by lifelong aspirin

The correct answer for this question is **aspirin and prasugrel for 1 year, followed by lifelong aspirin**. This is because the patient has undergone percutaneous coronary intervention (PCI) with a drug-eluting stent (DES) insertion, which requires dual antiplatelet therapy (DAPT) for at least one year according to UK guidelines. Aspirin is given as a lifelong therapy, while prasugrel, a P2Y12 inhibitor, is given in combination with aspirin for the first year. This regimen helps to prevent stent thrombosis and reduce the risk of recurrent ischemic events.

The option **aspirin and prasugrel for 1 month, followed by lifelong aspirin** is incorrect because DAPT duration of only 1 month is too short after DES placement. The minimum recommended duration of DAPT following DES implantation in patients with stable coronary artery disease or acute coronary syndrome (ACS) without high bleeding risk is 6 months and 12 months respectively.

The option **aspirin and prasugrel for 1 month, followed by lifelong prasugrel** is also incorrect because it does not provide adequate duration of DAPT after DES placement. Moreover, switching from DAPT to monotherapy with only prasugrel would not be appropriate as aspirin remains the cornerstone of long-term antithrombotic therapy in these patients.

Aspirin only, lifelong is not an appropriate option either since it does not include a P2Y12 inhibitor like prasugrel or clopidogrel that should be added to aspirin during the first year following PCI with DES insertion to minimize the risk of stent thrombosis.

Lastly, **prasugrel only, lifelong** would not be suitable as it does not include aspirin, which is a crucial component of long-term antithrombotic therapy in patients with coronary artery disease. Furthermore, prasugrel monotherapy has not been shown to be superior to DAPT with aspirin and a P2Y12 inhibitor in preventing recurrent ischemic events.

In summary, the best antithrombotic regimen for this patient would be **aspirin and prasugrel for 1 year, followed by lifelong aspirin**, as it provides optimal protection against stent thrombosis and recurrent ischemic events following PCI with DES insertion.

Question 1 of 129

A 45-year-old man is being worked up for chest pain.

He has a background of bipolar disorder for which he takes olanzapine but is otherwise fit and well.

He has had a cardiac MIBI scan that is reported as follows:

Images display a moderate perfusion abnormality in the right ventricle on the stress images with associated dyskinesis. This defect is also present at rest.

Given the above information, what is the most likely cause of these imaging findings?

A. Atherosclerosis
B. Hypertrophic cardiomyopathy
C. Kawasaki disease
D. Soft tissue attenuation defect
E. Transmural scarring

E. Transmural scarring

If a cardiac MIBI scan shows that the defect is present on both stress and rest, this is suggestive of a fixed defect such as myocardial necrosis and fibrosis secondary to infarction

Here we have a patient with a perfusion defect in the right ventricle at rest and during stress. This indicated that the lesion was not reversible.

Transmural scarring is correct. When scar tissue is formed within the myocardium, there will be a fixed perfusion defect as blood supply to this area never normalises.

Atherosclerosis is incorrect. Atherosclerosis commonly causes reversible defects that normalise at rest.

Soft tissue attenuation defect is incorrect. Although soft tissue attenuation defects can cause the appearance of fixed defects, there would not be associated wall motion abnormalities.

Kawasaki disease is incorrect. Although this vasculitis can be a cause of myocardial infarction it is very rare. It usually affects children under the age of five and is associated with fever, strawberry tongue, rash, and lymphadenopathy, none of which are seen in this case.

Hypertrophic cardiomyopathy is incorrect. This would be visible on MIBI scan as ventricular hypertrophy, if a defect is present, it is likely to be present only during stress.

Question 2 of 129

A 69-year-old man presents with central crushing chest pain. He also feels nauseous. He states that the pain came on at rest. He has a past medical history of hypertension and poorly controlled type 2 diabetes mellitus.

An ECG is performed:

P waves	Normal morphology
PR interval	120ms
QRS	115ms
T waves	Deeply inverted T waves in V2-V3
ST segments	No elevation or depression

What is the most likely diagnosis?

A. Critical stenosis of the right coronary artery
B. Critical stenosis of the left main stem coronary artery
C. Critical stenosis of the left anterior descending artery (LAD)
D. 75% stenosis of the left anterior descending artery (LAD)
E. 75% stenosis of the right coronary artery

C. Critical stenosis of the left anterior descending artery (LAD)

Wellens' syndrome is a pattern of deeply inverted or biphasic T waves in V2-3. It is highly specific for critical stenosis of the left anterior descending artery (LAD). It should be treated as a STEMI with urgent angiography and revascularisation

The patient has multiple major risk factors for cardiovascular disease making an acute coronary syndrome high up the differential list. The deeply inverted T waves in V2-V3 are very suggestive of Wellens' syndrome which is highly specific for critical stenosis of the left anterior descending artery (LAD). It should be treated as a STEMI with urgent angiography and revascularisation.

Right coronary artery pathology tends to present with ECG changes in the inferior leads (II, III and AVF).

Left main stem coronary artery occlusion tends to present with widespread ECG changes with horizontal ST depression (most prominent in leads I, II and V4-6), and ST elevation in aVR.

Question 3 of 129

A 28-year-old man is admitted with 4 episodes of shocks from his ICD in the past 24 hours. He states that the chest pain is like an electric shock, which is brief and lasts for only a second or two. He also complains of palpitations. He has a past medical history of hypertrophic obstructive cardiomyopathy (HOCM) and has an ICD due to previous episodes of ventricular tachycardia.

Whilst in the emergency department he suffers from 8 consecutive shocks from the ICD. A 12 lead ECG shows an irregularly irregular rhythm with a QRS of 0.10s, and absence of P waves. His heart rate is 140bpm and blood pressure = 130/75 mmHg. There is no signs of heart failure on examination.

What is the most appropriate initial management?

A. Apply a ring magnet over the ICD to temporarily deactivate it pending device interrogation
B. Rate control with calcium channel blocker
C. Chemical cardioversion with amiodarone
D. Synchronised electrical cardioversion
E. Rate control with beta blocker

A. Apply a ring magnet over the ICD to temporarily deactivate it pending device interrogation

Multiple episodes of inappropriate shocks from an ICD can be both unpleasant and dangerous. Ultimately the device will need interrogated from a pacemaker technician, however the most immediate management should be to place a ring magnet over the ICD to prevent further inappropriate shocks.

The patient has presented with multiple episodes of shocks from his ICD. The ICD was inserted to cardiovert life threatening arrhythmias such as ventricular tachycardia and fibrillation. However in this case, he has developed new onset AF, and has received multiple inappropriate shocks from his ICD.

Ultimately he will need the device interrogated from a pacemaker technician, however the most immediate management should be to place a ring magnet over the ICD to prevent further inappropriate shock, which can both be painful, and cause harm. This must only be done while the patient is on a cardiac monitor or telemetry so that one can monitor for any life threatening arrhythmias. If, at any time, you identify ventricular tachycardia or ventricular fibrillation on the cardiac monitor, the ring magnet must be removed immediately allowing normal firing of the ICD to resume after approximately 15 seconds.

One could argue that pharmacological cardioversion or rate control would resolve the problem. However the most immediate management would involve preventing further shocks with a ring magnet while the device is interrogated.

Question 4 of 129

A 48-year-old hypertensive, male who smokes presents with central crushing chest pain and nausea. His ECG shows new deeply inverted T waves in V2 and V3.

These ECG findings are consistent with critical stenosis to which coronary artery?

A. Left anterior descending artery
B. Left circumflex artery
C. Left main artery
D. Posterior descending artery
E. Right coronary artery

A. Left anterior descending artery

Wellens' syndrome is a pattern of deeply inverted or biphasic T waves in V2-3. It is highly specific for critical stenosis of the left anterior descending artery (LAD). It should be treated as a STEMI with urgent angiography and revascularisation

This patient presents with typical symptoms of acute coronary syndrome. Deeply inverted or biphasic T waves in V2 and V3 are consistent with a diagnosis of Wellens' syndrome. Wellens' syndrome is caused by critical stenosis of the left anterior descending (LAD) artery and is associated with a high risk of extensive anterior wall myocardial infarction. Patients may be pain-free at presentation or have minimally elevated cardiac enzymes. Patients with Wellens' syndrome typically require intervention with urgent angiogram and revascularisation, as in an acute ST-elevation myocardial infarction. The other listed coronary arteries are not associated with Wellens' syndrome.

The left circumflex artery supplies the left ventricle. The left main artery supplies the left ventricle and atrium.

The posterior descending artery supplies the inferior aspect of the myocardium.

The right coronary artery supplies the right ventricle, right atrium and the sino-atrial and atrioventricular nodes of the heart.

Question 5 of 129

An 8-year-old boy presents to the emergency department with seven days of fever. He originally attended six days previously and was discharged home with advice to return if the fever persisted. There has been no response to paracetamol treatment. He has not past medical history.

Observations:

- Heart rate 84 beats per minute
- Blood pressure 100/70 mmHg
- Temperature 39.4C

On examination, his conjunctivae are erythematous. He has red, cracked lips and a 'strawberry tongue'. There is unilateral cervical lymphadenopathy. The palms of his hands and soles of his feet are red. The examination is otherwise normal.

Urinalysis and a chest x-ray are unremarkable. Blood tests:

Hb	136 g/L	Male: (135-180)Female: (115 - 160)
Platelets	189 * 10 /L	(150 - 400)
WBC	8.9 * 10 /L	(4.0 - 11.0)
Na	140 mmol/L	(135 - 145)
K	4.2 mmol/L	(3.5 - 5.0)
Urea	5.2 mmol/L	(2.0 - 7.0)
Creatinine	77 µmol/L	(55 - 120)
CRP	35 mg/L	(< 5)
Bilirubin	14 µmol/L	(3 - 17)
ALP	88 u/L	(30 - 100)
ALT	64 u/L	(3 - 40)
Î³GT	44 u/L	(8 - 60)
Albumin	36 g/L	(35 - 50)

In addition to aspirin, what other treatment has the most evidence for benefit in treating this condition?

A. Antibiotic treatment
B. Cyclophosphamide
C. Intravenous immunoglobulin
D. Prednisolone
E. Rituximab

C. Intravenous immunoglobulin

Intravenous immunoglobulin , uses include: Kawasaki disease

Intravenous immunoglobulin is correct. The child has Kawasaki disease as evidenced by a fever lasting > 5 days, bilateral conjunctival congestion, cervical lymphadenopathy and changes of the lips and extremities. This is a medium-vessel vasculitis that occurs in children. In addition to aspirin, intravenous immunoglobulin should be administered to prevent the formation of coronary artery aneurysms, which is a feared complication of this condition.

Antibiotic treatment is incorrect. The clinical syndrome, in this case, suggests Kawasaki disease as the cause of fever, rather than infection. This is an inflammatory rather than infective condition and does not require antibiotic treatment.

Cyclophosphamide is incorrect. This has been used occasionally in refractory Kawasaki disease but it has less evidence than IVIG. Additionally, there is a risk of infertility associated with this treatment and therefore it should be avoided in people of child-bearing age unless absolutely necessary.

Prednisolone is incorrect. There is more limited evidence for the benefit of corticosteroids in this condition compared with IVIG. Adjunctive treatment with corticosteroids could be considered in particular high risk groups (Age < 12 months, Asian ethnicity) and in the setting if particular blood test abnormalities (ALT >100 IU/L, albumin ≤30 g/L, sodium ≤133 mmol/L, platelets ≤30 x 109/L, CRP >100 mg/L, anemia) or cardiac or coronary artery involvement on echo at presentation.

Rituximab is incorrect. This is an anti-CD20 monoclonal antibody that is used in the treatment of some vasculitis. There is limited evidence for its use in Kawasaki disease but it has been used occasionally in IVIG refractory disease.

Question 6 of 129

A 67-year-old woman attends Cardiology clinic. She has recently had a permanent pacemaker (PPM) inserted due to pre-syncopal episodes associated with bradycardia. She reports an improvement in these symptoms since insertion, but notes exertional chest pain since the procedure. She has a past medical history of type 2 diabetes mellitus and hypercholesterolaemia, for which she takes metformin and atorvastatin.

What is the most cause of her symptoms?

A. Pacemaker lead malposition
B. Costochondritis
C. Bornholm disease
D. Pacemaker syndrome
E. Coronary ischaemia

E. Coronary ischaemia

Pacemaker lead malposition, costochondritis and Bornholm disease (a viral infection causing pain in the lower chest) would not typically be associated with specifically exertional chest pain.

Pacemaker syndrome is typically associated with a VVI pacemaker that results in simultaneous atria and ventricle conduction. This can cause fatigue, dizziness and hypotension.

This patient is a good candidate for coronary artery disease with type 2 diabetes mellitus and hypercholesterolaemia. Pre-pacemaker insertion her heart had lower energy requirements due to conduction system disease. Pacing has improved her potential heart rate and therefore increased energy demands. This has unmasked her underlying coronary ischaemia. She now needs an angiogram!

Question 7 of 129

A 28 year old woman is admitted to the medical assessment unit complaining of pleuritic chest pain. She is 32 weeks pregnant into her first pregnancy. An ECG reveals a sinus tachycardia. Her blood tests are unremarkable, although the referring accident and emergency doctor added on a D-dimer test for completion and this has come back positive.

According to the Royal College of Obstetricians and Gynaecologists guidelines, what is the next suitable investigation for this patient?

A. Chest x-ray
B. Ventilation/perfusion scan
C. Compression duplex doppler of the legs
D. CT pulmonary angiogram (CTPA)
E. Cardiac catheterisation to assess right sided pressure

A. Chest x-ray

In suspected pulmonary embolism in pregnant patients the Royal College of Obstetricians and Gynaecologists recommend the following:

1. Chest xray to look for an alternative diagnosis.

2. If the chest xray is normal consider a compression duplex Doppler of both legs to exclude a DVT. If this is positive, the patient is treated with full dose low molecular weight heparin (LMWH) (warfarin is of course teratogenic).

3. If both the above investigations are normal, and there remains a strong suspicion of a pulmonary embolism, then clinicians should consider. a CTPA or ventilation perfusion/scan weighing up the risks vs benefits of each and also the local hospital protocol. Current guidance however favours a perfusion scan as it has lower lung radiation doses than a CTPA.

Question 8 of 129

A 54-year-old male previously treated for lymphoma is seen in clinic with a 6-month history of exertional dyspnoea which is progressive. He was treated four years ago and was told that there was no evidence of disease on the final CT scan. His GP treated him for a presumed lower respiratory tract infection two weeks ago. He returned from a business trip to Thailand six months ago, during which he consumed more alcohol than he usually does. He takes no medications and is otherwise fit and well, and is a non-smoker. On examination there is mild pedal oedema, his chest is clear and jugular venous pressure(JVP) is raised on inspiration. Auscultation of his heart reveals an extra heart sound very soon after S2.

What is the likely cause of his symptoms?

A. Superior vena cava obstruction
B. Cardiac tamponade
C. Left ventricular failure
D. Chronic obstructive pulmonary disease
E. Constrictive pericarditis

E. Constrictive pericarditis

Examination of this patient demonstrates Kussmaul's sign - paradoxical elevation of the JVP on inspiration. This sign is seen in constrictive pericarditis, cardiac tamponade and restrictive cardiomyopathy. This presentation gives no other features to suggest tamponade. His constrictive pericarditis here is likely cause by the previous radiotherapy for lymphoma. Other causes of constrictive pericarditis include TB and chronic pericarditis. Definitive management involves surgical pericardial stripping.

Question 9 of 129

A 38-year-old female presenting with 3 months of increasing abdominal and lower limb swelling. She has a past medical history of myasthenia gravis, associated with a thymic mass. Five years ago, she underwent a thymectomy and after positive margins were noted, she was commenced on a course of radiotherapy. She has no other past medical history. On examination, her heart sounds are quiet with no added sounds. Her chest is clear on auscultation. Her JVP is located at 6 cm above the angle of Louis. Abdominal examination reveals a pulsatile liver and smooth hepatomegaly. A chest x-ray demonstrates only minimal pleural and pericardial calcification.

What is the definitive treatment?

A. Intravenous furosemide
B. Intravenous methylprednisolone
C. Intravenous immunoglobulin
D. Pericardial drain insertion
E. Pericardiectomy

E. Pericardiectomy

The patient presents with clinical features of constrictive pericarditis. Her previous radiotherapy increases the aetiology of radiation-induced pericarditis. The patient has prominent and persistent symptoms for which pericardiectomy is the only definitive treatment available. While diuretics may hold the symptoms prior to surgery or be used in patients not suitable for surgery, pericardiectomy should be the definitive treatment despite the high operative risks. However, radiation induced constrictive pericarditis is known to demonstrate a worse long-term prognosis than other causes.

Question 10 of 129

A 76-year-old man with diabetes, hypertension, rheumatoid arthritis and gout presents with a 3 month history of deterioration. He is generally lethargic with reduced energy and has changed from someone who was walking with sticks to now using a frame. He occasionally vomits and has sweating at night time. His wife is concerned that he has lost weight.

On examination, he has clubbing present and a red macular rash across his hands. He complains of his fingers itching and in the abdomen, the spleen is palpable three centimeters below the costal margin. He has a pan systolic murmur and there are crepitations in both lungs. He starts to shake vigorously but remains awake and feels clammy. He is tender across his gluteal muscles.

Hb	109 g/l	Na +	130 mmol/l
Platelets	276 * 10 /l 9	K +	3.7 mmol/l
WBC	10.4 * 10 /l 9	Urea	4.5 mmol/l
Neuts	9.8 * 10 /l 9	Creatinine	89 µmol/l
Lymphs	0.4 * 10 /l 9	CRP	42 mg/l
Eosin	0.1 * 10 /l 9	ESR	32 mm/hr

Blood film	some microcytosis, schistocytes and cell fragments seen
ECG	sinus tachycardia rate 102/min
Chest x-ray	globular heart, bilateral interstitial shadowing

What is the likely diagnosis?

A. Lymphoma
B. Infective endocarditis
C. Congestive cardiac failure
D. Mycoplasma pneumonia
E. Polymyalgia rheumatica

B. Infective endocarditis

Strep endocarditis presents insidiously with new murmur

Important for me Less imp

This is a chronic inflammatory condition and it can be hard to tell apart the options. Congestive cardiac failure usually has a history of myocardial infarctions and an elevated JVP or ankle oedema which is not seen here. Lymphoma has palpable (cervical, axillary, inguinal) or radiological (mediastinal) evidence of lymphadenopathy although there is splenomegaly here. Polymyalgia rheumatica can present in unusual forms like this but we should look at the other options. *Mycoplasma* can give erythema multiforme which is a macular rash and there is hyponatraemia too. However, the history is too chronic for *mycoplasma* and the unexplained murmur, fevers and raised inflammatory markers should prompt thoughts of a *streptococcus* endocarditis.

Question 11 of 129

A 23-year-old woman is referred by the emergency department with sudden onset shortness of breath and pleuritic chest pain. She describes no cough or sputum. The pain is constant and not worse on movement. She is 24 weeks pregnant with her third child. She is normally fit and well and on no regular medication beyond her pregnancy supplements. On examination, she is slightly tachypnoeic at 21/min but her sats are 98% on room air. Her blood pressure is 110/78 mmHg and her heart rate is 86/min. Her chest is clear and the JVP is not elevated and her heart sounds are normal. Examination of her right leg reveals a swelling from above the right knee down to the ankle. There is pitting oedema on the right side as well as collateral superficial veins.

The emergency department registrar has sent several blood tests. The results are:

Hb	118 g/l
Platelets	443 * 10 /l
WBC	9.8 * 10 /l
Trop T	6ng/L (<14ng/L excludes cardiac damage)
D-dimer	1.3 mg/L (<0.5)
CRP 16	mg/L (<10)

Her chest x-ray is unremarkable. Her ABG shows:

pH	7.39
pO2	8.6kPa
pCO2	2.9kPa
HCO3-	21mmol/L (22-29)
Sats	97%
BE	1.1

She is seen on the post take ward round and the Consultant would like to exclude a pulmonary embolism.

What is the most appropriate first investigation for this lady?

A. Half dose CTPA
B. V/Q scan
C. USS Doppler of legs
D. Full dose CTPA
E. No need for investigation treat with LMWH

C. USS Doppler of legs

This lady could well have a PE. However, she also has signs of a deep vein thrombosis in the right leg.

The current guidelines in these situations recommend a CXR followed by USS Doppler of the legs. If a DVT is found they are anticoagulated without any radiation exposure to the baby. If the USS is negative and CXR normal they should then be offered the informed choice of V/Q scan or CTPA. V/Q carries a slightly higher risk to the baby of childhood cancers whilst a CTPA carries a slightly increased lifetime risk of breast cancer to the mother.

Question 12 of 129

A 22-year-old male presents to clinic complaining of several episodes of heart palpitations. He states that during the attacks his heart beats extremely fast and he feels dizzy.

You arrange a 7 day cardiac holter monitor. The results are as follows:

PR interval	95ms
QRS duration	105ms
Events	One episode of tachycardia of 145 bpm with normal QRS morphology and QRS 110ms

What is the most likely diagnosis?

A. Lown-Ganong-Levine syndrome
B. Wolff-Parkinson-White syndrome type A
C. Wolff-Parkinson-White syndrome type B
D. Monomorphic ventricular tachycardia
E. Polymorphic ventricular tachycardia

A. Lown-Ganong-Levine syndrome

Lown-Ganong-Levine syndrome (LGL) is a pre-excitation syndrome of the heart characterised by a short PR interval and normal QRS width

The key thing to note is the short PR interval. The normal PR interval is 120-200ms. A short PR interval is suggestive of a pre-excitation syndrome. Pre-excitation syndromes occur due to an accessory pathway between the atria with the ventricles, resulting in early depolarisation of the ventricles and a tendency for tachyarrhythmias. Pre-excitation through the accessory pathway results in a short PR interval.

The normal QRS width (normal width <120ms) precludes the diagnosis of Wolff-Parkinson-White syndrome which is characterised by a wide QRS with a slurred upstroke (delta wave). The normal QRS also rules out ventricular tachycardia.

The diagnosis is therefore Lown-Ganong-Levine syndrome (LGL) which is a pre-excitation syndrome of the heart characterised by a short PR interval and normal QRS width.

Question 13 of 129

A 60 year old man with a known history of congestive cardiac failure and asthma is reviewed in a cardiology clinic. He is noted to have a blood pressure of 95/63mmHg and a heart rate of 98bpm. An ECG confirms sinus rhythm. He has previously developed symptoms of wheeze with beta blockade. He is commenced on ivabradine 5mg twice daily by his cardiologist.

Which of the following should the patient be warned of as a recognised side effect of ivabradine?

A. Neutrophilia
B. Hypotension
C. Phosphenes
D. Diaphoresis
E. Renal failure

C. Phosphenes

Ivabradine use may be associated with visual disturbances including phosphenes and green luminescence

Ivabradine is a second line agent for rate control in the treatment of chronic heart failure in those in whom beta-blockade is contraindicated, not tolerated or therapy fails. Other rate limiting drugs such as the calcium channel blockers diltiazem or verapamil may sometimes be used but hypotension often limits their use. Digoxin too can sometimes be used, even in sinus rhythm although the rate limiting effect it has is relatively modest; often digoxin and ivabradine are ultimately used in conjunction.

NICE guidelines on the use of ivabradine give clear criteria on its use; the patient must have at least a moderate degree of heart failure with a left ventricular ejection fracture of <35%, they must be stable on optimal doses of other heart failure medications including angiotensin system modulators, an aldosterone receptor antagonist and a beta blocker if not contraindicated. It is important the patient is regularly monitored by a community heart failure team to titrate the dose. Ivabradine can also only be used in patients in sinus rhythm. It works by selectively inhibiting the If [ionic funny] channel which is a sodium/potassium symporter channel largely expressed within the sinoatrial node. Inhibition of this channel slows the intrinsic rhythmicity of the hearts pacemaker function. Pharmaceutical bradycardia aids in the management of cardiac failure as it allows improved diastolic filling and reduced myocardial oxygen usage.

Side effects of ivabradine include bradycardia, ventricular escape rhythms, dizziness, headache, muscle cramps and eosinophilia. Neutrophilia is rarely seen with ivabradine. Hypotension is also not seen with ivabradine use which makes it an important drug in the arsenal when treating patients with cardiac failure in whom other rate limiting drugs may be relatively contraindicated.

An important side effect with ivabradine however is that of visual disturbance including the phenomenon of phosphenes, or 'flashing lights'. Other visual symptoms described include green discolouration of visual field, blurring of vision and scintillating scotomata. These visual symptoms are usually transient and mild. They arise due to inhibition of similar ionic channels in the retina to the sinoatrial If channels. Approximately 20% of patients taking the drug develop some form of visual disturbance but only 1% need to discontinue therapy because of it.

The use of ivabradine in renal dysfunction is not well established however manufacturers recommend it may be used with estimated glomerular filtration rates above 15ml/min. There is no evidence its use causes renal dysfunction although mild rises in plasma creatinine levels may be seen

Question 14 of 129

A 44-year-old gentleman is admitted to hospital with a 2 week history of flu-like symptoms, wheeze, shortness of breath on lying flat and palpitations. There is no background history of note. His observations include a heart rate of 98/min, blood pressure of 96/50 mmHg and saturations of 96% while on 2L of oxygen. He is apyrexial.

On examination you note a low volume pulse with a raised JVP and a raised Y decent. A third heart sound is audible. There are no murmurs. There are bilateral crepitations and pitting oedema of the legs.

Blood investigations indicate:

Hb	158 g/L	Male: (135-180)Female: (115 - 160)
Platelets	335 * 10 /L	(150 - 400)
WBC	11.7 * 10 /L	(4.0 - 11.0)
Neuts	8.0 * 10 /L	(2.0 - 7.0)
Lymphs	2.3 * 10 /L	(1.0 - 3.5)
Mono	0.7 * 10 /L	(0.2 - 0.8)
Eosin	0.69 * 10 /L	(0.0 - 0.4)

Na	138 mmol/L	(135 - 145)
K	4.2 mmol/L	(3.5 - 5.0)
Urea	5.3 mmol/L	(2.0 - 7.0)
Creatinine	78 µmol/L	(55 - 120)
CRP	12 mg/L	(< 5)

Three blood cultures have been sent and the preliminary report is negative.

The ECG on admission shows sinus rhythm with left ventricular hypertrophy pattern but no features of ischaemia; the PR interval is normal.

An echocardiogram is performed and notes shows preserved systolic function with moderate concentric left ventricular hypertrophy. Both atria are dilated and there is evidence of diastolic dysfunction. There is no valvular pathology and no suspicious lesions surrounding the valves.

Given this scenario, which imaging modality would be most appropriate in identifying the diagnosis of this patient?

A. Transoesophageal echocardiography
B. Right and left cardiac catheterisation
C. Myocardial perfusion scan
D. Cardiac MRI
E. High resolution CT thorax

D. Cardiac MRI

Cardiac MRI is the investigation of choice alongside echocardiography in order to identify the potential cause of cardiomyopathy

The echocardiography and examination findings suggest the potential of restrictive cardiomyopathy. In addition this gentleman has significant eosinophilia which could alert us to the possibility of Loffler's endocarditis.

The options provided cover a wide spectrum of cardiac imaging modalities, however cardiac MRI yields the greatest detail with regards to function and structure of the heart in such scenarios.

The possibility of infective endocarditis remains low and a transoesophageal echocardiogram would not necessarily add anything further with regards to the diagnosis.

A catheterisation or myocardial perfusion scan will provide excellent information regarding ischaemia however the information regarding the cause of this restrictive cardiomyopathy will not be revealed.

There is no indication for a high resolution CT thorax given the high grade of suspicion regarding a cardiac primary problem.

Question 15 of 129

A 53-year-old man with a history of diabetes presents to the Emergency Department with crushing central chest pain, vomiting, and diaphoresis.

Observations :

- Pulse: 32 beats per minute.
- Blood pressure: 70/40 mmHg.
- Oxygen saturation: 88%

Examination reveals pale and cool skin. An ECG shows ST-elevation in leads II, III, and aVF, along with complete heart block, and his troponin test is positive. Following initial management, including percutaneous coronary intervention (PCI) with stenting, he is admitted to the intensive care unit.

Three days post admission, he develops severe dyspnoea, cold extremities, and diaphoresis. Auscultation reveals a holosystolic murmur on the left sternal edge. His observations:

Pulse: 123 per minute Blood pressure: 65/45 mmHg. Oxygen saturation: 80%

What is the most appropriate next step in management?

A. CT coronary angiography
B. Cardiac MRI
C. Immediate surgical consultation
D. Re-transfer to the catheter lab
E. Transthoracic echocardiography

E. Transthoracic echocardiography

Acute heart failure: echocardiography is indicated in new-onset heart failure, cardiogenic shock, suspected valvular or post-MI problems

Transthoracic echocardiography is the correct answer. In this scenario, the patient developed cardiogenic shock (hypotension, tachycardia and cool skin) with a holosystolic murmur following a complicated inferior wall STEMI (as denoted by complete heart block on ECG). The differential diagnoses include acute mitral regurgitation (MR) and acute ventricular septal rupture (VSR). A holosystolic murmur heard over the left sternal edge suggests an acute VSR. An urgent transthoracic echocardiogram is warranted to delineate the site and extent of the defect. This diagnostic step should be performed concurrently with medical therapy, preceding an immediate surgical consultation.

CT coronary angiography is not appropriate in this context. Although CT coronary angiography can be useful for patients with suspected stable angina, in this case, where acute VSR is likely following a wellestablished inferior wall STEMI, echocardiography will provide rapid and precise information about the lesion that will inform surgical management decisions.

Cardiac MRI would not be suitable in this setting. Although it can sometimes provide superior detail compared to echocardiography, its longer scan times, limited availability, and inadequacy for haemodynamically unstable patients render it a less favourable choice for imaging in this emergency.

Immediate surgical consultation plays a pivotal role in managing both acute MR and VSR; however, it should be preceded by a detailed lesion assessment using echocardiography to inform subsequent surgical intervention.

Re-transfer to the catheter lab would not benefit this patient. Such a step may be considered if re-infarction is suspected. Given that the clinical picture points towards an acute VSR post-myocardial infarction, echocardiographic evaluation remains crucial for diagnosis, and coronary angiography will not diagnose such a complication

Question 16 of 129

A 54-year-old female presents with heart palpitations. She has no past medical history and takes no regular medicines. Her cardiorespiratory exam is normal.

An ECG is performed:

Rate and rhythm	145 beats per minute. Regular rhythm
P waves	Not visible
QRS	145ms. RBBB pattern
QTc	420ms
Axis	Right axis deviation

What is the most likely explanation of these ECG results?

A. Ventricular tachycardia
B. Supraventricular tachycardia with bundle branch block
C. Torsades de pointes (TdP)
D. Atrial fibrillation with bundle branch block
E. Supraventricular tachycardi

B. Supraventricular tachycardia with bundle branch block

Distinguishing between VT and SVT with BBB can be challenging. The presence of RBBB and RAD favours a diagnosis of SVT with BBB

The ECG results confirm a broad complex tachycardia with a regular rhythm. The differential is therefore between supraventricular tachycardia (SVT) with bundle branch block (BBB) and ventricular tachycardia (VT). Distinguishing between VT and SVT with BBB can be challenging. The presence of RBBB and RAD favours a diagnosis of SVT with BBB. If you are unsure of the diagnosis, it is always safer to assume the the patient has VT and treat accordingly.

The absence of P waves should make you consider a diagnosis of atrial fibrillation (AF) with BBB, however the regular rhythm precludes this diagnosis. In SVT, the P waves are often buried within the QRS complex.

The normal QTc and regular rhythm precludes the diagnosis of Torsades de pointes (TdP).

Question 17 of 129

A 39-year-old male is admitted with sharp central chest pain which is worse on inspiration. He states that the pain is partially relieved by leaning forward. He has no past medical history and takes no regular medicines.

ECG results are as follows:

PR segment	Depression
ST segment	Global concave ST elevation

Blood results are as follows:

Troponin	9.82 ug/l (normal range 0.015 - 0.045)

What investigation is most useful diagnostically?

A. Cardiac magnetic resonance imaging
B. Serial troponins
C. Transesophageal echo
D. Myocardial Perfusion Imaging (MPI)
E. Serial ECGs

A. Cardiac magnetic resonance imaging

Cardiac magnetic resonance imaging has been shown to be very useful in diagnosing myocarditis by visualising markers for inflammation of the myocardium

The patient has clinical and electrocardiographic features of pericarditis. Although a small rise in troponin can be expected in pericarditis, a significant rise should alert you to the possibility of myopericarditis.

Cardiac magnetic resonance imaging has been shown to be very useful in diagnosing myocarditis by visualising markers for inflammation of the myocardium e.g. regional myocardial oedema, hyperaemia and inflammatory necrosis.

All the other options may be useful, however the most sensitive investigation for diagnosing myopericarditis is cardiac MRI.

Question 18 of 129

A 64-year-old man presents to cardiology outpatients with intermittent exertional chest pain. He has suffered from angina for several years and finds his glyceryl trinitrate spray effective, but he is keen on further treatment to help his symptoms. His past medical history includes angina, asthma, and type 2 diabetes. His current medication includes salbutamol, montelukast, Fostair, metformin, amlodipine, glyceryl trinitrate, aspirin and simvastatin.

Observations are temperature 37ºC, heart rate 52 beats/min, blood pressure 98/59 mmHg, respiratory rate 14 breaths/min, and oxygen saturation 96%.

What is the most appropriate additional therapy?

A. Bisoprolol
B. Isosorbide mononitrate
C. Ivabradine
D. Ranolazine
E. Verapamil

D. Ranolazine

For a patient with symptomatic stable angina on a calcium channel blocker but with a contraindication to a beta-blocker, the next line treatment should be long-acting nitrate, ivabradine, nicorandil or ranolazine

The correct answer is **ranolazine**. This patient has stable angina despite the use of first-line anti-anginal therapy with amlodipine. NICE recommends using either a beta-blocker or a dihydropyridine calcium channel blocker as first-line anti-anginal.

There are a number of options for next-line treatment for patients on a calcium channel blocker, including the addition of a beta-blocker, longacting nitrate, ivabradine, nicorandil or ranolazine. In this case, **ranolazine** would be the most appropriate, as the other options have either cautions or contra-indications.

Given that this patient has asthma, it would be best practice to avoid a beta-blocker such as **bisoprolol**, especially if other equally appropriate therapy options are available.

Given that this patient already has a slightly low blood pressure on the candesartan alone, it would be best to avoid adding in **isosorbide mononitrate** as this risks dropping the blood pressure further.

Ivabradine is contra-indicated in bradycardia. The BNF states 'do not initiate for angina if heart rate below 70 beats per minute'.

The patient is already on a dihydropyridine calcium channel blocker, therefore adding a non-dihydropyridine calcium channel blocker such as **verapamil** should be avoided.

Question 19 of 129

A 35-year-old woman presented to the emergency department with intermittent palpitations, breathlessness, and non-specific chest discomfort. The previous day she had found out that she was 14-weeks pregnant which had caused significant stress and anxiety. She has a long history of anxiety and depression, managed by cognitive behavioural therapy. She reported that she had intermittently had episodes of palpitations and shortness of breath for the past 10 years and had attributed this to her anxiety. These symptoms had worsened over the past couple of weeks and today her symptoms were intolerable. Her medications consisted of over-the-counter vitamin supplements.

She looked anxious, but otherwise, the examination was unremarkable. An ECG was performed:

ECG #1: Normal sinus rhythm. T wave inversion in V1 - V3.

Whilst in the emergency department, she complained of severe palpitations and dizziness.

Her ECG was repeated:

ECG #2: Wide complex regular tachycardia with left bundle branch block (LBBB) pattern. Heart rate 150bpm. Fusion beats and capture beats present.

As she was being transferred into resus, she spontaneously cardioverted back to sinus rhythm.

An echocardiogram was performed:

The right ventricle is dilated with depressed right ventricular systolic function and localised apical aneurysm. Left ventricular size and function are normal.

What is the most likely cause of her presentation?

A. Aortic dissection
B. Takutsubo cardiomyopathy
C. Peripartum cardiomyopathy
D. Arrhythmogenic right ventricular cardiomyopathy
E. Brugada syndrome

D. Arrhythmogenic right ventricular cardiomyopathy

Arrhythmogenic right ventricular cardiomyopathy - T wave inversion in V1-3

Arrhythmogenic right ventricular cardiomyopathy (ARVC) is characterised by the replacement of the myocardium of the right ventricle with fibrofatty tissue. The abnormal fibrofatty tissue predisposes to ventricular tachycardia (VT), right ventricular systolic dysfunction, and can cause sudden death. It is often inherited but can be sporadic. Presentation is often with nonspecific palpitations, chest discomfort, and syncope. This patient has a typical presentation: the pregnancy-associated increase in stroke volume is likely the trigger for her symptoms recently worsening. Her two ECGs are characteristic: typical changes include inverted T waves in the precordial leads V1-V3 (looking at the right ventricle) with episodes of ventricular tachycardia originating in the right ventricle (giving the ECG a LBBB morphology as the arrhythmia takes an abnormal route of conduction from right to left through slowly-conducting myocardium rather than conducting tissue). Epsilon waves may also be seen in ARVC. Her echocardiogram is also characteristic - infiltration and thinning of the right ventricular wall can occur to the point that systolic function is impaired and an aneurysm forms.

Note that the differential for a wide-complex regular tachycardia on an ECG includes SVT with aberrant conduction (as well as VT). However, the fusion beats and capture beats are more in keeping with VT in this case.

This patient's presentation is not typical for aortic dissection. An echo might see cardiac tamponade or new aortic regurgitation if this was the diagnosis but this is not the case here.

Takutsubo cardiomyopathy is caused by transient systolic dysfunction of the left ventricle (LV), presenting similarly to an acute coronary syndrome. Patients typically have chest pain, a raised troponin, and ECG findings consistent with ischaemia such as ST elevation/T wave inversion. This is typically triggered by recent physical or emotional stress. Patients are initially managed as an ACS but cardiac angiography shows no significant coronary artery disease. Instead, the LV wall shows hypokinesis or akinesis which creates the shape of an 'octopus pot' during systole. It may be argued that this patient has experienced a recent stressor (the news of pregnancy) however her presentation, long history of symptoms, and echo findings are more in keeping with AVRC.

Peripartum cardiomyopathy (PPCM) is a rare type of cardiomyopathy that is often dilated and occurs in late pregnancy or early postpartum (typically one month before or after birth). The aetiology is unclear but is thought to be multifactorial. Criteria for diagnosis of PPCM include impairment of LV systolic function (LVEF <45%) which was not the case in this patient. Furthermore, this patient presented in the early second trimester - too early for PPCM but late enough for pregnancy-related haemodynamic changes to trigger her underlying ARVC.

Lastly, Brugada syndrome is another important differential to consider in young patients with palpitations, ECG changes in V1-V3, and episodes of VT. Brugada is caused by an inherited mutation in the cardiac sodium channel. However, echocardiography is typically normal and an ECG may show unusual ST elevation in V1-V3 with T wave inversion in these leads.

Question 20 of 129

A 67-year-old woman with recurrent pulmonary emboli secondary to antiphospholipid syndrome presents with worsening dyspnoea on exertion. The dyspnoea is accompanied by chest pain when she climbs the stairs.

On examination, she is on 2L oxygen and saturating at 94%, there is cyanosis seen under the tongue, a raised jugular venous pressure is seen and a right ventricular heave is felt.

Troponin	3 ng/L	(<14)
Pro-Brain Natriuretic Peptide	2300 pg/ml	(<100)

The CT pulmonary angiogram does not demonstrate any large pulmonary emboli. An echocardiogram shows right ventricle hypertrophy and moderate tricuspid regurgitation. Upon right heart catheterisation, the pressures in the pulmonary artery are 30mmHg and after inhaled nitric oxide drops to 20mmHg.

What is the next best management option?

A. Bosentan
B. Iloprost
C. Increase apixaban dose
D. Nifedipine
E. Sildenafil

D. Nifedipine

Pulmonary arterial hypertension patients with positive response to vasodilator testing should be treated with calcium channel blockers

Nifedipine is the correct option as an oral calcium channel blocker to treat this vasodilator (nitric oxide) responsive form of pulmonary arterial hypertension. The calcium channel blocker has proven efficacious in dilating the pulmonary vasculature to a sufficient degree to improve her symptoms.

Bosentan is an incorrect option as it is an endothelin receptor antagonist, which is used in patients who do not respond to vasodilator testing.

Iloprost is an incorrect option as it is a prostacyclin analogue, best utilised when the patient has not responded to vasodilator testing.

Sildenafil is an incorrect option as this would be a viable treatment option for the majority of patients because the majority do not respond to vasodilator testing. Sildenafil is a phosphodiesterase type 5 inhibitor.

Increase apixaban dose when the patient is on the recommended dose is an incorrect option because this will be an unlicensed use of the apixaban and would not reverse pulmonary hypertension which has likely been established from previous, recurrent pulmonary emboli.

Question 21 of 129

A 55-year-old man presents to the emergency department with sudden cardiac arrest. The patient has no known history of heart disease. The initial pulse and rhythm analysis confirms pulseless ventricular tachycardia.

What medications are indicated as part of the advanced life support algorithm?

A. Adrenaline after third shock
B. Adrenaline and amiodarone after third shock
C. Adrenaline and amiodarone immediately
D. Adrenaline immediately
E. None

B. Adrenaline and amiodarone after third shock

In ALS, amiodarone 300 mg should be given to patients who are in VF/pulseless VT after 3 shocks have been administered.

This patient has a shockable rhythm (pulseless ventricular tachycardia (VT)) and therefore requires adrenaline and amiodarone to be administered after the third cycle.

Adrenaline after the third cycle is incorrect; this should be given with amiodarone for shockable rhythms.

As per the resuscitation guidelines, giving **adrenaline + amiodarone immediately** is incorrect as these should only be given after three cycles.

Adrenaline immediately is incorrect as this is the treatment for nonshockable rhythms, however pulseless VT is considered a shockable rhythm.

To give **none** is incorrect as the resuscitation guidelines state that both adrenaline and amiodarone have a role in shockable cardiac arrests.

Question 22 of 129

A 64-year-old man is reviewed on the coronary care unit and is making a good recovery 2 days post-myocardial infarction. On examination, he appears well and is haemodynamically stable. His heart rate is 72 beats/min and his blood pressure is 132/84 mmHg.

A 12-lead ECG is performed and shows isorhythmic dissociation between P waves and QRS complexes. The QRS complexes are wide and fusion beats are visible on the ECG.

Given the likely diagnosis, which of the following is the most appropriate management?

A. Amiodarone
B. Atropine
C. Discharge with standard follow up
D. Magnesium
E. Pacemaker insertion

C. Discharge with standard follow up

Accelerated idioventricular rhythm is common and unconcerning following recent MI

The diagnosis here is accelerated idioventricular rhythm (AIVR). This is a benign ectopic rhythm of ventricular origin. It usually occurs with reperfusion following a myocardial infarction. It is differentiated from ventricular bradycardias and tachycardia by its normal rate - in this case, 72 beats/min. Reperfusion of ischaemic tissue leads to increased depolarisation rate of ventricular myocytes, which then override the sinoatrial node, leading to dissociation between the atrial and ventricular rhythms as seen here (P waves and QRS are isorhythmically dissociated). As the patient is hemodynamically stable, treatment is not necessary;

AIVR is generally self-limiting.

Amiodarone would be used in the cases of ventricular tachycardia. Although there are some overlapping features on the ECG, the patient's heart rate is normal and AIVR is strongly suggested here. Antiarrhythmics are therefore unnecessary.

Atropine is occasionally used to increase the sinus rate and overcome AIVR, however, this is mainly in symptomatic patients. Generally, the condition is self-limiting and in hemodynamically stable patients no treatment is required.

Magnesium is used to treatment torsades de pointes, a polymorphic form of ventricular tachycardia which is recognisable on ECG. It plays no role in the management of AIVR.

Pacemaker insertion is unwarranted. As described above, this condition is self-limiting.

Question 23 of 129

You receive a phone call requesting advice from a GP. A patient who is on long term warfarin for atrial fibrillation has been found to have an INR of 10.0 following a recent course of antibiotics. She is not bleeding and only has long standing senile purpura on her arms with no new bruising.

What is the most appropriate advice to give?

A. Admit for intravenous vitamin K and monitoring
B. Give oral vitamin K and continue warfarin at usual dose
C. Give oral vitamin K and stop warfarin until INR < 5
D. Give oral vitamin K and stop warfarin until INR < 3
E. Stop warfarin and restart when INR < 3

C. Give oral vitamin K and stop warfarin until INR < 5

INR > 8.0 (no bleeding) - stop warfarin, give oral vitamin K 1-5mg, repeat dose of vitamin K if INR high after 24 hours, restart when INR < 5.0

The correct answer is to **give oral vitamin K and stop warfarin until INR < 5**. This is because the patient has a significantly elevated INR of 10.0 but no active bleeding or high risk of bleeding. According to the UK guidelines, in this situation, it is appropriate to give oral vitamin K (usually 1-5 mg) and withhold warfarin until the INR is below 5. This will allow for a rapid reduction in INR and reduce the risk of bleeding complications.

The option **admit for intravenous vitamin K and monitoring** is not the most appropriate choice in this case, as the patient does not have any active bleeding or high risk of bleeding. Intravenous vitamin K and hospital admission are generally reserved for patients with lifethreatening bleeding or an extremely high risk of bleeding.

The option **give oral vitamin K and continue warfarin at usual dose** would not be suitable, as continuing warfarin without interruption would maintain an elevated INR level, increasing the risk of bleeding complications. It is important to withhold warfarin temporarily to allow the INR to decrease.

The option **give oral vitamin K and stop warfarin until INR < 3** might seem reasonable; however, stopping warfarin until an INR of less than 3 could lead to subtherapeutic anticoagulation levels for this patient with atrial fibrillation. The goal therapeutic range for most patients on warfarin with atrial fibrillation is an INR between 2 and 3. Stopping warfarin until the INR falls below this range may increase their thromboembolic risk.

Finally, choosing to simply **stop warfarin and restart when INR < 3**, without administering oral vitamin K, would result in a slower reduction of INR. This could leave the patient at an increased risk of bleeding complications for a longer period. Oral vitamin K helps to lower the INR more rapidly and safely.

Question 24 of 129

A 65-year-old man is reviewed in the Emergency Department. Twelve months ago he had a prosthetic aortic valve replacement for progressive aortic stenosis. Since the operation he has been well but over the past two weeks he has been complaining of lethargy, fever and anorexia. Blood tests show a CRP of 215 mg/l. The chest x-ray and urine dip are unremarkable.

Given the suspicion of infective endocarditis an transoesophageal echocardiogram is performed which demonstrates a large vegetative lesion around the prosthetic aortic valve.

What is the most appropriate antibiotic treatment whilst awaiting blood cultures?

A. IV amoxicillin
B. IV teicoplanin + vancomycin
C. IV flucloxacillin + low-dose gentamicin
D. IV vancomycin + rifampicin + low-dose gentamicin
E. IV teicoplanin + rifampicin

D. IV vancomycin + rifampicin + low-dose gentamicin

IV vancomycin + rifampicin + low-dose gentamicin is the empirical treatment of choice in prosthetic valve endocarditis

The correct answer is **IV vancomycin + rifampicin + low-dose gentamicin**. In a patient with a prosthetic valve and suspected infective endocarditis, the most likely causative organisms are staphylococci (including methicillin-resistant Staphylococcus aureus [MRSA]), streptococci, and enterococci. The combination of IV vancomycin, rifampicin, and low-dose gentamicin provides coverage against these organisms while awaiting blood culture results. Vancomycin is effective against MRSA and other gram-positive bacteria, rifampicin has good penetration into biofilms on prosthetic material and enhances the activity of vancomycin, and gentamicin provides additional synergistic bactericidal activity.

IV amoxicillin is not the most appropriate choice in this case because it does not provide adequate coverage for MRSA or resistant enterococci. Amoxicillin is typically used as part of empirical treatment for native valve endocarditis caused by streptococci but would not be sufficient for a prosthetic valve infection where staphylococcal involvement is more common.

IV teicoplanin + vancomycin would not be an appropriate choice because combining two glycopeptides offers no additional benefit over using just one of them. Both teicoplanin and vancomycin have similar spectra of activity against gram-positive bacteria; however, they do not cover gram-negative organisms or provide synergy with other antibiotics.

IV flucloxacillin + low-dose gentamicin would be inadequate in this situation as flucloxacillin does not cover MRSA or resistant enterococci. Flucloxacillin is effective against methicillin-sensitive staphylococci but would not provide broad enough coverage for a prosthetic valve infection where resistant organisms are more likely to be involved.

IV teicoplanin + rifampicin is not the best choice because it lacks the synergistic bactericidal activity provided by the addition of low-dose gentamicin. Although teicoplanin and rifampicin would cover most gram-positive organisms, including MRSA, the absence of gentamicin could result in suboptimal treatment for enterococci and other resistant bacteria.

In conclusion, **IV vancomycin + rifampicin + low-dose gentamicin** is the most appropriate empirical antibiotic treatment for a patient with suspected infective endocarditis involving a prosthetic valve. This regimen provides broad coverage against common causative organisms while awaiting blood culture results and can be adjusted based on culture sensitivities once available.

Question 25 of 129

A 71-year-old patient presents to the Emergency Department with a 30 minute history of crushing central chest pain. ECG shows tall R waves in V1-2.

Which coronary territory is likely to be affected?

A. Lateral
B. Posterior
C. Anteroseptal
D. Anterolateral
E. Inferior

B. Posterior

Posterior MI typically present on ECG with tall R waves V1-2

Question 26 of 129

A 64-year-old man with mitral regurgitation attends the cardiology clinic for review. An ECG reveals new atrial fibrillation.

He is well and denies any shortness of breath or chest pain. His blood pressure is 115/98 mmHg and his heart rate is 64 beats per minute. On examination, he has an audible S3 and a 3/5 pansystolic murmur heard loudest in the mitral area, his apex beat is displaced.

A recent echo showed a regurgitant mitral valve with evidence of left atrial and ventricular dilatation but a preserved ejection fraction.

His current medications include naproxen for osteoarthritis and overthe-counter vitamin D supplements.

From the following options, what would be the most appropriate next step?

A. S Adopt a 'watch and wait' approach with serial yearly echocardiography
B. Commence bisoprolol
C. Commence sacubitril-valsartan
D. Refer for cardioversion
E. Refer for mitral valve replacementubmit answer

New AF in mitral regurgitation -> refer for mitral valve replacement

Refer for mitral valve replacement is the correct answer. This patient has evidence of asymptomatic severe mitral regurgitation as indicated by narrow pulse pressure, audible S3 and evidence of left ventricular dilatation. Given he has now developed atrial fibrillation on a background of severe mitral regurgitation he should be referred for consideration of a valve repair or replacement.

Commence bisoprolol is incorrect. The patient in this scenario has a preserved ejection fraction as indicated by the echo findings. If there was evidence of reduced ejection fraction current NICE guidelines advises considering starting a beta-blocker and ACE inhibitor.

Commence sacubitril-valsartan is incorrect. This would be considered a second-line treatment for heart failure with reduced ejection fraction. In this case, the patient has a preserved ejection fraction.

Adopt a 'watch and wait' approach with serial yearly echocardiography is incorrect. This could be considered as per patient preference, but in the case of severe mitral regurgitation with the development of new atrial fibrillation patients should be offered a referral for mitral valve replacement.

Refer for cardioversion is incorrect. In this case, the patient has known mitral regurgitation. Mitral disease is associated with the development of atrial fibrillation due to the associated structural changes in the left atrium. There is a much higher chance of maintaining sinus rhythm with mitral valve replacement compared to cardioversion. Mitral valve replacement is therefore the preferred option.

Question 27 of 129

You are the on-call medical doctor called to review a patient in the Emergency Department. A 54 year old male patient with a past history asthma, ischaemic heart disease and transient ischaemic attack has presented with palpitations. His admission ECG shows a regular, narrow complex tachycardia. Vagal manoeuvres have been tried in the department with no success. You decide to give intravenous adenosine in an attempt to chemically cardiovert. An initial dose of 6mg is given into a proximal vein with a large flush. The patient soon loses consciousness and an 11 second ventricular standstill is noted on the rhythm strip before slow return of sinus rhythm.

Which of the patient's medications is most likely to be responsible for this?

A. Phyllocontin
B. Dipyridamole
C. Bisoprolol
D. Simvastatin
E. Montelukast

B. Dipyridamole

The effects of adenosine are enhanced by dipyridamole

Adenosine is a useful medication in the investigation and treatment of regular narrow complex tachycardias. With reference to the Resuscitation UK guidelines, it has a place as a second line measure to vagal manoeuvres when no 'adverse features' are present. Adenosine works by transiently blocking the AV node by effect on the A1 receptor, causing potassium efflux and hyperpolarisation.

Dont be side-tracked by the fact that this patient is asthmatic, yes this is a relative contraindication to the administration of adenosine, but that is not the question. Theophyllines have been known to antagonise adenosine necessitating higher dosages. Dipyridamole blocks the cellular uptake of adenosine, increasing concentration at receptors and potentiating its effect. Adenosine should therefore be used with caution, if at all, in patients taking dipyridamole, and certainly low starting doses should be considered.

Question 28 of 129

A 65-year-old man is referred to cardiology outpatient clinic with exertional shortness of breath. He becomes breathless after ascending a single flight of stairs. He has a background of ischaemic heart disease and heart failure with reduced ejection fraction (30%). He is on aspirin, bisoprolol, ramipril, spironolactone, atorvastatin and lansoprazole. He does not smoke or drink alcohol.

His observations are heart rate 63 beats per minute, blood pressure 120/77 mmHg, respiratory rate 18/minute, oxygen saturations 96% on room air and temperature 37ºC.

On examination, he is euvolemic. Chest auscultation is normal. There is no peripheral oedema. The pulse is regular.

From the listed options, what is the best medication choice to improve his symptoms?

A. Add ivabradine
B. Add nicorandil
C. Add sacubitril-valsartan
D. Replace ramipril with ivabradine

E. Replace ramipril with sacubitril-valsartan

E. Replace ramipril with sacubitril-valsartan

Sacubitril-valsartan is considered in heart failure patients with a LVEF < 35% who are still symptomatic on ACE-inhibitors & betablockers

Replace ramipril with sacubitril-valsartan is the correct answer. This man has NYHA class II/III heart failure not controlled with an ace inhibitor, beta blocked and mineralocorticoid receptor antagonist. Given his ejection fraction is < 35%, replacing ramipril with sacubitril-valsartan is a suitable option to improve the clinical situation.

Add ivabradine is incorrect. This would be an option if his heart rate was > 75 beats per minute.

Add sacubitril-valsartan is incorrect. This should replace an ace inhibitor rather than be added to this treatment.

Replace ramipril with ivabradine is incorrect. This is not a substitution recommended by guidance and the patient's heart rate is too low for the addition of ivabradine.

Add nicorandil is incorrect. This medication is used in the treatment of stable angina, rather than heart failure.

Question 29 of 129

A 72-year-old man is reviewed in the cardiology outpatient department with worsening shortness of breath, which now occurs at rest. He has a past medical history of heart failure with reduced ejection fraction and ischaemic heart disease. His medications include bisoprolol, ramipril, and spironolactone.

On examination, he is clinically euvolemic.

An ECG demonstrates a QRS of 140ms and a left bundle branch block.

What intervention is most likely to improve the patient's symptoms?

A. Amiodarone
B. Biventricular pacemaker
C. Dual chamber pacemaker
D. Implantable cardiac defibrillator
E. Single chamber atrial pacemaker

B. Biventricular pacemaker

Cardiac resynchronisation therapy can be used in patients with a QRS duration of >130 msec and LBBB morphology to improve symptomatology

Biventricular pacemaker is correct. Cardiac synchronization therapy (CRT) with a biventricular pacemaker has been shown to improve heart failure symptoms in those patients with a QRS duration > 130ms and LBBB morphology, inadequately controlled on optimal medical therapy. He has NYHA class IV heart failure as he has symptoms at rest. Typically, leads of this pacemaker are placed in both ventricles and often one in the right atrium in addition. They are designed to help the heart contract more efficiently

Amiodarone is incorrect. This is typically used to control abnormal tachyarrhythmias. It has not been shown to improve symptoms in patients with chronic heart failure.

Dual chamber pacemaker is incorrect. This type of pacemaker paces both the right atrium and right ventricle. It is indicated in conditions such as high-grade atrioventricular block rather than advanced heart failure as in this patient.

Implantable cardiac defibrillator is incorrect. This is typically indicated in patients with serious ventricular arrhythmias at risk of sudden death. It can be used for some patients with NYHA class I-III heart failure and a prolonged QRS 120-149ms but without LBBB. However, if there is an LBBB morphology as in this case then a CRT is preferred to improve the symptoms of heart failure.

Single chamber atrial pacemaker is incorrect. This is a pacemaker that usually paces just the right atrium. This type of pacemaker has largely fallen out of favour as they require intact atrioventricular conduction and do not safeguard against this failure in the future. Typically, this type of pacing would only be used for severe symptomatic sinus bradycardia or sick sinus syndrome. It would not result in the required synchronization of ventricular contraction which would result in improved symptoms of heart failure

Question 30 of 129

A 56-year-old gentleman has presented with central chest tightness with a strangling feeling in his jaw. This started four hours ago and he has become increasingly breathless. He feels clammy and has vomited twice. He felt well prior to today and only has a history of refractory hypertension.

On examination, he is alert with a Glasgow Coma Scale of 14/15. He is clutching his chest with one hand. Both heart sounds are present with a loud pan systolic murmur and there is air entry heard throughout. His JVP is raised at 4cm and there are cannon V waves present. There is no ankle swelling or oedema. His liver is palpable and pulsatile. His blood pressure is 190/110 mmHg, heart rate 90/min and he is saturating at 92% in

Hb	130 g/l	Na	139 mmol/l
Platelets	356 * 10 /l	K	4.6 mmol/l
WBC	13.4* 10 /l	Urea	5.4mmol/l
Neuts	9.4 * 10 /l	Creatinine	76 µmol/l
Lymphs	2.3 * 10 /l	CRP	14 mg/l
Troponin	1640 ng/ml (normal range <20)		
ECG	ST depression in leads V1-V3, sinus rhythm		
Chest X-ray	enlarged cardiac shadow		

What is the likely diagnosis?

A. Infective endocarditis
B. Right ventricle myocardial infarction
C. Anteroseptal myocardial infarction
D. Dilated cardiomyopathy
E. Aortic dissection

B. Right ventricle myocardial infarction

Right ventricle infarction -> tricuspid regurgitation

This gentleman presents with acute onset chest pain and has been well prior to this. Infective endocarditis would present more insidiously and is typically painless. Cardiomyopathy does not present with chest pain. Aortic dissection usually has a tearing pain that radiates to the back. The ECG changes represent reciprocal changes seen in a posterior myocardial infarction. The cannon V waves are pathognomonic for a right sided heart failure which occurs during infarction of the right ventricle. Furthermore, the pansystolic murmur would reflect tricuspid regurgitation. Both these features occur in right ventricle infarcts.

Question 31 of 129

You review a patient in cardiology outpatient clinic. He has a left ventricular ejection fraction of 15% on maximal medical therapy. On reviewing his ECG you note a QRS duration of 135ms without evidence of left bundle branch block. Upon taking a history you note the patient is breathless at rest and struggles to walk around his bungalow.

Which implantable device would be appropriate to consider?

A. Cardiac resynchronisation therapy without defibrillator
B. Cardiac resynchronisation therapy with defrifibrillator
C. VVI pacemaker
D. No implantable device recommended
E. Implantable cardioverter defibrillator alone

A. Cardiac resynchronisation therapy without defibrillator

The patient has New York Heart Association Class IV symptoms as he is limited by breathlessness at rest and on minimal exertion. The NICE guidance on implantable devices and resynchronisation therapy would suggest cardiac resynchronisation therapy (without defibrillator) in the above case as QRS is between 120 and 149ms without evidence of left bundle branch block.

VVI pacing is useful in atrial fibrillation with slow ventricular response.

NICE TA314: Implantable cardioverter defibrillators and cardiac resynchronisation therapy for arrhythmias and heart failure.

Question 32 of 129

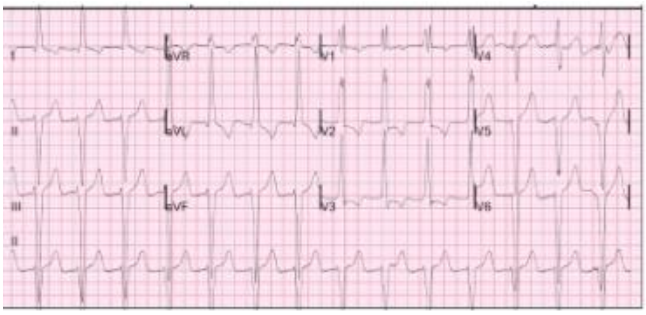
A 70-year-old presents to the outpatients' department having been referred by his GP with on-off palpitations for several months. The patient has a myocardial infarct over 20 years ago which was successfully managed via thrombolysis but otherwise, he has no significant past medical history.

The patient reports he has spontaneous episodes of palpitations approximately once every 2 weeks, and occasional feeling faint. He fainted once when walking quickly for the bus and so now takes it slowly when exerting himself and has no further similar episodes. He is otherwise very well and denies any episodes of chest pain etc.

He is on secondary preventive medication including:

- Aspirin low dose
- Bisoprolol 10mg twice daily
- Atorvastatin 40mgs once daily
- Ramipril 10mg once daily

His examination and observation are normal and an ECG performed is shown below.



What is the most appropriate management plan for the patient?

A. Admit for urgent ablation workup
B. Admit for urgent pacemaker workup
C. No additional management is required
D. Reduce the dose of his beta-blocker
E. Arrange for urgent 24-hour Holter monitor

B. Admit for urgent pacemaker workup

The patient's ECG show trifascicular block with a right bundle branch block (RBBB), left anterior hemiblock and 1st-degree heart block. This with the history of pre-syncope and syncope is concerning and is an indication for admission and pacemaker workup as the patient is at risk of further significant arrhythmias or cardiac blocks.

Trifascicular block is a ventricular escape rhythm that usually arises from the left anterior or posterior fascicle regions following ischaemic heart disease or structural heart disease. As such ablation is not a viable treatment option. Symptomatic trifascicular block is an indication for implantable cardiac pacemaker or permanent pacemaker work up.

The patient has concerning features of symptomatic trifascicular block with pre-syncope and syncope on exertion which warrants urgent assessment for a pacemaker.

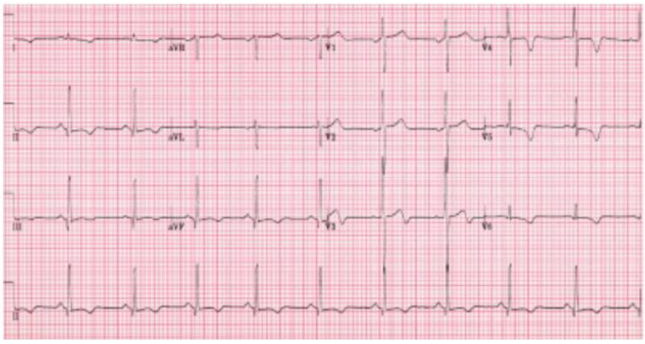
The patient's trifascicular block is very unlikely to be associated with his beta-blocker medication and therefore reducing the dose will not remove the risk of further significant arrhythmias or cardiac blocks.

Arranging for an urgent 24-hour Holter monitor is unnecessary as the patient's ECG is unlikely to change and the current risk of arrhythmias or heart block requires urgent intervention

Question 33 of 129

A 34-year-old is brought into the emergency department following a syncope episode whilst playing tennis. The patient reports he is now feeling well with no symptoms. On further questioning, he only plays tennis occasionally and does not exercise regularly. He reports experienced shortness of breath and chest discomfort when playing and has had a few episodes of feeling light-headed but has never fainted before. He is otherwise well with no recent illness or significant past medical history.

Observations are all within normal range and an ECG performed is shown below



What is the patient's most likely diagnosis?

A. Brugada syndrome
B. Dilated cardiomyopathy
C. Hypertrophic obstructive cardiomyopathy
D. Arrhythmogenic right ventricular dysplasia (ARVD)
E. Wellen's syndrome

C. Hypertrophic obstructive cardiomyopathy

This patient has presented with features in keeping with hypertrophic obstructive cardiomyopathy (HOCM). The condition is due to a genetic defect that causes a disorder of the cardiac muscle and, although patients can be asymptomatic, it is the commonest cause of sudden cardiac death in the young. Patients who do present with symptoms typically experience exertional dyspnoea, angina and syncopal episodes, commonly following or during exertion. ECG findings are of left ventricular hypertrophy, non-specific ST-segment and T-wave abnormalities, progressive T wave inversion and deep Q waves. On this patient's ECG widespread T wave inversion can be seen, especially in lead I & II as well as non-specific ST-segment changes in V2 & 3. The diagnosis can be confirmed via cardiac echo and management is dependant on the extent of obstruction and patient symptoms.

Brugada syndrome is another genetic cardiac disorder however unlike HOCM it results in electrical activity disorders and not structural issues. Features include syncope and sudden cardiac death however it is not always associated with exertion or exercise. There are several types of Brugada syndrome, each with variations seen on ECG. However, the only potentially diagnostic ECG abnormality is of coved ST-segment elevation in 2 or more of V1-3 followed by a negative T wave. This is classical of Brugada type 1 syndrome and is commonly known as the Brugada sign.

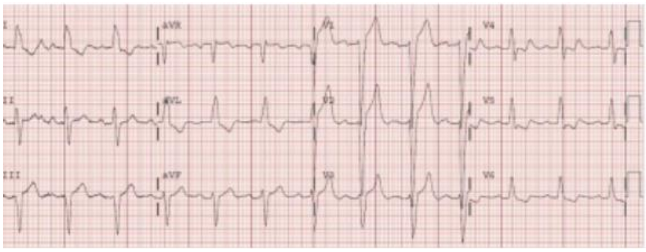
Dilated cardiomyopathy is a disease of the myocardial and characterised by progressive ventricular dilation and dysfunction. Presentation is normal with worsening biventricular failure including peripheral and pulmonary oedema. Syncope episodes are rare and symptoms are not fluctuant, as seen in this patient. ECG abnormalities are those associated with atrial and ventricular hypertrophy with conduction delays (e.g. LBBB), left axis deviation and poor R wave progression.

Arrhythmogenic right ventricular dysplasia (ARVD), also known as arrhythmogenic right ventricular cardiomyopathy (ARVC), is a result of a genetic defect affecting the desmosomes of the myocardium. ARVD results in non-ischemic cardiomyopathy, mainly affecting the right ventricle. The condition leads to hypokinetic areas of the ventricle wall and myocardium fibrofatty replacement with which results in associated arrhythmias. Patients typically present with palpitations, syncope, and potentially sudden cardiac death however ECG findings are of T wave inversion in leads V1 to V3 and of right bundle branch block which are not present in this case.

Wellen's syndrome is the ECG pattern of biphasic or deeply inverted T waves in the chest leads V2-3. It is highly specific for critical stenosis of the left anterior descending artery and therefore is normally seen in patients presenting with ischaemic-like symptoms

Question 34 of 129

A 76-year-old man is brought to the Emergency Department by the paramedics after experiencing an episode of chest pain. This came whilst he was walking his dog and lasted around 20 minutes. He is now pain free. The paramedics report that his blood pressure is 126/88mmHg, pulse 75/min and oxygen saturations 98% on room air. On examination you note a systolic murmur. An ECG is requested:



What does the ECG show?

A. Hypertrophic obstructive cardiomyopathy
B. Anterior myocardial infarction
C. Left bundle branch block
D. Brugada syndrome
E. Arrhythmogenic right ventricular cardiomyopathy

C. Left bundle branch block

The ECG shows typical changes of left bundle branch block (LBBB). The systolic murmur may be explained by aortic stenosis, a known association of LBBB. Cardiac ischaemia should of course be considered in patients with LBBB, particularly if it is new. A troponin level will help clarify matters but patients with ongoing chest pain and LBBB should be transferred to the cath lab.

Question 35 of 129

A 66-year-old gentleman requests to see his general practitioner, concerned regarding one of the medications that he was started on following a myocardial infarction. He tells you that the cardiology team has started him on atorvastatin, which he does not wish to take unless there is no other alternative, as he has had relatives that had bad experience with simvastatin, including muscle pain and disturbed sleep.

He has no family history of hypercholesterolaemia at a young age, but a number of his relatives are on statins following cardiac events.

What options exist with regards to his secondary prevention?

A. Ezetimibe 10 mg OD
B. Continue with atorvastatin at the current dose
C. Fenofibrate 160 mg OD
D. Lomitapide 5 mg OD
E. Niacin (as per product literature)

B. Continue with atorvastatin at the current dose

Statins are the only lipid-regulating drugs that are used in secondary prevention of cardiovascular disease (with the exception of ezetimibe in cases of primary hypercholesterolaemia)

With regards to secondary prevention the only NICE approved medication at the moment is statins. None of the other drugs mentioned above are licensed for secondary prevention with the exception of ezetimibe in primary hypercholesterolaemia. There is no indication, in a 66 year old gentleman that he is suffering from primary hypercholesterolaemia. As no dose is given in the question, one should assume that continuing at the current dose provided that the patient understands that no alternative exists is the right answer. Indeed if a dose was given, reducing the dose or switching to an even more hydrophilic agent (such as pravastatin) would be an appropriate option.

Question 36 of 129

A 50-year-old patient with no prior medical history presents to the Emergency Department with crushing central chest pain. On examination you find:

- Respiratory rate - 22/min
- Oxygen saturations 95% on room air Chest clear
- Pulse 118bpm
- Blood pressure 92/61mmHg
- JVP elevated 4cm above sternal angle

An ECG done in the department shows 3mm ST segment elevation in leads II, III and aVF. The patient undergoes a primary PCI to his right coronary artery with a good angiographic result. An echocardiogram is performed the following day.

What would you expect to see?

A. Regional wall motion abnormality in the inferior wall of the LV
B. Regional wall motion abnormality in the inferior wall of the LVwith a dilated and impaired RV
C. Normal biventricular function
D. Constrictive pericarditis
E. Regional wall motion abnormality in the inferior wall of the LVwith mitral regurgitation

B. Regional wall motion abnormality in the inferior wall of the LVwith a dilated and impaired RV

This patient has had an inferior STEMI, as evidenced by the fact that he has ST elevation is leads II, III, aVf. Given this presentation, you would expect an echocardiogram to show a regional wall motion abnormality (RWMA) in the inferior wall of the LV. However, there are some additional key points in his examination findings that are necessary to appreciate in order to correctly answer this question. Firstly, he has significant hypotension which is not usual in the setting of an uncomplicated inferior STEMI. In addition, the patient has an elevated JVP which is indicative of elevated RV filling pressures and therefore RV failure. The fact that he has a clear chest indicates that he has relatively normal LV filling pressures, thus ruling out RV failure secondary to LV failure. Putting these findings together, the diagnosis is an inferior STEMI with right ventricular infarction. Therefore you would expect an echo to demonstrate an inferior LV wall RWMA with a dilated and impaired RV.

Question 37 of 129

A 74-year-old man was admitted with chest pain. He had a past history of type 2 diabetes mellitus and hypertension. He underwent coronary angiography and a stent was inserted into his right coronary artery. Following the procedure he was noted to be oliguric.

On examination his blood pressure was 148/80 mmHg and he was euvolaemic. Mottled skin changes were noted over his legs.

Na +	138 mmol/L	(135 - 145)
K +	5.6 mmol/L	(3.5 - 5.0)
Bicarbonate	19 mmol/L	(22 - 29)
Urea	17.6 mmol/L	(2.0 - 7.0)
Creatinine	228 µmol/L	(55 - 120)
Creatine kinase	560 U/L	(24-170)
Hb	142 g/L	Male: (135-180)Female: (115 - 160)
Platelets	380 * 10 /L 9	(150 - 400)
WBC	13.6 * 10 /L 9	(4.0 - 11.0)
Neuts	9.1 * 10 /L 9	(2.0 - 7.0)
Lymphs	2.5 * 10 /L 9	(1.0 - 3.5)
Eosin	1.2 * 10 /L 9	(0.0 - 0.4)

What is the most likely diagnosis?

A. Contrast-induced nephropathy
B. Acute interstitial nephritis
C. Cholesterol emboli
D. ANCA associated vasculitis
E. Rhabdomyolysis

C. Cholesterol emboli

AKI following angiography can be caused by contrast-induced nephropathy or cholesterol emboli

Although the commonest cause of acute kidney injury following angiography is contrast-induced nephropathy, the raised eosinophil count and skin rash (livedo reticularis) is highly suggestive of cholesterol emboli.

Acute interstitial nephritis can cause eosinophilia but is not associated with contrast.

ANCA associated vasculitis can cause a rash and eosinophilia (e.g. Churg Strauss) but there are no other features such as anaemia or systemic upset to fit with this.

The elevated creatine kinase suggests muscle injury, which would fit with an embolic phenomenon but the CK is not high enough to diagnose rhabdomyolysis (usually >5000).

Question 38 of 129

A 59-year-old man presents with stridor which came on gradually over the course of the day. On examination you note swelling of the tongue and the mucous membranes of the oropharynx. He has a past medical history of rheumatoid arthritis, type 2 diabetes mellitus and heart failure.

He was recently started on a new medication.

What medicine would be the most likely cause of this presentation?

A. Nimodipine

B. Losartan
C. Lisinopril
D. Prednisolone
E. Sitagliptin

C. Lisinopril

Drug-induced angioedema without urticaria is most frequently associated with angiotensin converting enzyme inhibitors or, less frequently, angiotensin II receptor blockers

Angioedema is a well recognised side effect of angiotensin-converting enzyme (ACE) inhibitor therapy. Angioedema can also be seen with angiotensin receptor blocker (ARB) therapy but much less frequently than is the case with ACE inhibitors. Therefore lisinopril is a better answer than losartan.

ACE inhibitors block the enzyme ACE resulting in increased levels of bradykinin. Bradykinin then accumulates potentially resulting in angioedema.

All the other options can cause peripheral oedema, however not specifically angioedema as described

Question 39 of 129

A 22-year-old Afro-Caribbean man presents with chest pain. He states the pain is sharp in nature, worse on inspiration, and localised to the right lateral chest wall. He has no past medical history and takes no regular medicines.

An ECG is performed:

P waves	Normal morphology
PR interval	140ms
QRS	110ms
QTc	420ms
T waves	Inverted in V1-V6
ST segments	No elevation or depression

What is the most likely explanation of these ECG results?

A. NSTEMI
B. STEMI
C. Normal variant
D. Prinzmetal's angina (variant angina)
E. Stable angina pectoris

C. Normal variant

Widespread T wave inversion in the chest leads can be a normal variant in patients with Afro-Caribbean ethnicity

Coronary artery disease would be very unlikely in a man of this age. The absence of ST elevation makes an acute STEMI even more unlikely. Twave inversion can of course be a feature of an NSTEMI or stable angina, however due to the absence of risk factors, and the patients age, it would be a very unlikely diagnosis.

Prinzmetal's angina due to vasospasm would be a possibility. However this usually presents with ST elevation.

Widespread T-wave inversion in the chest leads can be a normal variant in patients with Afro-Caribbean ethnicity and this would be the most likely diagnosis in a young man with no cardiovascular risk factors.

The patients pain is pleuritic in nature and an alternative diagnosis should therefore be considered (e.g. pneumothorax).

Question 40 of 129

A 62-year-old man presents to the medical take with a persistent fever, fatigue, and a new-onset heart murmur. There is no relevant past medical history of note and no allergies.

The patient is clinically septic. He has a respiratory rate of 22/min, pulse of 112/min, blood pressure of 91/47 mmHg, and a temperature of 38.7ºC. Bedside echocardiography reveals vegetation on the mitral valve. Blood cultures are sent and results are awaited.

What is the most appropriate empirical antibiotic regimen in this case?

A. Amoxicillin +/- gentamicin
B. Benzylpenicillin +/- gentamicin
C. Flucloxacillin
D. Flucloxacillin + rifampicin + gentamicin
E. Vancomycin + gentamicin

A. Amoxicillin +/- gentamicin

IV amoxicillin is the empirical treatment of choice in native valve endocarditis

This patient has presented with infective endocarditis. It is important to note that he has native valves and no organism confirmed on blood cultures as of yet. Therefore, empirical treatment with **amoxicillin +/- gentamicin** should be started.

Benzylpenicillin is indicated in endocarditis confirmed to be of streptococcus origin, with gentamicin to be added depending on sensitivities.

Flucloxacillin is indicated when native valve endocarditis secondary to staphylococci has been confirmed on blood cultures.

Flucloxacillin + rifampicin + gentamicin is indicated in prosthetic valve endocarditis caused by staphylococcus.

Vancomycin + gentamicin is an option for empirical treatment in patients who are penicillin-allergic, however, amoxicillin is preferred where this is not the case.

Question 41 of 129

A 65-year-old man presents to his GP for evaluation. He has no acute complaints. He takes amlodipine and chlorothiazide for hypertension and paracetamol as required for back pain. Physical examination reveals a well-appearing man with a heart rate of 78 beats per minute with a regular rhythm and blood pressure of 162/78 mmHg. His JVP is not visible and you note that he has a sustained non-displaced apical impulse. There are no thrills or audible murmurs and his heart sounds are normal. His chest is clear to auscultation, his abdomen is soft, nontender, and there are no palpable masses or organomegaly, and he has trace pedal oedema. His GP reviews his ECG which was performed earlier that morning.

Which of the following is most likely to be identified on this gentleman's ECG?

A. Third degree heart block with a ventricular escape rhythm
B. Absent P-waves
C. Deep S waves in V1 and V2 and tall R-waves in V5 and V6
D. PR-segment depression and global ST-segment elevation
E. A tall S wave in lead I and Q wave with T wave inversion in lead III

C. Deep S waves in V1 and V2 and tall R-waves in V5 and V6

During a cardiology clinical examination, a sustained apical impulse is consistent with left ventricular hypertrophy, which can be verified on an ECG by identifying deep S waves in V1 and V2 and tall Rwaves in V5 and V6

This gentleman presents to his GP for a routine checkup. He has a medical history of hypertension and is on medication for this, but has evidence of elevated blood pressure in clinic today. Furthermore, on physical examination, he has a sustained non-displaced apical impulse, a finding that typically represents left ventricular hypertrophy, which can arise as a consequence of longstanding hypertension especially when it is not well controlled. Features of left ventricular hypertrophy can be identified on an ECG, and most typically include deep S waves in V1 and V2 and tall R-waves in V5 and V6.

Third-degree heart block with a ventricular escape rhythm typically results in significant bradycardia. This patient has a normal heart rate and so is unlikely to have a complete heart block.

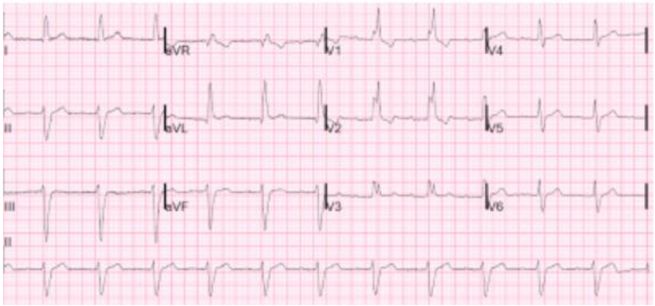
Absent P-waves is a feature of atrial fibrillation which can be detected on a clinical exam as an irregularly irregular pulse. This gentleman has a regular rhythm and is therefore unlikely to have atrial fibrillation.

PR-segment depression and global 'saddle-shaped' ST-segment elevation are findings consistent with acute pericarditis, which typically presents with pleuritic chest pain worse when lying flat, and relieved when sitting forward. This patient has no symptoms at present and so is unlikely to have acute pericarditis.

A tall S wave in lead I and Q wave with T wave inversion in lead III, socalled 'S1 Q3 T3' is a finding associated with an acute pulmonary embolus. This patient is asymptomatic and is unlikely to have a pulmonary embolus.

Question 42 of 129

A 77-year-old man is reviewed in the cardiology. He presented to his GP with intermittent dizziness and reduced exercise tolerance. An ECG accompanies the referral letter:



What does the ECG show?

A. Left bundle branch block
B. Long QT syndrome
C. ECG changes consistent with hypokalaemia
D. Bifascicular block
E. Trifascicular block

E. Trifascicular block

RBBB +left anterior or posterior hemiblock + 1st-degree heart block = trifascicular block

The ECG demonstrates RBBB + left anterior hemiblock + 1st-degree heart i.e. trifascicular block.

Question 43 of 129

A 56-year-old lady with previously diagnosed rheumatic heart disease with mitral stenosis attends for her annual review. She remains fit and well and can walk 100 metres before she develops breathlessness. She gets occasional burning pains in her chest that come on after rich meals and last 20-30 minutes.

She currently takes aspirin, ramipril and a statin. She does not smoke and is taking regular walks each day. On examination, she has a loud S1 heart sound with a mid-diastolic murmur heard in full expiration when in the left lateral position. Her face has a red flush in the cheeks and there is no JVP visible. Her chest is clear and there is no oedema.

Hb	104 g/l	Na +	139 mmol/l
Platelets	450 * 10 /l 9	K +	5.2 mmol/l
WBC	5.6 * 10 /l 9	Urea	4.5 mmol/l
Neuts	4.5 * 10 /l 9	Creatinine	78 µmol/l
Lymphs	0.3 * 10 /l 9	CRP	14 mg/l
Troponin	negative		
ECG	sinus rhythm, left ventricular hypertrophyChest X-ray enlarged cardiac shadow with loss of the aorto-pulmonary window		
ECHO (today)	mitral valve cross sectional area 0.8cm , LV ejection fraction 60%2		
ECHO (1yr ago)	mitral valve cross sectional area 1.1cm , LV ejection fraction 62%2		

What is the most appropriate intervention?

A. Refer to cardiothoracic surgery
B. Furosemide
C. Routine follow up with ECHO in 1 year
D. Repeat ECHO in 6 months
E. Coronary angiogram

A. Refer to cardiothoracic surgery

Mitral stenosis with cross sectional area of <1cm2 warrants surgical intervention

This lady has known mitral stenosis. She has a valve cross sectional area of less than 1cm which is an indication for mitral valve surgery and should be referred immediately. Furosemide would not benefit her as there is no sign of overload. She could continue ECHO surveillance but this will not change her situation. The burning chest pains do not sound cardiac in nature and it would probably be worth trialling omeprazole.

Question 44 of 129

A 64-year-old gentleman is admitted to the hospital's coronary care unit following an anterior ST-elevation myocardial infarction requiring revascularisation. This was successfully done using two drug eluding stents in his left anterior descending artery. Angiography during the procedure did not indicate any further lesions.

He has been pain free since his percutaneous intervention and his current examination is unremarkable. His observations include a blood pressure of 124/88 mmHg, a heart rate of 62/min, a respiratory rate of 18/min and he is apyrexial.

He has now had an echocardiogram which indicated moderate ventricular dysfunction (ejection fraction 40-42%), with no residual valvular disease.

His ECG currently is in sinus rhythm, with normal PR interval, a QRS complex of 100 ms, and normal QTc.

His bloods are as follows:

Hb	142 g/L	Male: (135-180)Female: (115 - 160)
Platelets	325 * 10 /L	(150 - 400)
WBC	8.6 * 10 /L	(4.0 - 11.0)
Na	139 mmol/L	(135 - 145)
K	3.9 mmol/L	(3.5 - 5.0)
Urea	6.2 mmol/L	(2.0 - 7.0)
Creatinine	110 µmol/L	(55 - 120)

He is currently on bisoprolol 5 mg OD, enalapril 2.5 mg BD, aspirin 75 mg OD, ticagrelor 90 mg BD, atorvastatin 80 mg OD and GTN spray PRN.

You are asked by your consultant to optimise his medications/management.

What would be your next step?

A. Add ivabradine
B. Increase the bisoprolol
C. Add isosorbide mononitrate modified release
D. Add eplerenone
E. Change ticagrelor to clopidogrel

D. Add eplerenone

Following myocardial infarction, patients with objective heart failure (moderate to severe left ventricular dysfunction) should be initiated on an ACE inhibitor, a beta-blocker, a statin and a mineralocorticoid receptor antagonist provided no contra-indications exist

Given the findings of his echocardiography, one should consider the possibility of addition of a mineralocorticoid receptor antagonist such as eplerenone.

He is currently not symptomatic at all, therefore there is no place in adding isosorbide mononitrate in this patient.

Additionally, given his heart rate one could wonder whether any increase in bisoprolol would render this patient bradycardic.

There is no indication to change his current dual antiplatelet therapy regime.

Ivabradine is a specific inhibition of the pacemaker current, which is used in the management of symptomatic heart failure and ischaemic heart disease that is not managed via the use of typical agents

Question 45 of 129

A 75-year-old gentleman has recently been diagnosed with heart failure following a myocardial infarction. You review him in clinic. He is currently not complaining of any dyspnoea, orthopnoea or paroxysmal nocturnal dyspnoea and he has no persistent leg oedema. There is no other relevant background medical history.

Examination indicates that he is euvolaemic.

His ECG shows sinus rhythm, Q waves in the inferior leads, a PR interval of 208/msec, normal QRS and no signs of intraventricular conduction delay.

He appears rather anxious and is wondering whether there is anything else that he can do in order to reduce his rate of hospitalisation other than maintaining a healthy lifestyle and taking his medications. He is aware that his brother, who suffers with chronic obstructive pulmonary disease, has a just-in-case box at home and is entitled to vaccinations. He is wondering whether there is any vaccinations or additional antibiotics/medications that could be useful to him.

What would you offer this gentleman?

A. Annual pneumococcus vaccination should be offered
B. Phenoxymethylpenicillin prescription (prophylactic)
C. Annual influenza vaccination
D. Haemophilus influenzae B vaccination
E. Pill-in-the-pocket bumetanide

C. Annual influenza vaccination

As part of the broad lifestyle approach to heart failure, annual influenza vaccine should be offered

Patients with heart failure are encouraged to have the influenza and pneumococcus vaccine, as common respiratory infections are more prevalent in that population and have also the propensity of worsening heart failure and causing decompensation.

The correct answer in this case is annual influenza vaccine; pneumococcus vaccination is usually an once-off regime, provided that the patient is not asplenic or suffering from other chronic diseases.

Haemophilus influenzae B vaccination is advocated in patients with asplenia as they are particularly vulnerable to encapsulated organisms but it has no position in the long-term management of heart failure patients.

Phenoxymethylpenicillin and other antibiotics as prophylaxis are not advocated in heart failure management with regards to reducing infection rates.

There is no strong evidence in supporting a 'pill-in-the-pocket' strategy in patients with heart failure, however patients are encouraged to be aware of the potential manipulation needed when it comes to diuretic therapy and when to seek urgent medical care.

Question 46 of 129

A 31-year-old woman is brought into the Emergency Department by her husband. He tells you that she is abnormally drowsy and he has difficulty rousing her. She has a background of depression which is medicated by her General Practitioner, but is otherwise well.

On examination she is afebrile but has dry, warm skin. Her heart rate is 140bpm with a blood pressure of 105/90mmHg. She has a respiratory rate of 8 breaths per minute and saturating at 92% on air. Her Glasgow coma scale (GCS) is 12 (E3V4M5) and she appears agitated. Her pupils are dilated and she has hyperreflexia. Whilst examining her abdomen it is noted that she has a palpable bladder and bowel sounds are absent.

Investigations: Arterial blood gas (ABG):

pH	7.29	(7.35-7.45)
PCO2	7.2 kPa	(4.9-6.1 kPa)
PO2	10.1 kPa	(10-13.1 kPa)
Bicarbonate (HCO3-)	28 mmol/L	(22-28 mmol/L)
Base Excess (BE)	3.1 mmol/L	(-2 to 2 mmol/L)
Sodium	134 mmol/L	(135-145 mmol/L)
Potassium	4.4 mmol/L	(3.5-5.5 mmol/L)
Chloride	105 mmol/L	(95-110 mmol/L)
Lactate	2.1 mmol/L	(0.2-1.6 mmol/L)
Glucose	5.3 mmol/L	(4-7 mmol/L)

ECG - Polymorphic ventricular tachycardia, QTc 500ms.

What is the most important next step in the management of this patient?

A. Synchronised DC cardioversion
B. Intravenous amiodarone
C. Intravenous magnesium sulphate
D. Intravenous lidocaine
E. Intravenous sodium bicarbonate

C. Intravenous magnesium sulphate

IV magnesium sulfate is used to treat torsades de pointes

This woman has had a tricyclic antidepressant (TCA) overdose. In excess, TCAs can cause QT interval prolongation which can predispose to ventricular arrhythmias. A polymorphic ventricular arrhythmia with a prolonged QT interval is characteristic of torsades de pointes. The treatment for this is intravenous magnesium sulphate.

As she currently has a stable blood pressure (systolic > 90mmHg), DC cardioversion would not be appropriate at this stage. Intravenous amiodarone and lidocaine can be used to medically cardiovert a monomorphic ventricular tachycardia, but are not the treatment of choice in torsades de pointes.

Sodium bicarbonate is the first-line treatment of TCA overdose. However, in the presence of torsades de pointes, intravenous magnesium sulphate is the more appropriate choice to give initially.

Question 47 of 129

A 72-year-old man attends the emergency department with mild epistaxis. He denies any other bleeding. He has a past medical history of atrial fibrillation and severe mitral stenosis for which he takes warfarin. Observations are as follows: heart rate 80 beats per minute, blood pressure 145/85 mmHg, respiratory rate 14 breaths per minute, SpO2 96% (on air), and temperature 37.5ºC.

Blood results are as follows:

Hb	144 g/L	Male: (135-180)Female: (115 - 160)
Platelets	72 * 10 /L 9	(150 - 400)
WBC	8.2 * 10 /L 9	(4.0 - 11.0)
(PT)	114 secs	(10-14 secs)
INR	10.2	(<1.1)
(APTT)	54 secs	(25-35 secs)
Fibrinogen	3.4 g/L	(2 - 4)

You withhold the patient's warfarin.

What further measures are required?

A. Fresh frozen plasma
B. Intravenous 10mg vitamin K
C. Intravenous 3mg vitamin K
D. Platelet transfusion
E. Prothrombin complex concentrate

C. Intravenous 3mg vitamin K

INR > 8.0 (minor bleeding) - stop warfarin, give intravenous vitamin K 1-3mg, repeat dose of vitamin K if INR high after 24 hours, restart when INR < 5.0

The results of the coagulation screen suggest warfarin toxicity. The INR (derived from the PT) is used to monitor warfarin since it is the most representative, however, it is important to remember that warfarin can also prolong the APTT.

Intravenous 3mg vitamin K is correct. The patient has a minor bleed with an INR >8.0. Thus the warfarin should be withheld and the patient should receive intravenous vitamin K 1-3mg. The warfarin should be restarted when the INR < 5.0.

Fresh frozen plasma is incorrect. Fresh frozen plasma (FFP) is not recommended for use as a warfarin reversal agent as it can take hours to work due to the volume required. In warfarin-associated major haemorrhages, FFP should only be given if prothrombin complex concentrate is unavailable.

Intravenous 10mg vitamin K is incorrect. For minor haemorrhage, the dose of intravenous vitamin K recommended is 1-3mg.

Platelet transfusion is incorrect. In the context of haemorrhage, a platelet level of <50 is the standard transfusion trigger, with the exclusion of CNS or posterior eye bleeds in which the transfusion trigger is <100. The patient in this clinical case has a platelet count of 72 and thus does not require a platelet transfusion.

Prothrombin complex concentrate is incorrect. Prothrombin complex concentrate (PCC) is recommended only for major bleeding. PCC will reverse anticoagulation within minutes of administration however administration of vitamin K is also required to counteract the long halflife of warfarin.

Question 48 of 129

A 50-year-old female is admitted on the acute medical take with palpitations. She recalls on-and-off palpitations for over one year, however today they have been ongoing. On examination, she is haemodynamically stable with an irregularly irregular pulse of 140 b.p.m. There is no clinical evidence of heart failure. Her ECG shows atrial fibrillation.

Which of the following medical conditions is a contraindication to initiating beta-blocker therapy in this patient?

A. Intercurrent treatment with dihydropyridine calcium-channel
B. blockers
C. Phaeochromocytoma on phenoxybenzamine
D. Sick sinus syndrome with a permanent pacemaker
E. Second-degree heart block
F. Mild peripheral vascular disease

E. Second-degree heart block

Given the ubiquity of beta-blockers in medical practice it is important to know contra-indications for their use.

Although the most commonly cited contraindication to beta-blocker therapy is asthma / COPD, there is increasing evidence that in the latter cohort, cardioselective beta-blockers (atenolol, bisoprolol, metoprolol) are well-tolerated with a favourable safety profile.

Beta-blockers slow conduction at the AV node and therefore should be avoided in AV nodal disease as they can lead to complete heart block. Thus they should be avoided in second-degree heart block.

Beta-blockers also inhibit beta1-adrenoceptor activity at the sinoatrial node (i.e. are negative chronotropes) so are contraindicated in sick sinus syndrome. However, in the presence of a permanent pacemaker (PPM), beta-blocker use is safe.

Due to the synergistic effect of beta- and calcium-channel blockade on myocardial excitability, non-dihydropyridine calcium channel blockers (diltiazem and verapamil) should not be co-administered with beta blockers.

An untreated phaeochromocytoma causes increased alpha- and betaadrenergic activity. Thus the administration of a beta-blocker alone will lead to unopposed alpha-agonism which can lead to vasoconstriction and refractory hypertension. However, if a patient is taking an alphablocking agent (e.g. phenoxybenzamine), then beta-blockers may also be initiated.

Question 49 of 129

A 57-year-old man with a 2 year history of hypertension is referred for further evaluation due to his blood pressure being difficult to control. The following results were obtained prior to commencing medications:

Na +	148 mmol/l
K +	3.2 mmol/l
Creatinine	130 µmol/l
Renin	102 (7-50 IU/mL ambulatory)
Aldosterone	1105 (N: 80-800 ng/dL ambulatory)
Renin:Aldosterone Ratio	10.8 (< 500)
Plasma Metanephrines	0.35 (<0.50 nmol/L)

These results are most consistent with which of the following:

A. Congenital Adrenal Hyperplasia
B. Phaeochromocytoma
C. Cushing's disease
D. Renovascular disease
E. Primary hyperaldosteronism

D. Renovascular disease

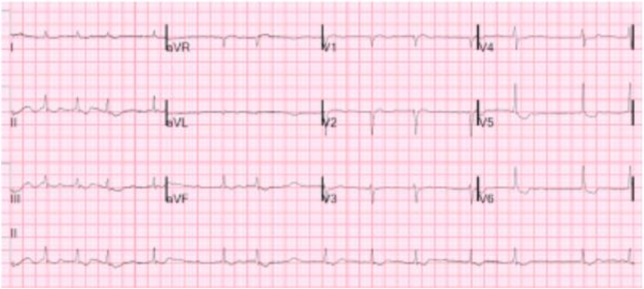
The key underlying diagnosis is renal artery stenosis. The key is that the renin-aldosterone ratio is normal. If it was Conn's disease, or primary hyperaldosteronism the renin would be suppressed. The plasma metanephrines indicate that no pheochromocytoma is present as the sensitivity of plasma metanephrines for a pheochromocytoma approaches 100%.

Question 50 of 129

A 77-year-old woman is seen in a heart failure clinic. Her past medical history includes congestive cardiac failure, atrial fibrillation, hypertension, and depression. Her regular medications include bisoprolol, digoxin, amlodipine, furosemide, and citalopram.

Repeat digoxin levels are displayed below:

Digoxin level	1.1 ng/mL	(0.7-2.0)



Which agent is responsible for the abnormalities displayed in her ECG?

A. Amlodipine
B. Bisoprolol
C. Citalopram
D. Digoxin
E. Furosemide

D. Digoxin

This woman's ECG displays 'scooped' ST depression in leads II, III, aVF, V5, and V6 in the context of atrial fibrillation. This is a classical sign seen in the digoxin effect, the ECG changes that arise with digoxin within normal therapeutic levels. It is important to note that in digoxin toxicity, the most common dysrhythmias are supraventricular tachycardia with a slow ventricular response due to blocking of the AV node.

Whilst calcium channel blockers can significantly disrupt the ECG, this is associated primarily with non-dihydropyridines such as verapamil or diltiazem, rather than dihydropyridines such as amlodipine which are less cardioselective.

Bisoprolol can give rise to bradycardia and PR prolongation on ECG but the ST changes observed here are consistent with the digoxin effect. Therefore, bisoprolol is incorrect.

Citalopram is a selective serotonin reuptake inhibitor and therefore can result in QT prolongation. The ST changes as described here are not consistent with this syndrome and therefore this is an incorrect answer.

Furosemide is a loop diuretic that can result in hypokalemia and therefore flattened and inverted T waves. The ST segment in this ECG shows the classical 'scooped' appearance and therefore is more consistent with the digoxin effect rather than hypokalemia. Furosemide is therefore incorrect

Question 51 of 129

An 82-year-old man sees his GP to discuss his medication. He complains of shortness of breath on minimal exertion and says that he now has to sleep upright in his armchair. His medication includes lisinopril, metoprolol, spironolactone, furosemide, aspirin, simvastatin, nifedipine and metformin.

Physical examination shows a heart rate of 69 beats per minute with a regular rhythm and blood pressure of 118/100 mmHg. His peripheries are cool, and his carotid pulse is difficult to palpate. He has a sustained apical impulse palpable in the 5th intercostal space in the mid-clavicular line. He has a palpable thrill in the second intercostal space at the right sternal border where his GP hears a loud crescendo-decrescendo murmur which radiates into his neck. He has a quiet S1, an inaudible S2, and a loud S4. His chest is clear to auscultation and he is noted to have 1+ peripheral oedema.

Which of the following physical examination findings are most suggestive of severe aortic stenosis?

A. Loud crescendo-decrescendo murmur
B. Quiet S1
C. Inaudible S2
D. Palpable thrill
E. Narrow pulse pressure

C. Inaudible S2

Aortic stenosis - a soft S2 is a feature of severe disease

The feature identified on physical examination that is most suggestive of severe aortic stenosis is an inaudible second heart sound.

The loudness of a murmur does not correlate with its severity. In the case of regurgitant murmurs, for example, quiet murmurs can represent severe regurgitation as there is early equalisation between the two cardiac chambers involved.

A quiet S1 may reflect significant aortic valve calcification but does not necessarily reflect the severity of the stenosis

A palpable thrill and narrow pulse pressure can occur in severe aortic stenosis but are not as indicative of severe aortic stenosis as an absent second heart sound

Question 52 of 129

A 71-year-old patient presents to the Emergency Department with a two hour history of crushing central chest pain. He is known to have a history of ischaemic heart disease. The ECG shows the following:

ST elevation greater in lead II than in lead III with abnormal Q waves in II, III, and aVF

ST depression, tall, broad R waves and upright T waves in V1-3. Dominant R wave in V2

ST elevation in V5-V6

Where is the lesion most likely to be?

A. Left anterior descending
B. Left circumflex
C. Right coronary artery
D. Left main stem
E. Posterior interventricular

B. Left circumflex

Ischaemic changes in leads I, aVL +/- V5-6 - left circumflex

These are classical findings of a circumflex occlusion. The table below shows how the changes correspond to the cardiac anatomy:

ECG changes	Component of infarction
ST elevation greater in lead II than in lead III with abnormal Q waves in II, III, and aVF	Inferior component of infarction
ST depression, tall, broad R waves and upright T waves in V1-3. Dominant R wave in V2	Posterior component of infarction
ST elevation in V5-V6	Lateral component of infarction

Question 53 of 129

A 24-year-old male presents to the emergency department with a collapse. The previous night he had been out drinking heavily with his friends in a nightclub. A friend attends with him and states that some of the lads had taken MCAT (Mephedrone) but he is unsure whether Michael also took MCAT. Michael felt well when he woke up but realised that he was late for his football match so rushed out of the house without any breakfast. He had just scored the first goal of the match when he collapsed. When he came around he complained of palpitations.

His past medical history includes asthma and hay fever for which he takes regular antihistamines. During the ambulance ride to hospital the paramedic performed some observations and an ECG. He was found to have a blood glucose of 3.6 and was given some GlucoGel and a biscuit. Other observations included a blood pressure of 90/60 mmHg, heart rate of 135/min which was irregular, irregular in character, respiratory rate of 16/min, afebrile.

The emergency department doctor reviewed the ECG and diagnosed atrial fibrillation with a fast ventricular response and prescribed digoxin in view of his asthma history and blood pressure readings. The patient had a normal QTc. Five minutes later the cardiac monitor showed a broad complex tachycardia and Michael became unresponsive.

What was the cause of the patient's collapse?

A. Hypertrophic cardiomyopathy (HCM)
B. MCAT related arrhythmia
C. Wolff-Parkinson-White syndrome
D. Alcohol induced atrial fibrillation
E. Torsades de pointes

C. Wolff-Parkinson-White syndrome

Wolff-Parkinson-White syndrome (WPW) is a disorder of the conduction system also known as a pre-excitation syndrome. The incidence of WPW is between 0.1% and 0.3% in the general population. Sudden cardiac death in people with WPW is rare (incidence of less than 0.6%).

In WPW there is an abnormal accessory pathway called the 'Bundle of Kent' found between the atria and ventricles. If this pathway is used signals may stimulate the ventricles to contract prematurely, resulting in atrioventricular re-entrant tachycardia.

Normally the AV node acts as a gatekeeper limiting the amount of electrical activity that can reach the ventricles. If a patient has an arrhythmia whereby the atria generate an excessively fast rhythm such as atrial flutter or fibrillation the AV node is able to reduce the amount of electrical activity that reaches the ventricles. However, the Bundle of Kent does not possess the ability to limit the electrical signals to the ventricles. If the person has an atrial arrhythmia the signals can pass down the bundle and cause the ventricles to contract at the same rate. In the extreme heart rates and when the accessory pathway is used the patient may develop dangerous cardiac arrhythmias which can trigger ventricular fibrillation and lead to death.

In WPW certain medications that can block the AV node or those that can enhance conduction down the accessory pathway by increasing the refractory period in the AV node should be avoided. These include digoxin, adenosine, diltiazem, verapamil, other calcium channel blockers and beta blockers. In this case, the patient's WPW was worsened by administration of digoxin and her arrhythmia developed into ventricular fibrillation.

People with WPW are usually asymptomatic. Symptomatic patients may complain of shortness of breath, palpitations, dizziness and syncope. Diagnosis is usually made by ECG as patients may have the characteristic 'delta wave'. Patients may also be found to be in atrial fibrillation or have SVT Treatment can include cardioversion in emergencies otherwise amiodarone. Definitive treatment would be ablation.

Question 54 of 129

A 65-year-old gentleman is reviewed in cardiology clinic with known cardiomyopathy. He has started to develop symptoms of progressive exertional shortness of breath and is concerned about having this treated. He has not had any other symptoms and on examination, there is no peripheral oedema or chest signs. A recent echocardiogram demonstrates a provoked left ventricular outflow gradient of 64mmHg.

What would be the most appropriate medical therapy?

A. Phosphodiesterase type 5 inhibitor
B. Beta-blocker
C. Nitrate
D. Digoxin
E. Ace-inhibitor

B. Beta-blocker

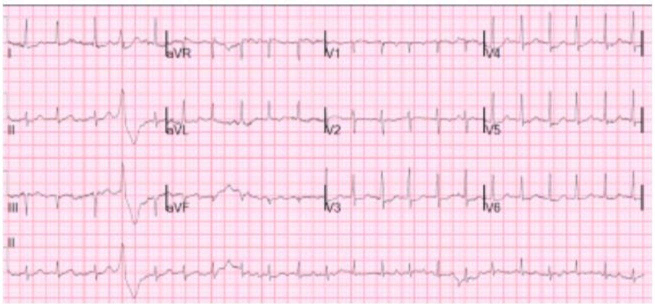
The correct answer is beta-blocker. Beta-blockers and verapamil can help with symptom management as they control the heart rate to the point where ventricular outflow obstruction is unlikely to occur. Nitrates and ace-inhibitors are contra-indicated as they lower blood pressure which can be very dangerous when combined with hypotension when the outflow is obstructed, whilst phosphodiesterase type 5 inhibitors and digoxin would offer no benefit. Phosphodiesterase type 5 inhibitors have a greater role in pulmonary hypertension whilst digoxin can be used to control the heart rate in AF when the patient is sedentary or there is coexisting heart failure.

Question 55 of 129

A 54-year-old man presents with a sensation of his heart racing. He has noticed this intermittently over the last several weeks. On examination, his heart sounds are normal with no added sounds. He has a heart rate of 90 beats per minute and a blood pressure of 124/66 mmHg.

His past medical history includes mild asthma only. He is otherwise fit and well and regularly cycles 30 - 40 miles at the weekend. He previously had electrical cardioversion 2 years ago which restored normal sinus rhythm at the time.

His ECG is 0shown below



As well as anticoagulation, what additional medication could be considered before electrical cardioversion?

A. Amiodarone
B. Aspirin
C. Digoxin
D. Flecainide
E. Ramipril

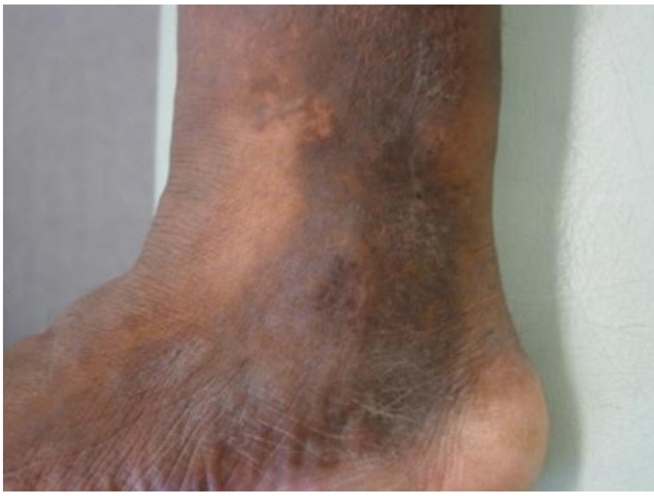
Not answered in the file

Question 56 of 129

A 69-year-old man visits his General Practitioner complaining of discolouration to his lower legs. He denies itch but complains of intermittent pain that is progressively worsening. He does not visit his doctor regularly and has not had a check-up in several years. He takes no regular medications and chooses to take herbal supplements daily.

Ankle brachial pressure index	1.1

His legs are examined, as shown below:



What is the recommended management for this patient?

A. Compression stockings
B. Low dose aspirin and statin
C. Oral flucloxacillin
D. Topical corticosteroids
E. Topical emollients

A. Compression stockings

This patient has evidence of discolouration to the lower leg, in keeping with haemosiderin deposition as seen in chronic venous insufficiency. It is likely that this patient has an underlying diagnosis of venous insufficiency and varicose veins. Risk factors for varicose veins include female sex, obesity, pelvic congestion (e.g. in pregnancy) which increases venous pressure in the lower limbs and increasing age. Other changes that can be seen in patients with varicose veins include lipodermatosclerosis (hardening of the skin of the lower limb) and varicose eczema. The management of choice consists of conservative measures in the first instance including exercise, weight loss and **compression stockings**.

Low dose aspirin and statin is incorrect. Although there is a history of pain, the normal ankle brachial pressure index is reassuring that this patient's pain is not secondary to arterial ischaemia. Therefore, aspirin and statin therapy is not indicated.

Oral flucloxacillin is incorrect. This is used in the management of cellulitis which typically appears as an erythematous and inflamed area of skin that is warm and tender to the touch. This is not in keeping with this patient's presentation.

Topical corticosteroids is incorrect. It is used in the management of some inflammatory skin disorders such as eczema, urticaria and psoriasis. However, it plays no role in the management of varicose veins.

Topical emollients is incorrect. This is used in the treatment of dermatitis which is visualised as dry and exfoliative skin that may show signs of excoriations and hyperpigmentation. This is not seen here.

Question 57 of 129

A 79-year-old woman is reviewed in cardiology clinic as part of the ongoing assessment for possible intervention for severe aortic stenosis. The patient had been diagnosed with severe aortic stenosis 4 months previously, after experiencing progressive exertional breathlessness and reduced exercise tolerance. At her initial review at cardiology clinic, the patient had expressed interest in undergoing intervention for aortic stenosis: either surgical aortic valve replacement (SAVR) or transcatheter aortic valve insertion (TAVI). Subsequently, the patient had undergone a variety of investigations to assess her suitability for the above procedures.

The patient had a good functional status, leaving independently with her daughter and still participating in a wide range of community activities. The patient had a long-standing diagnosis of hypertension and had suffered a left cortical stroke 3 years previously. In addition, the patient had chronic obstructive pulmonary disease, although she had stopped smoking 30 years previously. The patient's regular medications were amlodipine, ramipril, clopidogrel, simvastatin and an ipratropium inhaler. The patient used a salbutamol inhaler as required. The patient had no known drug allergies.

A summary of the investigations undergone by the patient is given below.

Haemoglobin	128 g / dL
Mean cell volume	87.1 fl
White cell count	6.3 x 10>3 / microlitre
Platelets	324 x 10>3 / microlitre
Urea	5.3 mmol / L
Creatinine	124 micromol / L
Sodium	136 mmol / L
Potassium	4.6 mmol / L

Investigation	Result
Transthoracic echocardiogram	Severe aortic stenosis (valve area 0.85 cm2); no other valve disease; normal systolic function; no anatomic contraindications to TAVI
Coronary angiography	No evidence of coronary artery disease
Iliofemoral angiography	Severe calcification and tortuosity of iliac arteries; unsuitable for transfemoral TAVI
Pulmonary function tests	Moderate obstructive lung disease

Following the above assessment, surgical aortic valve replacement was estimated to carry a 4.1 % risk of mortality and 3.7 % risk of permanent stroke (intermediate risk). No contraindications to transapical transcatheter aortic valve insertion were identified.

What is the recommended choice of intervention for the patient's aortic stenosis?

A. Surgical aortic valve replacement and transcatheter aortic valve
B. insertion inappropriate
C. Surgical aortic valve replacement with mechanical valve
D. Transapical transcatheter aortic valve insertion
E. Surgical aortic valve replacement with bioprosthetic valve
F. Transfemoral transcatheter aortic valve insertion

E. Surgical aortic valve replacement with bioprosthetic valve

The patient has severe, symptomatic aortic stenosis and without intervention is likely to have a poor prognosis. Given the patient's age, if she was to undergo surgical aortic valve replacement (SAVR) then a bioprosthetic valve would be preferred over a mechanical valve, freeing the patient from the need for lifelong anticoagulation. Typical risk stratification for SAVR defines the estimated risk of peri-operative death as low (< 4 %), intermediate (4-8 %) and high (> 8 %). Due to her comorbidities, the patient has an intermediate risk associated with SAVR, calculated using the STS risk calculator (http://riskcalc.sts.org/stswebriskcalc/#/).

Transcatheter aortic valve insertion (TAVI) was initially developed for patients considered unfit for SAVR due to high predicted mortality. However, a recent randomised controlled trial comparing outcomes for TAVI versus SAVR as an intervention for severe, symptomatic aortic stenosis has demonstrated that transfemoral TAVI can be a suitable intervention for some patients with a low or intermediate risk associated with SAVR. Transfemoral TAVI is increasingly favoured over SAVR as patient age increases.

In contrast, outcomes for transapical TAVI are inferior to SAVR, so this intervention is only appropriate for individuals with an unacceptably high surgical risk. For patients where both SAVR and transapical TAVI are possible, SAVR is strongly favoured at all patient ages.

Therefore, for this patient, bioprosthetic SAVR is the intervention of choice, favoured over transapical TAVI. Transfemoral TAVI is technically impossible in this patient due to the anatomy of her iliac arteries.

Question 58 of 129

A patient attends a cardiology clinic following a recent myocardial infarct (MI) which was successfully managed via percutaneous coronary intervention (PCI). The patient has been taking their secondary prevention medications regularly and reports no issues or symptoms.

Their examination is unremarkable and the patient's observations are as follows:

Heart rate 55 beats per minute Blood pressure 134/80 mmHg Respiratory rate 16 breaths per minute Oxygen saturations 98% on air Temperature 36.2 ºC

An ECG performed has widened QRS complexes and atrioventricular dissociation in keeping with accelerated idioventricular rhythm (AIVR) with a rate of 55 beats per minute.

What management should be advised?

A. Arrange for urgent repeat PCI
B. Arrange for urgent pacemaker insertion
C. No additional management is required
D. Stop the patient's beta blocker and continue all other medications
E. Urgent atropine

C. No additional management is required

Accelerated idioventricular rhythm is common and unconcerning following recent MI

AIVR is a benign ectopic rhythm of ventricular origin. It is common postMI following the reperfusion of an ischaemic myocardium. AIVR is usually self-limiting and therefore treatment is not necessary, especially as the patient is asymptomatic and haemodynamically stable with a normal heart rate and blood pressure.

Arranging for urgent repeat PCI is unnecessary as there is no indication of new/further ischaemic damage or an ischaemic event having occurred. AIVR is due to an ectopic rhythm and therefore PCI is of no value.

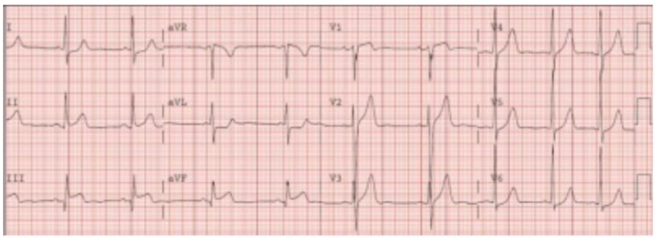
Pacemaker insertion is not required as AIVR is due to a benign ectopic rhythm and therefore pacemaker insertion would not remove the cause. As the patient is asymptomatic and haemodynamically stable no treatment is required.

Stopping the patient's beta-blocker is unnecessary as this will not eliminate the AIVR. Beta-blockers are an important part of secondary prevention post-MI and therefore should be continued.

Atropine could be considered to overcome AIVR via increase the sinus rate however this is rarely required. As the patient is symptom-free and haemodynamically stable the use of atropine is unnecessary.

Question 59 of 129

A middle-aged man is admitted to the resuscitation room of the Emergency Department with crushing central chest pain. His ECG is shown below:



Where is the lesion most likely to be?

A. Left circumflex
B. Right coronary
C. Left anterior descending
D. Posterior interventricular
E. Left main stem

B. Right coronary

Ischaemic changes in leads II, III, aVF - right coronary

There is inferior ST elevation (II, III and aVF) suggestive of a right coronary lesion. Some reciprocal ST depression in I and aVL is also seen.

Question 60 of 129

A 14-year-old female presented to her GP having collapsed at school. Her friends stated that whilst she was playing hockey she inexplicably collapsed and had lost consciousness for a few seconds. This was her first episode of collapse. No seizure activity was observed, and she made a full and spontaneous recovery a few moments later. She denied the presence of prodromal symptoms, and other than occasional palpitations upon exertion she was otherwise well. She had a cochlear implant inserted in early childhood for congenital hearing impairment but otherwise had no past medical history of note, and was not taking any medications. There was no family history of note.

Examination revealed the presence of a healthy athletic 14-year-old female. Her heart rate was 58bpm and regular, and her blood pressure 108/78 mmHg. Cardiovascular examination revealed a JVP of 3cm and was otherwise unremarkable with normal heart sounds. Gastrointestinal examination and neurological examinations were likewise unremarkable.

Initial investigations revealed the following results:

ECG: 57bpm normal, sinus rhythm QRS 112 ms, PR 122ms, QTc 502ms, normal ST and T wave morphology.

What is the most likely diagnosis?

A. Brugada syndrome
B. Romano Ward syndrome
C. Wolff Parkinson White syndrome
D. Jervell & Lange-Nielsen syndrome
E. Hypertrophic obstructive cardiomyopathy

D. Jervell & Lange-Nielsen syndrome

Inherited long QT syndrome, sensorineural deafness - Jervell and Lange-Nielsen syndrome

This female has long QT syndrome (LQTS), as per the findings of her ECG in combination with a history of collapse on exertion. The main differential diagnosis here is Romano-Ward syndrome, which is also a LQTS but is not associated with deafness, as opposed to Jervell & LangeNielsen syndrome, which is associated with deafness. A normal cardiac examination with no evidence of hypertrophy on ECG would make the diagnosis of hypertrophic obstructive cardiomyopathy less likely.

Question 61 of 129

A 59-year-old woman presents to the emergency department with shortness of breath. She has been progressively getting worse over two weeks and has now started to feel short of breath on rest. She is known to have mitral valve prolapse and is awaiting surgery but has so far not been given a date. She also has polycystic ovarian syndrome, type 2 diabetes and depression. She takes metformin, sertraline and furosemide.

On examination, she looks unwell. She has bilateral crepitations with no wheeze on auscultation of her chest, a raised JVP, and a systolic murmur. A chest X-ray shows pulmonary oedema. She is treated with IV diuretics but remains breathless and hypoxic.

What kind of ventilatory support would be most appropriate?

A. No ventilator support is appropriate
B. Negative pressure ventilation
C. Intubation
D. Bilevel positive airway pressure (BIPAP)
E. Continuous positive airway pressure (CPAP)

E. Continuous positive airway pressure (CPAP)

Acute heart failure not responding to treatment - consider CPAP

The correct answer is continuous positive airway pressure (CPAP). This is a patient with a known mitral valve prolapse presenting with decompensation. It can be assumed that the lesion is significant as she is due to have surgery to repair it. She has developed signs of pulmonary oedema and she is not responding to treatment with diuretics. Vasodilators and opiates could be a plan of further medical management, but CPAP could help reduce pulmonary oedema. BIPAP would be appropriate in ventilatory failure in COPD, whilst negative pressure ventilation is more of historical treatment for polio.

Mitral valve prolapse has a prevalence of 2-3% in the population and the majority of cases will be asymptomatic. Risk factors for progression the severe disease include:

- Comorbidities: atrial fibrillation, left sided cardiac failure, hypertension and obesity.
- Echocardiogram findings: mitral leaflet thickness >5mm, prolapse of the posterior leaflet, moderate or severe regurgitation.
- Stress echocardiogram findings: regurgitation during exercise but not at rest.

Question 62 of 129

Whilst covering medical receiving overnight, you are referred a 52-yearold man presenting to the Emergency Department with palpitations. He describes 'fluttering in the chest' for the last 48 hours. His past medical history is remarkable only for type 1 diabetes, adequately controlled with insulin. His admission ECG shows atrial fibrillation with a ventricular rate of 130-140 beats per minute. Examination reveals; blood pressure 88/60 mmHg, with capillary refill 3 seconds. His chest is clear to auscultation. He scores 15/15 on the Glasgow coma score. The patient feels light headed but is otherwise uncomplaining.

What is the most appropriate next step in management?

A. Monitor and observe the patient on the Coronary Care Unit
B. Load with 300mg, intravenous amiodarone
C. Attempt vagal manoeuvres
D. Give 5mg intravenous metoprolol as a slow bolus
E. Arrange DC cardioversion

E. Arrange DC cardioversion

In the context of a tachyarrhythmia, a systolic BP < 90 mmHg → DC cardioversion

The Resuscitation Council (UK) gives clear advice on the management of tachyarrhythmias (see link below for the relevant algorithm). Essentially patients with tachyarrhythmias should be categorised first by the presence or absence of adverse features, these are:

1) Shock - Systolic blood pressure of less than 90mmHg, cold extremities, prolonged capillary refill time etc.

2) Syncope

3) Heart failure pulmonary oedema plus or minus raised jugular venous pressure

Question 63 of 129

A 45-year-old man presents to the Emergency Department with shortness of breath, heavy chest pain and syncope. The chest pain and shortness of breath have been worsening over the last week. He is currently undergoing adjunct chemotherapy for a non-resectable soft tissue sarcoma. He has known metastasis in his thorax and mediastinum.

Hb	90 g/L	Male: (135-180)Female: (115 - 160)
Platelets	100 * 10 /L	(150 - 400)
WBC	12.3 * 10 /L	(4.0 - 11.0)
Na	131 mmol/L	(135 - 145)
K	3.1 mmol/L	(3.5 - 5.0)
Bicarbonate	19 mmol/L	(22 - 29)
Urea	8.0 mmol/L	(2.0 - 7.0)
Creatinine	145 µmol/L	(55 - 120)

On physical examination his JVP is raised at 6cm, there are no precordial thrills, and he has quiet S1 and S2.

Observations show:

- Heart rate of 115/min and regular
- Blood pressure 95/55mmHg
- Respiratory rate 28/min
- Temperature 36.5ºC
- AVPU - A

What is the more specific ECG finding associated with this diagnosis

A. Electrical alternans
B. RBBB
C. S1Q3T3
D. ST elevation in leads V1-4
E. Sinus tachycardia

A. Electrical alternans

Electrical alternans is suggestive of cardiac tamponade

Electrical alternans is the correct answer. The patient has metastatic disease within the thorax and mediastinum increasing his risk of a pericardial effusion. Electrical alternans is seen in massive pericardial effusions as an alternating height of QRS complex across the rhythm strip. Other ECG findings to look for are tachycardia and low voltage QRS. The website Life in the Fast Lane has a library of ECGs with examples of this.

RBBB is the incorrect answer because this is not a specific finding for massive pericardial effusion. There are a variety of causes of RBBB. Examples include congenital heart disease, cardiomyopathy, pulmonary embolus, right ventricular hypertrophy, ischaemic heart disease and others. In an acute PE setting, which is a differential, in this case, you would also look for right heart strain due to the sudden increase in right ventricular pressure caused by the obstruction to the pulmonary vasculature.

S1Q3T3 is the incorrect answer as this is associated with a pulmonary embolism (PE). However, this is only seen in approximately 15% of PEs. The more common ECG finding is sinus tachycardia.

ST elevation in leads V1-4 is the incorrect answer because this is seen in an anterior occlusive, or type 1, myocardial infarction. This typically affects the left anterior descending artery and confers a high risk of a ventricular arrhythmia. The patient requires urgent PCI or, if that is not available within 120 minutes, then thrombolysis.

Sinus tachycardia is the incorrect answer as it is a non-specific finding on ECG. You would expect a patient with a massive pericardial effusion to mount a tachycardic response. This is because a decrease in diastolic relaxation and the subsequent volume of ventricular filling decreases your stroke volume. To maintain an adequate cardiac output, the patient will increase HR. As CO = SV x HR. However, this is a non-specific finding and is seen in all types of shock; septic, cardiogenic, distributive and hypovolaemic. Answer 1 is a much more specific ECG finding.

Question 64 of 129

A 24-year-old man is brought in by ambulance to the emergency department after a witnessed collapse. He was unconscious for 10-15 seconds before quickly recovering. There were no preceding symptoms and no witnessed seizure activity. He has no past medical history and is not on any regular medications.

Observations:

Heart rate 82 beats per minute Blood pressure 120/80 mmHg Respiratory rate 16/minute Oxygen saturations 96% on room air Temperature 37C

The examination is unremarkable and his Glasgow coma scale is 15/15.

An ECG demonstrates possible coved ST elevation in leads V1-V3 without reciprocal changes.

Troponin	6 ng/L	(< 14)

Given the likely diagnosis, the administration of which anti-arrhythmic will make the ECG changes more apparent?

A. Dobutamine
B. Flecainide
C. Isoprenaline
D. Metoprolol
E. Quinidine

B. Flecainide

ECG changes in Brugada syndrome are more apparent following the administration of flecainide or ajmaline - this is the investigation of choice in suspected cases

Flecainide is the correct answer. The patient presents with syncope and ECG changes that raise the possibility of Brugada syndrome (coved ST elevation in leads V1-V3). Administration of flecainide can make these ECG changes more apparent and aid diagnosis.

Dobutamine is incorrect. This medication can be used in stress echocardiography to determine if there is any inducible ischaemia but is not used to make the ECG changes of Brugada syndrome more apparent.

Metoprolol is incorrect. Typically, a short-acting beta-blocker is administered prior to CT coronary angiography with the aim of reducing the heart rate to a level of approximately 60 to facilitate interpretation of this form of imaging. It is not used to unmask the ECG changes of Brugada syndrome.

Quinidine is incorrect. This has been suggested in some reports to be useful as an anti-arrhythmic in Brugada syndrome. It does not make the ECG changes more prominent.

Isoprenaline is incorrect. This has been shown to normalise the electrocardiographic pattern in Brugada syndrome.

Question 65 of 129

A 25-year-old male is brought to the emergency department with an episode of syncope, which occurred without warning. He has no past medical history and does not take any regular medications. There is no history of substance misuse.

Observations:

- Heart rate 88 beats per minute
- Blood pressure 120/88 mmHg
- Respiratory rate 18/minute
- Oxygen saturations 96% on room air
- Temperature 37.1C

The examination is unremarkable.

An ECG demonstrates T wave inversion in leads V1-V3. Blood tests:

Hb	136 g/L	Male: (135-180)Female: (115 - 160)
Platelets	189 * 10 /L	(150 - 400)
WBC	4.8 * 10 /L	(4.0 - 11.0)
Na	141 mmol/L	(135 - 145)
K	4.2 mmol/L	(3.5 - 5.0)
Urea	5.1 mmol/L	(2.0 - 7.0)
Creatinine	88 µmol/L	(55 - 120)
CRP	4 mg/L	(< 5)
Troponin	4 ng/L	(<14)

An echocardiogram demonstrates a dilated, hypokinetic right ventricle. A CT pulmonary angiogram excludes a pulmonary embolism.

Given the likely diagnosis, what is the most specific ECG finding suggestive of this condition?

A. Coved ST segment elevation in V1-V2
B. Delta wave
C. Epsilon wave
D. PR depression
E. Low voltage QRS and electrical alternans

C. Epsilon wave

The most characteristic ECG finding in arrhythmogenic right ventricular dysplasia (ARVD) is the epsilon wave (a small positive deflection at the end of the QRS complex)

Epsilon wave is correct. The patient presents with syncope without warning, suggesting a cardiogenic cause. T wave inversion in leads V1-V3 and an echocardiogram demonstrating a dilated, hypokinetic right ventricle is characteristic of arrhythmogenic right ventricular dysplasia (ARVD). The most specific finding for this condition is an epsilon wave, which is a small positive deflection at the end of the QRS complex).

Delta wave is incorrect. This is a slurred upstroke of the QRS complex and suggests Wolff-Parkinson-White (WPW) syndrome.

Coved ST segment elevation in V1-V2 is incorrect. This is characteristic of Brugada syndrome, which is another cause of sudden cardiac death.

PR depression is incorrect. The ECG finding, in the correct clinical context, is associated most specifically with pericarditis.

Low voltage QRS and electrical alternans is incorrect. This combination of ECG findings suggests a massive pericardial effusion.

Question 66 of 129

A 78-year-old man who is usually fit and well and takes levothyroxine for hypothyroidism presents to the acute medical take after two episodes of syncope. He recalls sitting at the dinner table and passing out with no dizziness prior. He can usually walk 100 metres at a time.

On examination, he has a loud systolic murmur heard over the 2nd intercostal space on the left side radiating to the carotid. He has a slow rising pulse and his chest is clear. No JVP is seen and there is no ankle oedema.

Chest xray	enlarged heart with calcification of the aortic knuckle
ECG	sinus, 75 beats per minute
ECHO	Aortic valve cross sectional area of 0.8mm^2 with a pressure gradient of 42mmHg, cusps appear calcified and poorly mobile

What other investigation should be performed?

A. Coronary angiography
B. Thyroid function test
C. CT chest
D. Treadmill ECG testing
E. HbA1c

A. Coronary angiography

Aortic stenosis co-occurs with atherosclerotic disease -> perform angiogram prior to surgical intervention

This gentleman meets the ECHO criteria for severe aortic stenosis and should be listed for aortic valve replacement (AVR) immediately. He shows no signs of failure or haemodynamic compromise and is therefore fit. Given that AVR involves opening the chest, it is a perfect time to perform coronary artery bypass grafting and since there is a correlation of aortic stenosis with atherosclerosis, it is beneficial to perform an angiogram prior to the AVR procedure. The patient effectively gets two procedures for one. CT chest would be important is he had respiratory disease. HbA1c and thyroid function tests are not essential unless there was a suspicion that these were abnormal. Treadmill testing is contraindicated in symptomatic and severe aortic stenosis where is can cause syncope.

Question 67 of 129

A 25-year-old male presents to the emergency department with palpitations. His observations are performed, heart rate 180/min, blood pressure 134/78 mmHg, respiratory rate 16/min. His past medical history includes asthma for which he takes salbutamol PRN and fluticasone BD. Examination reveals a clear chest. Heard sounds are too rapid to distinguish S1 and S2. ECG reveals a narrow complex tachycardia, consistent with supraventricular tachycardia (SVT). Multiple attempts at vagal manoeuvres have failed to cardiovert the patient. He remains symptomatic.

Bloods are taken:

Hb	135 g/l
Platelets	344 * 10 /l
WBC	10.1 * 10 /l
Na	141 mmol/l
K	4.2 mmol/l
Creatinine	65 µmol/l
CRP	2 mg/l

Arterial blood gases on room air:

pO2	12.1 kPa
PCO2	4.1 kPa
pH	7.39
Base excess	-0.2

Chest X-ray: No abnormality detected

Which pharmacological agent would be most appropriate for cardioverting this man?

A. Verapamil
B. Adenosine
C. Digoxin
D. Amiodarone
E. Bisoprolol

A. Verapamil

Use of adenosine is contraindicated in patients with asthma as it can cause bronchospasm. The next best option here is verapamil, which slows atrioventricular (AV) nodal conduction. The other options are not widely used to restore sinus rhythm in SVT.

Question 68 of 129

A 40-year-old male who is otherwise fit and well presents to clinic with dyspnoea on exertion. This has progressed over the last year to the point where he can only walk 0.5 miles before stopping. He denies having a cough. He has no chest pain. He has smoked 15 cigarettes per day for 25 years. He reports that his father had a myocardial infarction (MI) aged 54 years, but otherwise reports no cardiac-related family history. His body mass index (BMI) is 24 kg/m², heart rate 80/min, blood pressure 130/77mmHg, respiratory rate 18/min and he is saturating at 97% on air. Chest auscultation reveals occasional expiratory wheeze and he has no pedal oedema. Auscultation of the heart reveals a fixed split S2. His jugular venous pressure is not elevated.

Chest X-ray: dilated pulmonary vessels.

ECG: sinus rhythm, 78/min, right bundle branch block (RBBB)

What is the likely cause of his exertional dyspnoea?

A. Atrial septal defect
B. Ischaemic heart disease
C. Bronchial malignancy
D. Chronic obstructive pulmonary disease
E. Chronic pulmonary embolism

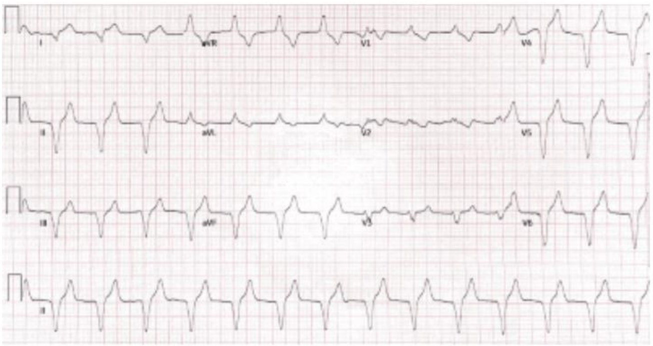
A. Atrial septal defect

The underlying cause here is likely to be an atrial septal defect. Presentation often occurs late in life with an incidental finding of RBBB. It may not be picked up in infancy as a murmur may not be audible. Presentation with symptoms occurs later in life as the right atrium becomes dilated and hence the cardiac efficiency decreases. A fixed-split S2 is a common finding in such patients

Question 69 of 129

A patient attends a cardiology clinic following a recent myocardial infarct (MI) which was successfully managed via percutaneous coronary intervention (PCI). The patient was stated on secondary prevention medications and has reports no further issues or symptoms. The patient is currently on aspirin, clopidogrel, bisoprolol, ramipril and atorvastatin.

Examination is normal and vital signs within normal range. An ECG performed is shown below.



What definitive management should be advised?

A. Arrange for urgent pacemaker insertion
B. Arrange for urgent repeat PCI
C. No additional management is required
D. Stop the patient's beta blocker and continue all othermedications
E. Arrange for urgent 24-hour Holter monitor

C. No additional management is required

The patient's ECG is a typical example of the benign ventricular ectopic rhythm, accelerated idioventricular rhythm (AIVR). It is associated with atrioventricular dissociation; P waves present but not associated with QRS complexes, wide QRS complexes (>120ms) and a rate between 50- 110 beats per minute. AIVR is common post-MI following the reperfusion of an ischaemic myocardium. It is usually self-limiting and therefore treatment is not necessary, especially as the patient is asymptomatic and haemodynamically stable.

Pacemaker insertion is not required as the atrioventricular dissociation and QRS complexes seen in AIVR does not affect the ventricular rate which is normally between 50-110 beats per minute. AIVR is due to a benign ectopic rhythm and so pacemaker insertion would not remove the cause. As the patient is asymptomatic and haemodynamically stable no treatment is required.

Arranging for urgent repeat PCI is unnecessary as there is no indication of new/further ischaemic damage or an ischaemic event having occurred. AIVR is due to an ectopic rhythm and therefore PCI is of no value.

Stopping the patient's beta-blocker is unnecessary as this will not eliminate the AIVR. Beta-blockers are an important part of secondary prevention post-MI and therefore should be continued.

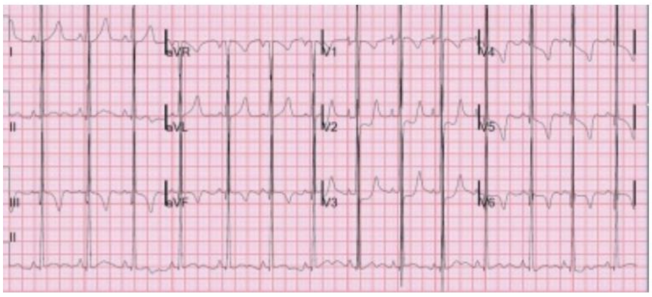
Arranging an urgent 24-hour Holter monitor is unnecessary as the patient's ECG is unlikely to change and there is no indication of any other underlying arrhythmia. The patient can be monitored in the outpatient setting as per any other post-MI patient.

Question 70 of 129

A 32-year-old female presents following a collapse whilst out jogging. The patient reports she experienced a subacute onset of chest discomfort that was quickly followed by symptoms of breathlessness and feeling faint. She thinks she 'blacked out' for a few seconds before recovering. Now she is feeling well with no symptoms.

On further questioning, she does not exercise regularly as she has experienced shortness of breath and chest discomfort in the past. She believes she has had a few episodes of feeling light-headed but has never fainted before. She is otherwise well with no recent illness or significant past medical history.

Observations are all within normal range and an ECG performed is shown below.



What is the patient's most likely diagnosis?

A. Brugada syndrome
B. Dilated cardiomyopathy
C. Hypertrophic obstructive cardiomyopathy
D. Wellen's syndrome
E. Arrhythmogenic right ventricular dysplasia

C. Hypertrophic obstructive cardiomyopathy

This patient has presented with features in keeping with hypertrophic obstructive cardiomyopathy (HOCM). The condition is due to a genetic defect causing a disorder of the cardiac muscle and, although patients can be asymptomatic, it is the commonest cause of sudden cardiac death in the young. Patients who do present with symptoms typically experience exertional dyspnoea, angina and syncope episodes, commonly during or following exertion. ECG findings, as seen in this case, are of left ventricular hypertrophy, non-specific ST-segment and Twave abnormalities, progressive T wave inversion and deep Q waves. The diagnosis can be confirmed via cardiac echo and management is dependent on the extent of obstruction and patient symptoms.

Brugada syndrome is another genetic cardiac disorder however unlike HOCM it results in electrical activity disorders and not structural issues. Features include syncope and sudden cardiac death however it is not always associated with exertion or exercise. There are several types of Brugada syndrome with variation seen on ECG however the only potentially diagnostic ECG abnormality is of coved ST-segment elevation in 2 or more of V1-3, followed by a negative T wave. This is classical of Brugada type 1 syndrome and is commonly known as Brugada sign.

Dilated cardiomyopathy is a disease of the myocardial and is characterised by progressive ventricular dilation and dysfunction. Presentation is normal with worsening biventricular failure including peripheral and pulmonary oedema. Syncope episodes are rare and symptoms are not fluctuant, as seen in this patient. ECG abnormalities are those associated with atrial and ventricular hypertrophy with conduction delays (e.g. LBBB), left axis deviation and poor R wave progression.

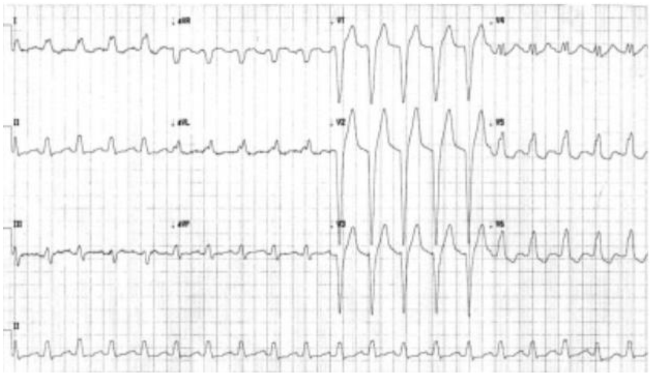
Wellen's syndrome is the ECG pattern of biphasic or deeply inverted T waves in the chest leads V2 and 3. It is highly specific for critical stenosis of the left anterior descending artery and therefore is normally seen in patients presenting with ischaemic-like symptoms.

Arrhythmogenic right ventricular dysplasia (ARVD), is due to a genetic defect affecting the desmosomes of the myocardium. ARVD results in a non-ischemic cardiomyopathy mainly affecting the right ventricle leading to areas of hypokinesia and myocardium replacement with fibrofat. These changes can results in associated arrhythmias. Patients typically present with palpitations, syncope, and potentially sudden cardiac death however ECG findings are of T wave inversion in leads V1 to V3 and of right bundle branch block which are not present in this case.

Question 71 of 129

A 72-year-old woman presents with palpitations. Her past medical history includes ischaemic heart disease and chronic obstructive pulmonary disease. Her pulse is 120/min, blood pressure 110/76 mmHg and the chest is clear on auscultation.

The ECG is shown below:



What is shown on the ECG?

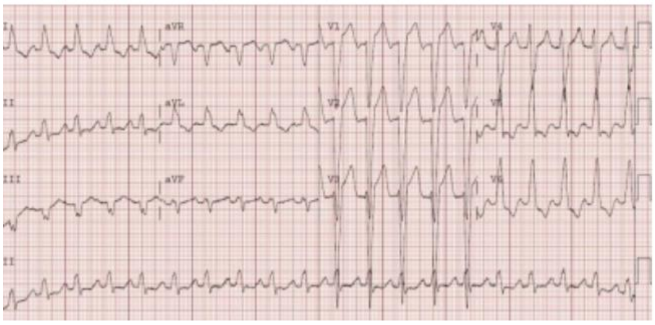
A. Left bundle branch block
B. Anterior ST elevation myocardial infarction
C. Right bundle branch block
D. Narrow complex tachycardia
E. Atrial flutter

A. Left bundle branch block

The morphology of the QRS complexes is diagnostic for left bundle branch block (LBBB). This finding can be associated with a wide variety of underlying problems but in an acutely unwell patient myocardial ischaemia needs to be excluded.

Question 72 of 129

A 58-year-old man presents with intermittent chest pains for the past two days. Whilst you are taking the history he complains of worsening pain and you arrange an ECG immediately:



He is tachycardic and sweaty with a blood pressure of 128/88mmHg.

What does the ECG show?

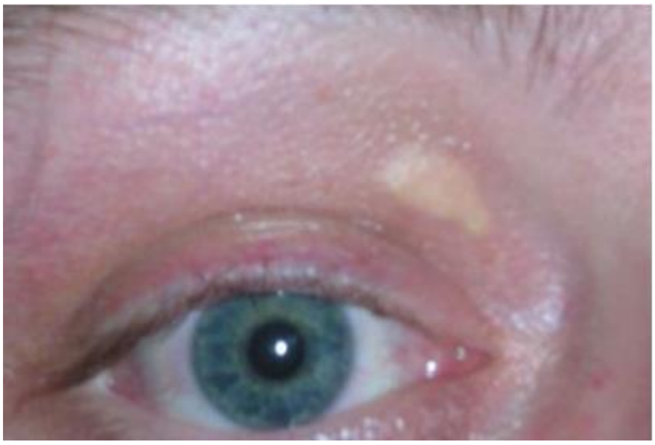
A. Narrow complex tachycardia with ST elevation
B. Right bundle branch block with ST elevation
C. Left bundle branch block with ST elevation
D. Ventricular tachycardia with ST elevation
E. Posterior myocardial infarction

C. Left bundle branch block with ST elevation

The ECG shows left bundle branch block (LBBB). The relationship between new LBBB and acute coronary syndrome (ACS) is complicated with many cardiologists ignoring the standard guidelines (both UK and US) to initiate reperfusion therapy due to the poor correlation seen in clinical practice between coronary lesions and standard LBBB. The Sgarbossa criteria have therefore been developed to evaluate whether new LBBB represents an ACS. In this particular ECG there is more ST elevation than is normally expected with LBBB indicating a likely ACS.

Question 73 of 129

A 40-year-old man presents to surgery as he has noted an abnormality around his right eye:



What is the most likely diagnosis?

A. Hypertriglyceridaemia
B. Hypercholesterolaemia
C. Hypothyroidism
D. Wilson's disease
E. Diabetes mellitus

B. Hypercholesterolaemia

The correct answer to this question is **Hypercholesterolaemia**. The image shows xanthelasma, which are yellowish plaques that commonly occur on or around the eyelids. Xanthelasma is often associated with hypercholesterolaemia, a condition characterised by high levels of cholesterol in the blood. Hypercholesterolaemia can lead to atherosclerosis and subsequently cardiovascular diseases.

Hypertriglyceridaemia is an incorrect option. While it is true that hypertriglyceridaemia can also cause xanthoma (fatty deposits under the skin), these usually present as eruptive xanthomas (small red-yellow bumps) or tuberous xanthomas (larger nodules), rather than xanthelasma.

Hypothyroidism, another incorrect option, can sometimes be associated with hypercholesterolaemia due to decreased metabolism of lipids. However, hypothyroidism itself would not directly cause xanthelasma. Hypothyroidism presents with symptoms such as fatigue, weight gain, cold intolerance, constipation, and dry skin.

The diagnosis of **Wilson's disease** does not fit the given scenario either. Wilson's disease is a rare inherited disorder causing copper to accumulate in vital organs like the liver and brain. The classic ocular manifestation in Wilson's disease is Kayser-Fleischer rings - copper deposition at the corneal limbus - rather than xanthelasma.

Lastly, **Diabetes mellitus** is also an incorrect answer for this case scenario. Diabetes could lead to various eye problems like diabetic retinopathy or cataracts but it does not cause xanthelasma. It's important for medical students to remember that while diabetes and hyperlipidemias often coexist due to their common risk factors, they are distinct conditions with different manifestations and complications.

Question 74 of 129

A 53-year-old Syrian refugee is referred to the cardiology clinic by her GP with dyspnoea and increasing peripheral oedema. Her past medical history is significant for rheumatic fever as a child. She gives a six month history of increasing dyspnoea on exertion. Her dyspnoea is now so severe that she has marked limitation in activity, being only able to walk 30 meters before stopping because of symptoms. She experiences no chest pain but tells you that she has taken to sleeping in a chair at night and has a chronic cough productive of pink sputum. More recently, she has awoken breathless from sleep terrified that she is going to die. On examination she has clinical evidence of severe mitral stenosis. She has an irregularly-irregular pulse and an ECG confirms atrial fibrillation at a rate of 89 bpm. Auscultation reveals a mid-diastolic murmur, an opening snap and a loud P2. She has bibasal crepitations and pitting oedema to below the knee. Her echo confirms severe mitral stenosis with a mitral valve area of 0.7cm2 and a mean gradient of 12mmHg. There is no evidence of mitral regurgitation and the sonographer comments that she has favourable mitral valve morphology.

Assuming a TOE excludes a left apical thrombosis, how should this lady be managed?

A. Diuresis, anticoagulate with one of the oral direct thrombin inhibitors/ factor Xa inhibitors and percutaneous mitral valve balloon valvotomy

B. Diuresis, anticoagulate with warfarin and refer for mitral valvereplacement
C. Diuresis, anticoagulate with warfarin and repeat echo in threemonths time
D. Diuresis, anticoagulate with one of the oral direct thrombin /factor Xa inhibitors and refer for mitral valve replacement
E. Diuresis, anticoagulate with warfarin and percutaneous mitralvalve balloon valvotom

E. Diuresis, anticoagulate with warfarin and percutaneous mitralvalve balloon valvotom

The correct answer is diuresis, anticoagulate with warfarin and percutaneous mitral valve balloon valvotomy.

The following is taken from the 2014 AHA/ACC consensus guidelines:

Percutaneous mitral balloon valvotomy is recommended for symptomatic patients with severe MS (mitral valve area 1.5 cm2) and favourable valve morphology in the absence of left atrial thrombus or moderate-to-severe MR.

Mitral valve surgery (repair, commissurotomy, or valve replacement) is indicated in severely symptomatic patients (NYHA class III to IV) with severe MS (mitral valve area 1.5 cm2) who are not high risk for surgery and who are not candidates for or who have failed previous percutaneous mitral balloon valvotomy.

Concomitant mitral valve surgery is indicated for patients with severe MS (mitral valve area 1.5 cm2) undergoing cardiac surgery for other indications.

With respect to anti-coagulation, the direct acting/ non-vitamin K antagonists are only indicated in non-valvular AF. Once adequate diuresis has been achieved, the addition of a beta-blocker should also be considered.

Question 75 of 129

A 48-year-old female presents with one day history of palpitation and shortness of breath. Her blood pressure is 150/90 mmHg, pulse is 190/min, respiratory rate is 21/min and oxygen saturation is 99% on air. ECG shows irregularly irregular rhythm and intermittent delta waves.

Which of the following drug should be used to control the heart rate?

A. Flecainide
B. Digoxin
C. Diltiazem
D. Verapamil
E. Adenosine

A. Flecainide

Delta waves suggest this patient has underlying Wolff-Parkinson-White syndrome in addition to atrial fibrillation. According to the NICE guidelines, in this condition, flecainide can be used as an alternative to electrical cardioversion for attempting pharmacological cardioversion. Atrialventricular node-blocking agents such as digoxin, diltiazem or verapamil should not be used. Adenosine is also an atrialventricular node-blocking agent.

Question 76 of 129

A 38-year-old man is referred to the pharmacology clinic for review. He is currently managed for hypertension with three anti-hypertensive agents, (ramipril, amlodipine and indapamide). His GP is concerned as he has still not achieved blood pressure target. On examination his blood pressure is 155/95 mmHg, pulse is 78 beats per minute and regular. There are no heart murmurs or bruits. Abdomen is soft and non tender with no masses and his body mass index is 24 kg/m².

Investigations:

Na +	142 mmol/l
K +	3.1 mmol/l
HCO3 -	30 mmol/l
Urea	7.0 mmol/l
Creatinine	90 µmol/l

Which of the following is the most likely diagnosis?

A. Bartter's syndrome
B. Conn's syndrome
C. Cushing's syndrome
D. Licorice overdose
E. Renal artery stenosis

B. Conn's syndrome

This patient has resistant hypertension in the presence of hypokalaemia and high bicarbonate, despite initiation of three different antihypertensive medications. Although the lack of symptoms or signs of cardiovascular disease doesn't rule out renal artery stenosis completely, it makes it less likely versus Conn's or licorice overdose as a cause of hypertension with hypokalaemia. Licorice overdose is made less likely by the fact that most licorice sold in shops in the UK now has glycyrrhizic acid removed, and this is the active agent which leads to hypokalaemia and hypertension. Conn's is the single most common cause of secondary hypertension, making up between 5 and 10% of cases of hypertension overall.

Bartter's syndrome leads to hypokalaemia with hypotension and is therefore not an appropriate answer here.

Question 77 of 129

A 28-year-old lady pregnant lady of 37 weeks gestation presented to the Emergency Department a few hours earlier with new onset chest pain. The pain was sharp and made worse with inspiration. She also complained of rapid onset progressively increasing shortness of breath, affecting her ability to complete sentences. She denied the presence of a cough or sputum production and did not suffer from haemoptysis or calf pain. Other than a successful external cephalic version for a breech presentation five days ago and a placenta praevia which spontaneously resolved, her pregnancy was unremarkable. She was in good health with an unremarkable past medical history and normal routine investigations throughout her pregnancy. She smoked 15 cigarettes per day and did not consume alcohol. Her mother suffered from an unexplained deep vein thrombosis when she was 42 years old but otherwise her family history was unremarkable.

Initial examination revealed a heart rate of 122bpm, respiratory rate 24/min, oxygen saturations of 98% on air, a temperature of 37.6ºC and a blood pressure of 112/72 mmHg. She was struggling to complete full sentences. Examination of her cardiorespiratory system revealed good air entry in both bases, a JVP of 3cm, the absence of pedal oedema and soft and non-tender calves. Examination of her gastrointestinal system was unremarkable and ultrasound auscultation revealed the presence of a fetal heartbeat.

As she was being tended to for initial investigations, her condition rapidly deteriorated. Her oxygen saturation dropped to 88% on air, and her blood pressure was recorded as 88/66mmHg. She appeared cool and clammy, and her respiratory rate increased to 32/min. The doctor tending to venepuncture noted the presence of oozing of blood from the wound. She was promptly transferred the intensive care unit and the following investigations were conducted:

Hb	101 g/l
Platelets	75 * 10 /l
WBC	12.2 * 10 /l
Na	136 mmol/l
K	4.8 mmol/l
Urea	14.1 mmol/l
Creatinine	158µmol/l
INR	3.9
APTT	84 s
D-Dimer	2920 ng/ml

Urinalysis: ketones ++, leucocytes/nit/prot/blood/glucose negative
Portable chest x-ray: normal appearance of heart and lung fields
ECG: sinus tachycardia 148bpm, T wave inversion leads V3-V6
Arterial blood gases on air:

pH	7.48
Pa02	5.9 kPa
PaC02	2.2 kPa
BE	1
HC03	24 mmol/l

She was commenced on 15 litres/min oxygen via non-rebreathe mask, and an arterial line and central venous line were sited, as well as the presence of two large bore peripheral cannulae. She was immediately commenced on 2 litres of Hartmann's solution stat.

What is the most likely diagnosis?

A. Pulmonary embolus
B. Septic shock
C. Peripartum cardiomyopathy
D. Aortic dissection
E. Amniotic fluid embolus

E. Amniotic fluid embolus

In this instance, pulmonary embolism would not account for the presence of DIC and there is no evidence of deep vein thrombosis clinically. Septic shock can follow a similar path to amniotic fluid embolus, but in this instance, there is little evidence for a focus of sepsis.

Question 78 of 129

A 77-year-old man has attended the Emergency Department as he is very concerned about a purple lacy rash that has appeared on his arms and legs over the past few days. It is not painful and does not itch. His past medical history includes hypertension, type 2 diabetes, diverticulitis and atrial fibrillation. He takes amlodipine, ramipril and metformin and was started on warfarin one week ago by his GP. Examination confirms a widespread purple mottled rash across all four limbs and the abdomen. His chest is clear, heart sounds are normal and the abdomen is soft, nontender.

Blood results on admission show:

Hb	119 g/l
MCV	85 fl
Platelets	142 * 10 /l9
WBC	10.1 * 10 /l9
Neuts	4.5 * 10 /l9
Lymphs	1.9 * 10 /l9
Eosinophils	1.5 * 10 /l9
Na +	142 mmol/l
K +	4.6 mmol/l
Urea	14.6 mmol/l
Creatinine	169 µmol/l

What is the most likely diagnosis?

A. Amyloidosis
B. Antiphospholipid syndrome
C. Cholesterol atheroemboli
D. Churg-Strauss syndrome
E. Focal segmental glomerulosclerosis

C. Cholesterol atheroemboli

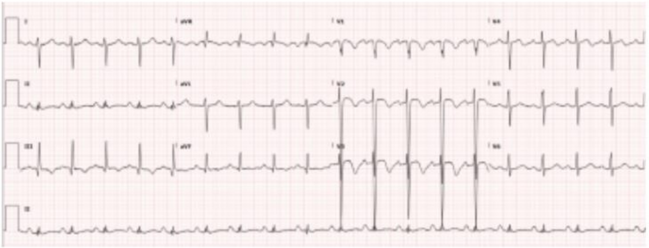
The purple, lacy, non-itchy rash described here is livedo reticularis. Antiphospholipid syndrome, Churg-Strauss syndrome and cholesterol atheroemboli can all cause livedo reticularis

Churg-Strauss and cholesterol atheroemboli could both result in raised eosinophils as seen here. However, this patient was started on warfarin a week ago by his GP which a recognised precipitant of cholesterol atheroemboli which makes it the most likely diagnosis here.

Question 79 of 129

A man in his 50's is brought to the emergency department by ambulance and was found collapsed in the street. His vital signs are as follows: Blood pressure 90/60 mmHg, pulse 120 bpm, respirations 25 bpm and temperature 37.9 ºC.

The 12 lead ECG showed:



What is the most likely cause of the patient's presentation?

A. Non-ST elevation myocardial infarction
B. Pulmonary embolism

C. ST elevation myocardial infarction
D. Severe hypokalaemia
E. Takotsubo cardiomyopathy

B. Pulmonary embolism

The ECG shows sinus tachycardia with right axis deviation, P pulmonale, Q waves in lead III and inverted T waves in the right precordial leads (V1- V3) and lead III.

Pulmonary embolism is the correct answer. Although the ECG is neither sensitive nor specific for pulmonary embolism, the classical findings of S1Q3T3 highly suggest right ventricular strain. Hence, the clinical picture is highly suspicious for pulmonary embolism, and the patient should promptly have an urgent CT pulmonary angiogram and thrombolysis if indicated. Other acute causes for right ventricular strain, like pneumothorax, upper airway obstruction and exacerbation of asthma/COPD, should also be considered in the differentials.

Non ST elevation myocardial infarction is incorrect. ECG changes like ST depression or T wave inversion in NSTEMI are often non-specific. If new, the T-wave inversions in this patient may suggest acute coronary syndrome, particularly right ventricular infarction, given the right ventricular strain pattern; however, NSTEMI is unlikely to cause collapse. The classical S1Q3T3 pattern in ECG, along with the clinical findings of tachycardia, tachypnoea and low-grade temperature, is more suggestive of pulmonary embolism in this patient.

ST elevation myocardial infarction is incorrect. Classically, STEMI is diagnosed if there is > 1-2 mm ST elevation in 2 contiguous leads or a new left bundle branch block (LBBB). Both of these are not found in this patient's ECG.

Severe hypokalaemia is incorrect. Although hypokalaemia may present with T-wave inversions in the precordial leads, there are no ST depression or U waves commonly seen in hypokalaemia. While hypokalaemia can cause muscle tetany, paralysis and respiratory failure, it is unlikely to cause collapse.

Takotsubo cardiomyopathy is incorrect. ECG changes like T wave inversion in Takotsubo cardiomyopathy are often non-specific. Moreover, the classical S1Q3T3 pattern in ECG, along with the clinical findings of tachycardia, tachypnoea and low-grade temperature, is more suggestive of pulmonary embolism in this patient.

Question 80 of 129

A 50-year-old woman was referred to the clinic with a 6-month history of shortness of breath. She gave a history of progressive exertional breathlessness and now could only walk a couple of hundred meters. In addition she also noted swelling of her ankles that started 3 weeks ago and had experienced 1 episodes of loss of consciousness. She had been a wheezy child but had grown out of it. She was a non-smoker and drank approximately 5 units of alcohol per week. She took no regular medications and had no known allergies. Her General Practitioner had prescribed salbutamol and beclomethasone inhalers for her but this had made no difference.

On examination her heart rate was 86 beats per minute, blood pressure 135/90 mmHg, respiratory rate 20 breaths per minute and oxygen saturations were 93% on room air.

Initial investigations are shown below:

Full blood count, urea and electrolytes, liver function tests: normal
Autoantibody screen: negative

Chest x-ray: clear lung fields, no cardiomegaly

Lung function tests:

- -forced expiratory volume in 1-second 2.8L (115% predicted) -forced vital capacity 3.2L (120% predicted)
- -total lung capacity 5.8L (120% predicted)
- -carbon monoxide diffusion in the lungs 92%

Transthoracic echocardiogram:

- -left ventricle: normal size, ejection fraction 65% (>55) -normal aortic and pulmonary valves
- -right ventricle dilated -tricuspid regurgitation (mild)
- -pulmonary artery systolic pressure: 55 mmHg

CT scan on chest: normal

Ventilation-perfusion (V/Q) scan: low probability for thromboembolic disease

What is the appropriate next investigation?

A. Bronchoscopy and bronchoalveolar lavage
B. Cardiac catheterisation
C. CT guided lung biopsy
D. CT pulmonary angiography
E. Genetic testing for cystic fibrosis

B. Cardiac catheterisation

The history and investigations in this case suggest a diagnosis of idiopathic pulmonary arterial hypertension (IPAH).

This condition is more common in women and commonly presents with progressive exertional breathlessness, lethargy, fatigue or syncope. On examination a loud pulmonary heart sound may be heard and eventually murmurs of pulmonary and tricuspid regurgitation may also be heard. As the disease progresses and right heart failure develops examination may reveal an elevated jugular venous pulse, hepatomaegaly, ascities and peripherial oedema.

Aside from idiopathic common causes include congenital/acquired cardiac lesions. Chronic obstructive pulmonary disease (COPD) of other lung pathology, pulmonary thromboembolic disease and vasculitic disorders.

ECHO is a good screening test but to confirming the diagnosis requires the gold standard investigation, which is cardiac catheterisation. Cardiac catheterisation will show the oxygen saturations in the veins and chambers of the heart and allow for accurate measurement of right atrial, ventricular and pulmonary artery pressures and pulmonary capillary wedge pressure, pulmonary vascular resistance and response to acute vasodilators (important implications for treatment).

The definition of IPAH includes: mean pulmonary artery pressure > 25 mmHg, pulmonary artery wedge pressure <= 15 mmHg and pulmonary vascular resistance > 3 Wood units.

Question 81 of 129

A 55-year-old man presents for review in cardiology clinic for his angina pectoris. He developed exertional chest pain two years ago and has been managed medically since that point with GTN, aspirin, atorvastatin and atenolol. He finds that he is unable to manage his angina and frequently needs the use of his GTN spray, which causes him headaches. He has a past medical history of gout and depression as well. He works as a paralegal and has quit smoking successfully five months ago. He is keen to better control the pain.

What is the most appropriate management option?

F. Add on nicorandil
G. Add on long-acting nitrite
H. Add on ivabradine
I. Refer for coronary arterial bypass graft
J. Add on calcium channel blocker

E. Add on calcium channel blocker

If angina is not controlled with a beta-blocker, a longer-acting dihydropyridine calcium channel blocker should be added

The correct answer is to add on a calcium channel blocker to his regular medications. He has been managed on monotherapy with a beta-blocker and prophylactic medication to prevent further ischaemic heart disease. Since first line monotherapy has failed, it should be combined with a calcium channel blocker following full up-titration of the monotherapy medication. If a combination of beta-blocker and calcium channel blocker fail to control symptoms then a long-acting nitrate, ivabradine, nicorandil or ranolazine can be added. Once medical management fails, surgery should be considered for further management.

Question 82 of 129

A 34-year-old woman comes to haematology clinic. She presented with a shortness of breath three days ago to a local emergency department. She underwent a CT pulmonary angiogram scan which demonstrated a pulmonary embolus. She was discharged with injections of low molecular weight heparin at treatment dose with a view of attending the anticoagulation clinic to start warfarin. She has no past medical history or allergies and only takes Microgynon as an oral contraceptive.

She is not pregnant and agrees to start warfarin.

What should be done with her heparin whilst starting warfarin?

A. Switch to prophylactic dose until INR is known to be above 2
B. Continue for one week whilst on warfarin
C. Stop same day as starting warfarin
D. Continue alongside warfarin for two weeks
E. Continue until INR is known to be above 2

E. Continue until INR is known to be above 2

The correct answer is to continue until INR is known to be above 2. This is a patient is started on warfarin whilst being covered with lowmolecular weight heparin for a pulmonary embolism. As she has not contraindications for warfarin, such as pregnancy or malignancy, warfarin should be started with a target INR of 2.5 (acceptable range 2-3). Starting warfarin can be variable and there are several protocols to achieve hospital induction, but whilst the patient has not yet been known to be at therapeutic anticoagulation, low-molecular weight heparin should be continued until a therapeutic INR is confirmed. Once this is achieved, the low-molecular weight heparin can be stopped.

Question 83 of 129

An 84-year-old female presents with a three-day history of constant chest discomfort associated with productive green sputum. On admission to the emergency department, a temperature of 38.2 degrees is recorded and she reports a recent history of 'shivering and shaking' over the past 24 hours. She reports no nausea or vomiting and was last well 72 hours ago, when she was living alone with BD carers. She has a known history of bronchiectasis, angina, hypertension, type 2 diabetes mellitus and hypertrophic obstructive cardiomyopathy.

On examination, you note bibasal coarse inspiratory crackles and an ejection systolic murmur. She has noted no deterioration in her exercise tolerance of 150 yards on flat ground. Her blood pressure is 140/85 mmHg heart rate 90/min and regular, Sats 94% on air, respiratory rate 26/min. Her ECG demonstrates ST depression and T wave inversion in V4 to V6, S wave in V1 and R wave in V5 add to 55 mm. You have no previous ECGs to compare this to. Her blood tests are as follows:

Hb	137 g/l
Platelets	402 * 10 /l
WBC	18.3 * 10 /l
Neutrophils	16.3 * 10 /l
Na	144 mmol/l
K	4.3 mmol/l
Urea	6.2 mmol/l
Creatinine	90 µmol/l
CRP	145 mg/l
Troponin < 0.03 (normal < 0.03)	

Her chest radiograph demonstrates left basal shadowing on a background of bibasal tramlining and fibrotic changes. Previous sputum cultures demonstrate pseudomonas colonisation. New sputum and blood cultures have been taken.

What is the most appropriate treatment?

A. Intravenous tazocin
B. Intravenous tazocin and acute coronary syndrome treatment
C. Acute coronary syndrome treatment only
D. Oral amoxicillin only
E. Oral amoxicillin and acute coronary syndrome treatment

A. Intravenous tazocin

The patient is elderly and has a number of comorbidities. The presenting condition is clearly infective exacerbation of bronchiectasis on a background of previous pseudomonal colonisation. As a result, oral amoxicillin is insufficient. The second question is regarding these abnormal ECG changes on a background of no cardiac chest pain and a negative first troponin. The key is to notice the history of HOCM with an ejection systolic murmur, suggesting a degree of left ventricular outlet obstruction. Her ECG demonstrates LV hypertrophy and the ST segment and T wave inversion are suggestive of LV strain, an expected pattern for patients with HOCM. No ACS treatment is thus necessary.

Question 84 of 129

A 54-year-old male business executive is referred to you after a heart murmur is detected at a medical examination he received after he transferred to a new company. He has no known past medical history or family history. He is well and leads an active lifestyle. On examination, you note a pansystolic murmur in the apex. Chest auscultation is unremarkable. An ECG demonstrates sinus rhythm at 64 beats/minute with no changes suggestive of ventricular hypertrophy. A transthoracic echocardiogram demonstrated good views with severe mitral regurgitation, preserved left ventricular function (EF 85%) and pulmonary arterial systolic pressure of 15 mmHg.

Which of the following is appropriate management?

A. Mitral valve replacement
B. Mitral valve repair
C. Percutaneous mitral valve repair (Mitraclip)
D. Infective endocarditis prophylaxis and 6 monthlyechocardiogram
E. 6 monthly echocardiogram

E. 6 monthly echocardiogram

The patient has severe chronic non-ischaemic mitral regurgitation that has not decompensated. The aetiology is unclear: primary causes include mitral valve prolapse, flail leaflet or rheumatic heart disease. Secondary causes include cardiomyopathy or coronary artery disease. The patient does not meet indications for intervention on the mitral valve, which include symptoms, left ventricular dysfunction, pulmonary hypertension, new atrial fibrillation and dilated left ventricle. It is uncommon for intervention to take place without any of the indications mentioned above. Instead, serial monitoring with 6 monthly echocardiograms is appropriate. Infective endocarditis prophylaxis is no longer indicated for patients in the absence of a prosthetic valve repair or replacement.

Percutaneous mitral valve repair is an emerging technique that is available to patients considered too high risk for mitral valve surgery, which uses a device to individually approximate the regurgitant leaflet. Comparing Mitraclip with mitral valve replacement or repair in severe mitral regurgitation patients with NYHA III/IV heart failure symptoms, one-year and four-year survival were similar with similar improvements in symptoms, LV size and function. However, Mitraclip patients demonstrated significantly higher incidence of requiring further surgery but significantly lower risks of major adverse events post-procedure.

Question 85 of 129

A 75-year-old male presents with increasing chest pain on exertion. He was first diagnosed with stable angina 7 years ago. His past medical history includes hypercholesterolaemia and hypertension. His drugs history includes bisoprolol and nifedipine, which he has been taking for the past 3 years. A recent angiogram was arranged as an outpatient by his GP, demonstrating stenoses in his left circumflex artery, distal right coronary artery and mid-left anterior descending artery.

What is the most appropriate long-term treatment of choice for his angina?

A. Coronary artery bypass graft
B. Percutaneous coronary intervention with 3 stents
C. Addition of ivabradine
D. Addition of nicorandil
E. Addition of ranolazine

A. Coronary artery bypass graft

NICE guidelines recommend the use of no more than 2 antianginals prior to consideration of reperfusion therapies. In the case of this gentleman, the consideration would be whether CABG or PCI would offer greater benefits: although the risks of recurrent MIs and stroke are similar for both PCI and CABG at one year, patients with complex anatomy, triple vessel disease or proximal left mainstem disease report better long-term survival and freedom from MI is greater with CABG. CABG is thus favoured to PCI in this patient according to American Heart Association and European Society of Cardiology guidelines.

Question 86 of 129

A 70-year-old male presents back to hospital 3 weeks after undergoing a primary percutenaous coronary intervention for anterior STEMI. He reports 24 hour history of increasing shortness of breath, fever over 39 degrees and pleuritic chest pain, different from the pain he reported when he initially presented with his ischaemic chest pain. On examination, his calves are soft, his heart sounds are normal with no added sounds and his lungs are clear on auscultation. His serum tests reveals a mildly raised troponin and ECG that demonstrates ST elevation in V2 to V4 but no reciprocal change. Angiography was performed again, demonstrating good radiological flow with no evidence of stent thrombosis. An transthoracic echocardiogram demonstrates a mild to moderate pericardial effusion with no tamponade.

What is the optimal initial treatment?

A. Aspirin, clopidogrel, low molecular weight heparin as per acute
B. coronary syndrome protocol
C. Fondaparinux
D. Bivaluridin
E. Oral prednisolone
F. Oral ibuprofen

F. Oral ibuprofen

The diagnosis is Dresslers syndrome, an inflammatory post-cardiac injury complication, characterised by high fever, chest pain with a possible pleural or pericardial rub and raised inflammatory markers. It is distinguished by late presentation compared to acute reperfusion injury, which normally occurs days to weeks after the acute MI. Dresslers syndrome is thus thought to be immune mediated instead. Most patients symptoms resolve completely with oral NSAIDs but a few refractory cases require corticosteroid therapy.

Question 87 of 129

A 43-year-old gentleman who is admitted and found to have a STEMI has chest pain on the ward. He is currently recovering from his percutaneous coronary intervention the day before and has felt well until now. The pain is over the left side of the chest and severe if he breathes. He has no cough or fevers and his observations are within normal limits.

On examination, he has normal heart sounds and a clear chest. He is mildly tender over the chest wall. His femoral puncture sites are clean with no haematoma. He is in sinus rhythm on the cardiac monitor and has soft non-oedematous calves. No JVP is seen.

Na	156 mmol/l
K	3.4 mmol/l
Urea	5.6 mmol/l
Creatinine	78 µmol/l
CRP	45 mg/l
Hb	134 g/l
Platelets	398 * 10 /l
WBC	13.0 * 10 /l
ECG	sinus rhythm, T wave inversion in V1-V3, PR 180ms and flat in most leads
Chest xray	clear lung fields, heart is at the upper limit of normal for size

What is the likely diagnosis?

A. Hospital acquired pneumonia
B. Pericarditis
C. Pulmonary embolism
D. Coronary artery dissection
E. Pleurisy

B. Pericarditis

Pleuritic chest pain at <48hrs after MI -> pericarditis

This gentleman develops chest pain in the first 24-48hrs after an MI. A hospital-acquired pneumonia would usually take longer to develop. A coronary artery dissection can be a complication of PCI but there would most likely be ischaemic changes seen on the ECG. Pleurisy is usually a post inflammatory condition after a lower respiratory tract infection. Pulmonary embolism is possible but patients with MI are anticoagulated and this is early for a PE to develop. It is recognised that a number of MI patients develop and acute pericarditis in the first 48hrs. Treatment is supportive and it is key to rule out other complications first.

Question 88 of 129

A 63-year-old female patient attends the Emergency Department with crushing central chest pain and 3mm ST segment elevation in leads II, III and aVF. She is taken to the cardiac catheter laboratory where she has a primary PCI with a satisfactory angiographic result.

Six hours later, whilst on CCU, she develops complete heart block. The patient is asymptomatic and her haemodynamic parameters are as follows:

Pulse 44bpm, regular

Blood pressure - 123/75mmHg

What is the most appropriate course of action?

A. Synchronised direct current cardioversion (DCCV)
B. Continue close monitoring and observation of the patient
C. Start an infusion of isoprenaline
D. Insertion of a permanent pacemaker
E. Insertion of a temporary pacing wire

B. Continue close monitoring and observation of the patient

In the description of the patient's presentation, it is stated that her ECG showed ST elevation in leads II, III and aVf, indicating an inferior STEMI. Complete heart block soon after an inferior MI is not uncommon, and usually resolves without the need for intervention. The key to this question is the fact that the patient is asymptomatic and haemodynamically stable. In view of this fact, it is sensible to closely monitor and observe on the assumption that given enough time postreperfusion she will return to sinus rhythm. If she was haemodynamically unstable, a temporary pacing wire would be the best option in the first instance, with an upgrade to a permanent system if she did not recover to sinus rhythm in due course.

Question 89 of 129

A 28-year-old man presented with heart palpitations. He states that he has had a a couple of episodes each week for the past 3 months. He describes the palpitations as a rapid beating of his heart. He has no past medical history and takes no regular medicines.

An ECG is performed:

P waves	Normal morphology. Inverted in lead I
PR interval	130ms
QRS	110ms. Loss of R wave progression in chest leads
QTc	410ms
Axis	Right axis deviation

What is the most likely explanation of the ECG results?

A. Torsades de pointes
B. Wolff-Parkinson-White (WPW) syndrome
C. Misplacement of the limb lead
D. Dextrocardia
E. AV nodal reentrant tachycardia (AVNRT)

D. Dextrocardia

Dextrocardia is associated with an inverted P wave in lead I, right axis deviation, and loss of R wave progression

The inverted P wave in lead I, right axis deviation, and loss of R wave progression should alert you to dextrocardia. Misplacement of the limb leads can cause a similar picture with inverted P wave in lead I and right axis deviation (RAD), however we would not expect loss of R wave progression, making dextrocardia the best answer in this question.

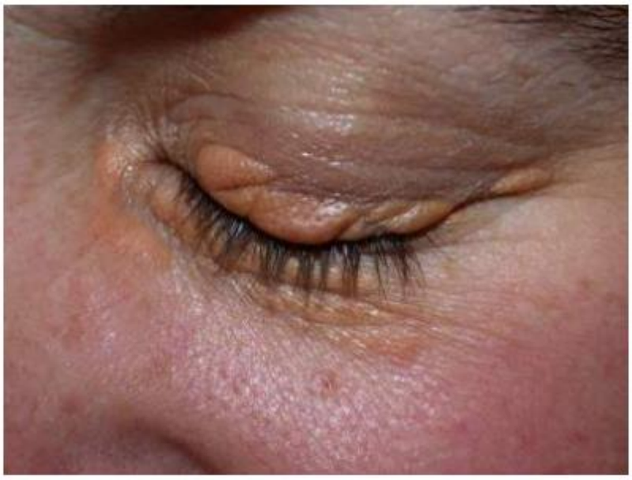
The PR interval is not short, and there is no delta waves present, making Wolff-Parkinson-White (WPW) unlikely.

The QTc is normal making Torsades de pointes unlikely

Question 90 of 129

A 56-year-old woman presents to her GP with discolouration around her eyes which has been present for some months. She feels very conscious about its appearance but denies any pain, itch or discomfort.

On examination, she appears well. The area in question appears as follows:



Given the likely diagnosis, which of the following would be an appropriate management option?

A. Topical emollient
B. Topical hydrocortisone
C. Topical ketoconazole
D. Topical trichloroacetic acid
E. Urgent referral to secondary care

D. Topical trichloroacetic acid

The photo above demonstrates xanthelasma - high lipid levels leading to soft yellow/orange plaques, periorbitally. They are not of clinical concern except for the underlying lipid profile which should be investigated and treated accordingly. They can be left alone, but if patients are keen for treatment, a commonly used option is topical trichloroacetic acid.

Topical emollient is unnecessary. This will not help to alleviate the appearance of the xanthelasma.

Topical hydrocortisone is inappropriate here. As a steroid, this would not help to reduce the appearance of xanthelasma.

Topical ketoconazole is also inappropriate. This is an antifungal and may be used for seborrhoeic dermatitis, which would instead present with dry, flaky skin and erythema - not yellow/orange plaques.

An urgent referral to a hospital is also unwarranted. Xanthelasma is of no clinical concern and requires no further investigation.

Question 91 of 129

A 32-year-old woman presented to the emergency department with a 2- hour history of acute shortness of breath. Helical CT pulmonary angiography shows no evidence of pulmonary embolus but reveals dilatation of her ascending aorta to 4.4 cm.

On examination, she is afebrile and has a heart rate of 102 beats/min, respiratory rate of 19 breaths/min, and a BP of 132/46mmHg. Her cardiac examination reveals a normal S1 with an end-diastolic murmur over the aortic valve.

Which of the following physical examination findings are most likely to be present in this patient?

A. De musset's sign
B. Presence of atrial fibrillation
C. Pulsus bigeminus
D. Pulsus paradoxus
E. Pulsus parvus et tardus

A. De musset's sign

De Musset's sign (head bobbing) is a clinical sign of aortic regurgitation

The typical presentation of severe acute aortic regurgitation includes sudden, severe shortness of breath; rapid development of heart failure and chest pain. Heart sounds usually reveal a decrescendo diastolic murmur over the aortic area. The likely diagnosis here is severe aortic regurgitation as the patient has sudden shortness of breath with an enddiastolic murmur and wide pulse pressure and the dilatation of the aorta in CT angiography.

Wide pulse pressure is due to an incompetent aortic valve that allows the diastolic pressure to fall significantly often below 60mmHg.

Some of the important aortic regurgitation clinical signs are

- Corrigan pulse - A 'water hammer' or 'collapsing' pulse is characterised by a rapidly rising and falling arterial pulse during late systole and diastole with wide pulse pressure. This finding is best appreciated by palpation of the radial or brachial arteries (exaggerated by raising the arm) or the carotid pulses.
- De Musset's sign - A head bob occurring with each heartbeat.
- Traube's sign - A pistol shot pulse (systolic and diastolic sounds) heard over the femoral arteries.
- Duroziez's sign - A systolic and diastolic bruit heard when the femoral artery is partially compressed.
- Quincke's pulses - capillary pulsations in the fingertips or lips.
- Mueller's sign - systolic pulsations of the uvula.

Pulsus paradoxus is described as a drop in systolic blood pressure of >10mmHg during the inspiratory phase, seen mostly in pericardial tamponade or severe COPD.

Pulses bigeminus occur in premature ventricular beats following a low volume pulse after a normal beat.

Pulsus parvus et tardus is more commonly known as a slow-rising pulse is a sign where on palpation of the pulse, it is late (tardus) relative to contraction of the heart and weak/small (parvus), found classically in severe aortic stenosis.

The presence of atrial fibrillation is usually present in enlarged left atrial like severe mitral stenosis or mitral regurgitation.

Question 92 of 129

A 77-year-old man with known atrial fibrillation is admitted following an upper gastrointestinal haemorrhage. His atrial fibrillation is managed using bisoprolol and warfarin. Since his admission, he has had four large episodes of haematemesis. You, the emergency department doctor, request the patient's INR to be checked as one of a series of investigations. The haematology laboratory phone through and inform you his INR is 8.5. He is currently hypotensive (90/45 mmHg) and tachycardic (120 beats per minute). You begin resuscitation using 0.9% saline, and send a cross match, group and save.

What is the most appropriate treatment of this patients INR?

A. Fresh frozen plasma + stop warfarin
B. Vitamin K + stop warfarin
C. Prothrombin complex concentrates
D. Prothrombin complex concentrates + vitamin K + stop warfarin
E. Stop warfarin

D. Prothrombin complex concentrates + vitamin K + stop warfarin

Major bleeding - stop warfarin, give intravenous vitamin K 5mg, prothrombin complex concentrate

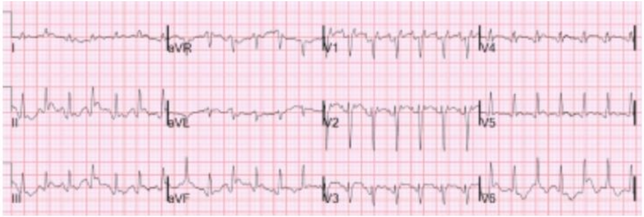
The nub of this question is the emergency management of haemorrhage in patients on warfarin. This patient has an INR greater than 8 and is actively bleeding. Therefore the answer is 4.

Patients on warfarin have reduced levels of Factor X, IX, VII and II. Rapid correction is most effectively achieved through administration of prothrombin complex concentrates.

The British Journal of Haematology states that: 'Emergency anticoagulation reversal in patients with major bleeding should be with 2550 u/kg four-factor prothrombin complex concentrate and 5 mg intravenous vitamin K

Question 93 of 129

A 70-year-old man is admitted with fever, chills and dysuria. He has a medical history of ischaemic heart disease, hypertension, hyperlipidaemia and diabetes. His current medications include aspirin, ramipril, bisoprolol, atorvastatin, metformin and dapagliflozin. He is an ex-smoker and requires assistance with personal care. He is started on cefuroxime for urosepsis. His admission ECG shows:



What is the most likely diagnosis?

A. Atrial fibrillation
B. Atrial flutter
C. Sinus tachycardia
D. Ventricular tachycardia
E. Wolff Parkinson White syndrome

B. Atrial flutter

The ECG shows an atrial rate of 300 bpm and ventricular rate of 150 bpm suggesting atrial flutter with a 2:1 AV block (regular p waves seen as notched t waves can be better appreciated in lead III, V1 and V2).

Atrial flutter is the correct answer. There are regular saw-toothed P waves at 300 bpm. There are regular QRS complexes at 150 bpm. Thus, this is atrial flutter with a rapid ventricular response and 2:1 AV block.

Atrial fibrillation is incorrect. Atrial fibrillation, unlike atrial flutter, is irregular with fibrillatory p waves caused by disorganised and chaotic atrial electrical activity. Contrary to atrial fibrillation, atrial flutter is more amenable to cardioversion than medications.

Sinus tachycardia is incorrect. In sinus tachycardia, every P wave is followed by a QRS complex and the PR interval is <200 ms. However, in this patient's ECG, not every p wave is followed by a QRS complex suggesting a 2:1 AV block. Moreover, the saw-toothed P waves at 300 bpm and prolonged PR interval is suggestive of atrial flutter.

Ventricular tachycardia is a broad complex tachycardia. The QRS complex here is narrow measuring < 120 ms and regular at 150 bpm suggesting an Atrial Flutter with 2:1 block.

Wolff Parkinson White syndrome is incorrect. WPW is a pre-excitation syndrome characterised by PR interval <120 ms, delta wave and QRS >110 ms which are absent in this patient's ECG.

Question 94 of 129

A 77 year old woman is brought to the Emergency Department by blue light ambulance in acute respiratory distress. She has a background history of congestive cardiac failure (NYHA II) due to hypertension and a previous anterior myocardial infarction which was treated medically. She is also a type 2 diabetic and has chronic kidney disease stage 2. She takes aspirin 75mg od, simvastatin 40mg od, ramipril 5mg od, bisoprolol 5mg od, furosemide 40mg bd and vildagliptin 5mg od.

On assessment her airway is patent. Respiratory rate is 32 per minute. Fingertip oxygen saturations are 90% on 15L/min oxygen. There are bilaterally crackles in the chest to above the midzones. Heart rate is 112bpm. Blood pressure is 125/75mmHg. ECG shows sinus tachycardia with no acute ischaemic change. There is peripheral oedema to the mid thigh and the JVP is elevated 6cm.

Which medical therapy is most appropriate immediately?

A. 3mg bumetanide orally
B. 80mg furosemide intravenously
C. 300mg aspirin, 300mg clopidogrel and 2.5mg subcutaneousfondaparinux
D. 50mg glycerol trinitrate infusion, commencing at 1mg/hr
E. 5mg metoprolol intravenously

B. 80mg furosemide intravenously

IV loop diuretic is the treatment for acute pulmonary oedema

This question concerns the pharmacological management of acute decompensation of left ventricular failure. It is important to point out that a patient presenting with symptoms of acute heart failure does not automatically imply an acute ischaemic event. Cardiac failure can be precipitated by changes in medication, intercurrent illness, worsening of other medical conditions or even dietary changes and it is wrong to assume a patient presenting with symptoms of cardiac failure have an acute coronary syndrome de facto. These patients should not routinely be given anticoagulant triple therapy unless there is also evidence of coronary ischaemic disease since this is unnecessary and significantly increases risk of haemorrhage. Prophylaxis against thromboembolic disease in cardiac failure is important however.

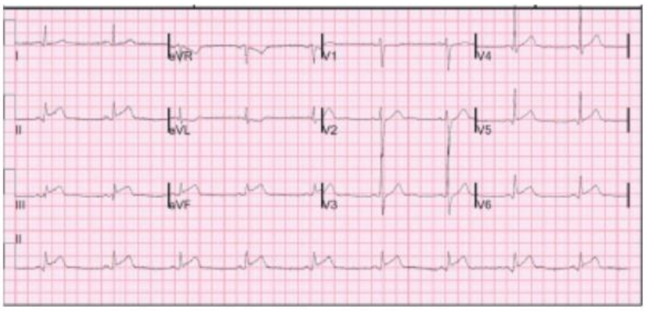
Current guidelines issued by the European Society of Cardiology and echoed by NICE guidance in the UK recommend diuretic therapy as first line medical intervention in acute cardiac failure. Intravenous administration is suggested rather than oral since this route is more rapid, there is a higher bioavailability and gut absorption can be hampered by gastrointestinal oedema in severe heart failure. Even pending the results of renal function, if pulmonary oedema is so severe to be impeding gas exchange, a bolus of furosemide is recommended to commence offloading. In patients with pre-existing renal disease, higher doses of diuretics will be needed to achieve adequate diuresis. Renal disease in itself it not a reason to avoid diuretics in heart failure, however diuretic use will result in a deterioration in renal function. Diuretic use will cause a rapid depletion in intravascular volume but the aim is to reduce tissue fluid by reducing hydrostatic pressure and a balance must be struck between deteriorating renal function due to intravascular loss and failure of therapy. Careful fluid balance monitoring is required.

Other options for fluid offloading include positive pressure ventilation which has a good evidence base behind it but patients find it uncomfortable and claustrophobic. Pressure ventilation causes an increase in intrathoracic pressure so venous return to the heart is reduced which in turn reduces cardiac output; care must be taken in hypotensive patients. Similarly glycerol trinitrate (GTN) can be used to offload pulmonary fluid by dilating arteries and to a lesser extent veins. This reduces both afterload and preload respectively reducing pulmonary oedema. Evidence demonstrates that this effect is negligible however when compared to standard diuretic therapy and should not be used first line. Indeed, it is usually only recommended in hypertensive patients with heart failure; neither GTN nor opioids are considered appropriate initial treatment of heart failure any longer, despite ongoing ubiquitous use. Side effects of GTN include hypotension, rapid tolerance, headache and complete cardiovascular collapse in cardiac outflow tract obstructions; GTN should never be given to patients with severe aortic stenosis for this reason.

Question 95 of 129

A 64-year-old man presents to the emergency department with a 2-hour history of chest pain. He has never experienced pain like this before. There is no history of shortness of breath and he reports to have been well in himself prior to today. His past medical history includes hypertension and hyperlipidaemia.

An ECG is taken, as shown below:



What is the most likely diagnosis?

A. Anterior myocardial infarction
B. Anterolateral myocardial infarction
C. Inferior myocardial infarction
D. Inferolateral myocardial infarction
E. Septal myocardial infarction

D. Inferolateral myocardial infarction

Inferolateral myocardial infarction is correct. This patient's ECG shows ST elevation in leads II, III, aVF, V5 and V6 along with ST depression in aVL. This is indicative of an inferolateral myocardial infarction. The inferior wall of the myocardium is supplied by both the right coronary artery (medial inferior wall) and left circumflex artery (lateral inferior wall). Given the ST elevation in the inferior and lateral leads, the left circumflex artery is more likely to be the occluded vessel.

Anterior myocardial infarction is incorrect. This presents with ST elevation in leads V1-V2 (septal) and V3-V4 (anterior). This is not seen here.

Anterolateral myocardial infarction is incorrect. This presents with ST elevation in the anterior leads (V3 and V4) +/- the septal leads (V1 and V2) in addition to ST elevation in the lateral leads (I, aVL, V5 and V6). Although there is ST elevation in V5 and V6, there are no ST segment changes in the anterior leads.

Inferior myocardial infarction is incorrect. Although there is ST elevation in the inferior leads (II, III and aVF), there is also ST elevation in V5 and V6 which makes an inferolateral myocardial infarction the diagnosis.

Septal myocardial infarction is incorrect. A septal myocardial infarction presents with ST elevation in leads V1 and V2 which is not seen here.

Question 96 of 129

An 82-year-old lady presented with shortness of breath and palpitations. These came on suddenly whilst she was watching television. She had a sedentary lifestyle and recently had been confined to bed with an upper respiratory tract infection. After this she had noticed that her ankles and legs were more swollen than usual, in particular her left leg. On further questioning she said that she had always needed three pillows to sleep but had recently required an extra pillow.

Her past medical history included hypertension, hypothyroidism, asthma and known coronary artery disease.

On examination her pulse was irregularly irregular. The jugular venous pressure was 6cm above the manubriosternal angle. Heart sounds were normal with no added sounds. On auscultation of the lung fields there were coarse crepitations at both bases. There was bilateral pitting oedema to the knees more marked on the left. The circumference of the left leg was 2cm greater than the right.

Observations:

- Heart rate: 128 beats per minute
- SaO2: 92% on room air
- Respiratory rate: 22 breaths per minute Temperature: 36.8 degrees Celsius Blood pressure: 110/68 mmHg

Investigations:

- 12-lead electrocardiogram: atrial fibrillation with a fast ventricular response, left ventricular hypertrophy
- Chest X-ray: cardiomegaly, Kerley B lines, small bilateral pleural effusions, pulmonary oedema
- Echocardiogram: poor biventricular function with an ejection fraction of 35%
- Computed Tomography Pulmonary Angiogram: small segmental pulmonary embolus

This lady was anticoagulated appropriately. Following this acute episode she improved clinically but remained in atrial fibrillation.

Given the clinical information available which of the following is the most appropriate drug to control her heart rate in the long term?

A. Metoprolol
B. Digoxin
C. Diltiazem
D. Flecainide
E. Warfarin

B. Digoxin

Digoxin is a good choice as this patient also has evidence of congestive cardiac failure which may also benefit from digoxin therapy. It is important to note that NICE recommends the use of digoxin only in sedentary patients and not those with an active lifestyle.

Metoprolol, a beta blocker, is not an appropriate choice here given the past medical history of asthma.

Warfarin is not a drug that is used for rate control; it is an anticoagulant.

Diltiazem is a rate-limiting calcium channel blocker and unlike digoxin does not have the added benefits in congestive cardiac failure.

Flecainide, a class 1c anti-arrhythmic drug, is used for chemical cardioversion in patients with no underlying structural heart disease as part of a pill-in-the-pocket regimen.

Question 97 of 129

A 68-year-old lady, who is a retired teacher, is admitted with lightheadedness. She has a history relevant for depression, osteoarthritis and sciatica. Her medication history includes codeine 30mg QDS, ibuprofen 400mg TDS, pregabalin 50mg TDS and amitriptyline 100mg ON. She is also on nitrofurantoin 50mg OD for recurrent urinary tract infections. Electrocardiogram (ECG) confirms normal sinus rhythm with a heart rate of 80bpm. There are no dynamic ST/T changes. PR interval is 140ms and QTc is prolonged at 526ms.

Which medication is most likely to be implicated?

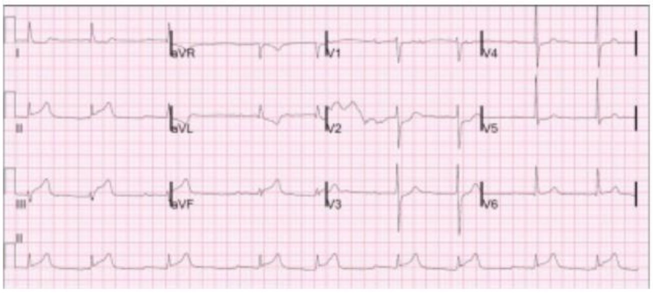
A. Nitrofurantoin
B. Pregabalin
C. Amitriptyline
D. Ibuprofen
E. Codeine

C. Amitriptyline

The correct answer is amitriptyline. Tricyclic anti-depressants are associated with prolongation of QTc interval. Other medications that can cause this include quinidine, erythromycin, digoxin, amiodarone and lithium. Prolongation of QTc interval, if marked, can result in R on T phenomenon, whereby a ventricular stimulus causes premature depolarisation of cells that have not completely repolarised. This may result in ventricular tachycardia or ventricular fibrillation. This lady is presenting with dizziness, and one should be concerned that this is a result of dysrhythmia secondary to pharmacotherapy. Any implicated medications should be temporarily discontinued, with alternatives sought.

Question 98 of 129

A 67-year-old man presents to the Emergency Department with central crushing chest pain. His ECG is shown below:



What is shown on the ECG?

A. Anterior non-ST elevation MI + atrioventricular block
B. Inferior ST elevation MI + atrioventricular block
C. Inferior ST elevation MI + left bundle branch block
D. Inferior ST elevation MI + right bundle branch block
E. Anterior non-ST elevation MI + complete heart block

B. Inferior ST elevation MI + atrioventricular block

There is ST elevation in the II, III and aVF diagnostic of an inferior myocardial infarction. The PR interval is significantly prolonged (first degree heart block) suggestive that the AV node may have been affected by the infarction.

Question 99 of 129

A 54-year-old woman presents to the emergency department with shortness of breath. She has slight associated pleuritic chest pain, but no cough and denies coughing up any blood and denies feeling hot or cold. Her symptoms started four hours ago and came on suddenly whilst at rest. She was well prior to today.

She has a past medical history of hypothyroidism and underwent a wide local excision for ductal carcinoma in situ one year ago. Her observations are all within normal range except for oxygen saturations which are at 93%. She has never smoked. Chest examination reveals few scattered crepitations heard over the right lower zone. Blood tests, including a Ddimer, are pending.

What is the most appropriate next step?

A. Warfarin
B. Chest X-ray
C. CT pulmonary angiogram
D. Ventilation-Perfusion scan
E. Treatment dose low molecular weight heparin

B. Chest X-ray

A chest xray is an essential investigation when investigating a PE

The correct answer is chest X-ray. This is a patient presenting with a typical history of a pulmonary embolism (PE). Whilst a CT pulmonary angiogram should be arranged urgently, a chest X-ray should be done first to exclude alternative pathologies such as a chest infection or pneumothorax. If a chest X-ray was done and a definitive scan such as a CT pulmonary angiogram or ventilation-perfusion scan was not available immediately then starting treatment with low molecular weight heparin would be appropriate.

'Venous thromboembolic diseases: diagnosis, management and hrombophilia testing' Clinical guideline [CG144]. The National Institute for Health and Care Excellence, June 2012.

Question 100 of 129

A 69-year-old gentleman is reviewed in cardiology clinic. He has been suffering from angina for five years and despite optimal medical management he finds that his symptoms are worsening. He has a past medical history of type 2 diabetes mellitus, gout and hypertension. He is currently taking felodipine, atenolol, atorvastatin, aspirin, ramipril and metformin. He has recently undergone an angiogram which has delineated triple vessel disease. He is very keen to optimise his survival chances as he is concerned about dying of a heart attack.

What intervention would have the greatest survival advantage?

A. Aggresive control of blood pressure, cholesterol and diabetes
B. Percutaneous coronary intervention (PCI) with insertion of stents
C. Coronary arterial bypass graft (CABG)
D. No intervention will give a further survival advantage
E. Heart transplant

C. Coronary arterial bypass graft (CABG)

The correct answer is coronary arterial bypass graft (CABG). This is a gentleman with severe ischaemic heart disease and angina. The greatest benefits are in either PCI or CABG as they address the underlying problem; reduced coronary artery blood flow. CABG is favoured over PCI in the case of people who are over 65 years old, have diabetes or have triple vessel disease. This patient has all three and therefore is a better option, but both options should be discussed in an MDT setting and with the patient.

Question 101 of 129

A 64-year-old woman presents to the emergency department with breathlessness. She has been feeling progressively breathless over the last two weeks with marked reduction in her exercise tolerance. She now finds that even a few steps make her short of breath whilst normally she would be able to walk to her local shops and back. She finds that she cannot lie down as it makes her feel worse. She has a past medical history of hypertension, previous myocardial infarction and asthma.

On examination, she has a raised JVP, bilateral crepitations on auscultation, reduced air entry in both bases and dull percussion. On her chest X-ray, there is evidence of pulmonary oedema and bilateral pleural effusions.

What is the most appropriate management plan?

A. Treat medically with diuretics
B. Pleural aspirate and if exudate insert chest drain
C. Insert chest drain on one side
D. Insert chest drain on both sides
E. Non-invasive ventilation

A. Treat medically with diuretics

The correct answer is to treat medically with diuretics. The history, examination and imaging all strongly suggest the underlying cause for her shortness of breath to be due to heart failure. The presence of the pleural effusions are therefore most likely to be transudates and it is reasonable to first treat the underlying cause before attempting to drain one or both of the effusions. If medical management with diuretics and nitrates failed to control this acute exacerbation of heart failure then non-invasive ventilation would be appropriate.

Question 102 of 129

A 45-year-old gentleman is advised by his GP to attend hospital following a routine blood test demonstrating an increase in creatinine two weeks following a dose increase in his ramipril. His ramipril was increased from 5mg daily to 5mg twice a day.

He has a letter which demonstrates investigation results, shown below. He reports no new symptoms. He reports that home monitoring of blood pressure has shown his control to be better.

21/11/2016	6/11/2016	
Na +	140 mmol/l	138 mmol/l
K +	4.5 mmol/l	4.1 mmol/l
Urea	5.5 mmol/l	5.4 mmol/l
Creatinine	110 µmol/l	92 µmol/l

How should his ramipril be managed and monitored?

A. Continue ramipril and repeat U&Es in 1-2 weeks
B. Reduced the dose of ramipril and repeat U&Es in 1-2 weeks
C. Stop ramipril and repeat U&Es in 1-2 weeks
D. Stop ramipril and arrange for an urgent out-patient imaging toexclude renal artery stenosis
E. Continue ramipril and repeat U&Es within 48 hours

A. Continue ramipril and repeat U&Es in 1-2 weeks

The correct answer is to continue ramipril and repeat U&**Es** in 1-2 weeks. Up to an increase in creatinine of 30% can be tolerated following the start or increase in an ACE-inhibitor such as ramipril, but U&Es should be repeated in 1-2 weeks. If the increase is greater than 30% then the ACEinhibitor should be stopped.

Question 103 of 129

A 75-year-old gentleman with a background of type II diabetes mellitus, ischaemic heart disease (IHD) and New York Heart Association (NYHA) III heart failure presents to your clinic with continuing fatigue and shortness of breath on minimal exertion. He currently takes Aspirin 75mg, Bisoprolol 10mg, Ramipril 5mg, Furosemide 40mg twice daily and Spironolactone 25mg. His electrocardiograph demonstrates left bundle branch block (old) and his ejection fraction is 32% measured by echocardiography at the clinic today. His chest x-ray shows an enlarged cardio-thoracic ratio but no obvious pulmonary oedema. Blood tests taken with his general practitioner this week are as follows:

Haemoglobin	110g/l
White cell count	11.0 *10^9
Neutrophils	7.2 *10^9
Platelets	240 *10^9
Na+	137 mmol/l
K+	4.2 mmol/l
Urea	9 mmol/l
Creatinine	125 µmol/l

What evidence-based treatment, if any, is available to treat this gentleman's cardiac failure?

A. Increase dose of Spironolactone to 50mg once daily
B. Increase dose of Furosemide to 80mg twice daily
C. Cardiac-resynchronisation therapy
D. Angiography +/- coronary stenting
E. He is already on maximal treatment

C. Cardiac-resynchronisation therapy

NICE guidance has recommended that cardiac resynchronisation therapy can be offered to patients who fulfil all of the following criteria:

- NYHA III or IV heart failure
- Ejection fraction of <35%
- The heart is beating regularly with evidence of electrical conduction disease\
- They are medication that is most effective for them

This patient is on appropriate doses of secondary-prevention and heart failure medications and there is no evidence of an acute coronary syndrome but the previous IHD is likely the cause of his cardiac failure. He satisfies all of the above criteria for cardiac resynchronisation and this has a good evidence-base.

Question 104 of 129

A 76-year-old woman is brought into the Emergency Department following an episode of loss of consciousness. On more detailed review, she reveals that she has been suffering increasingly with shortness of breath on exertion, with great difficulty in managing her shopping and cleaning.

Her past medical history include right hip fracture 6 years ago after a fall walking down the stairs; this required a total hip replacement.

She takes no regular medication and has no drug allergies.

On examination, her pulse was 80 beats per minute, blood pressure 104/89 mmHg, and respiratory rate 16 breaths per minute. She had a slow rising pulse, and on auscultation of the chest there was an ejection systolic murmur heard loudest at the aortic area, and radiating to the carotid arteries.

Electrocardiogram (ECG) shows sinus rhythm and criteria for left ventricular hypertrophy.

Chest x-ray shows clear lung fields. Transthoracic echocardiogram:

Aortic valve area	0.8 cm² (3-4)
Transvalvular gradient	55 mmHg (0)

The diagnosis of aortic stenosis is explained to the patient, and she opts for open surgical valve replacement.

What other investigation is warranted before proceeding to aortic valve replacement?

A. Exercise tolerance testing
B. Dopamine stress echocardiogram
C. 24 hour holter ECG
D. Transoesophageal echocardiogram
E. Coronary angiography

E. Coronary angiography

The valve area <1 cm² and transvalvular gradient >50 mmHg on transthoracic echocardiogram confirm the diagnosis of severe aortic stenosis, and the patient has opted for open surgical valve replacement.

Patients undergoing open surgical valve replacement should first undergo coronary angiography to exclude any coronary stenosis that could simultaneously be treated with bypass grafting.

This patient has undergone an 12 lead ECG and transthoracic echocardiogram. Further ECG or echocardiogram studies are not usually necessary.

Question 105 of 129

A 50-year-old man is recovering 12 hours after a stent placement for a ST elevation myocardial infarction in which there was a small stenosis of the circumflex artery. He has returned to the coronary care unit and you are called to see him due to changes on the cardiac monitor.

There are runs of broad complexes lasting 3-10 seconds at a rate of 80 per minute. He is asymptomatic and on waking is able to tell you that he has no chest pain or palpitations.

Na +	135 mmol/l
K +	3.2 mmol/l
Urea	5.3 mmol/l
Creatinine	89 µmol/l
Magnesium	0.61mmol/l (normal range 0.6-1.0)
Troponin	3000pg/ml (normal range <20)
ECG	Broad complexes at 73 beats per minute running in sets of 3-15 with normal sinus rhythm between. PR 150ms, QTc 450ms. No ST elevation at present

What action should be taken?

A. Reassure
B. IV magnesium sulfate
C. Transcutaneous pacing
D. DC cardioversion
E. Amiodarone

A. Reassure

Accelerated idioventricular rhythm is common and unconcerning following recent MI

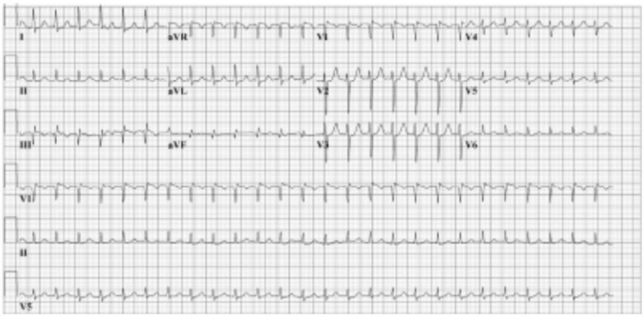
This gentleman has an MI that has been successfully treated and is recovering well. He is at risk of malignant arrhythmias like ventricular tachycardia during this time. However these broad complexes are not tachycardic. This phenomenon is accelerated idioventricular rhythm which commonly follows an MI in which ventricular ectopic tissue take over the pacemaker role. This usually resolves with time but can be helped by atropine if the patient is symptomatic. In this case, there are no symptoms and so the best action is to reassure the patient and continue monitoring. All other options would not be appropriate given the benign nature of this rhythm.

Question 106 of 129

A 41-year-old woman attends the emergency department with a 6-hour history of palpitations and breathlessness. She denies chest pain or recent illness. Her past medical history includes type 1 diabetes and she smokes 15 cigarettes/day.

On examination, she has a heart rate of 140bpm with a blood pressure of 122/77mmHg. Peripheral pulses are present bilaterally with a capillary refill of < 2 seconds. On auscultation, her heart sounds are normal with vesicular breath sounds throughout.

An ECG is taken, as shown below:



What is the most likely diagnosis?

A. Atrial flutter
B. Sinus tachycardia
C. Supraventricular tachycardia
D. Ventricular tachycardia
E. Wolff-Parkinson White syndrome

C. Supraventricular tachycardia

This patient's ECG shows a regular, narrow-complex tachycardia in keeping with supraventricular tachycardia (SVT). Given the absence of clear P waves, it is likely that the cause of this SVT is an atrioventricular nodal re-entry tachycardia (AVNRT) secondary to a re-entry circuit causing premature ventricular contraction. SVT is more common in women with a heart rate varying from between 140-280bpm. Symptoms include palpitations and breathlessness, as seen here, and may occur spontaneously or following provocation with caffeine, drugs or alcohol.

Atrial flutter is a form of supraventricular tachycardia caused by a reentry circuit into the right atrium. This typically causes an atrial rate of 300bpm. The ventricular rate is determined by the degree of the atrioventricular block at the atrioventricular node e.g. a 2:1 block would result in a heart rate of 150bpm, whilst a 3:1 block would give a rate of 100bpm. Symptoms include palpitations or shortness of breath, whilst common ECG findings include a narrow complex tachycardia with inverted flutter waves in leads II, III and aVF, giving rise to a saw-tooth appearance on the baseline of the ECG. These are not seen here, making it a less likely diagnosis.

Sinus tachycardia is a common ECG finding in patients presenting with palpitations. However, the absence of discernable P waves makes sinus tachycardia less likely.

Ventricular tachycardia presents as a broad-complex tachycardia with widened QRS complexes. This ECG shows a narrow-complex tachycardia, making ventricular tachycardia unlikely.

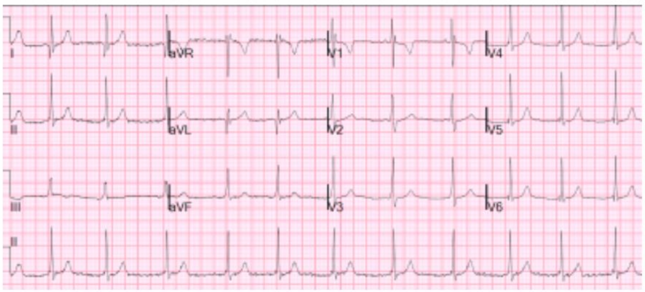
Wolff-Parkinson White (WPW) syndrome causes a tachyarrhythmia secondary to an accessory excitatory pathway stimulating premature ventricular contraction. WPW syndrome may present as palpitations, as seen in this patient. However, the ECG findings in Wolff-Parkinson White include a shortened PR interval and a slurred upstroke of the QRS complex (delta wave) causing QRS interval prolongation which is not seen here

Question 107 of 129

A 40-year-old woman presents to the emergency department with a 10- day history of intermittent palpitations. She reports that her symptoms have worsened over the last 8 hours and her palpitations are now persistent and are associated with lightheadedness. She has no past medical history of note and takes no regular medications.

On examination, she appears clammy and warm to the touch. Her heart rate is 72 beats/min with a blood pressure of 125/83mmHg.

An ECG is taken (as shown below):



What is the most likely diagnosis?

A. Accelerated junctional rhythm
B. Atrial flutter
C. Normal sinus rhythm
D. Supraventricular tachycardia
E. Wolff-Parkinson white syndrome

E. Wolff-Parkinson white syndrome

This patient has **Wolff-Parkinson White syndrome** a pre-excitation syndrome that causes clinical features of palpitations, shortness of breath, chest pain and in severe cases collapse and shock, including cardiac arrest. Wolff-Parkinson White is caused by an accessory pathway conducting impulses from the sinoatrial node to the ventricles giving rise to an atrioventricular re-entry tachycardia. ECG features include a shortened PR interval, left axis deviation or right axis deviation (depending on the location of the accessory pathway) and a widened QRS complex from the 'delta' wave (a slurred upstroke of the QRS complex). The delta wave can be seen in this patient's ECG, along with a shortened PR interval.

Accelerated junctional rhythm is incorrect. Accelerated junctional rhythm is when the atrioventricular node conducts at a faster rate than the sinoatrial node. This gives rise to an ECG with appearances of a narrow complex rhythm, with a ventricular rate between 60-100 beats/min. Retrograde P waves can be seen. Causes include digoxin toxicity, beta-blockers and myocardial ischaemia.

Atrial flutter is incorrect. Atrial flutter is an irregular supraventricular arrhythmia. Although it can cause similar symptoms to those experienced by the patient, a characteristic ECG finding is a saw-tooth appearance of the baseline of the ECG which is not seen here.

Normal sinus rhythm is incorrect. Although the patient's rhythm is sinus with QRS complexes following a P wave. There are clear delta waves preceding the QRS complexes that are consistent with Wolff-Parkinson White syndrome.

Supraventricular tachycardia (SVT) is incorrect. SVT is caused by a reentry circuit within the atrioventricular node. This is in contrast to atrioventricular re-entry tachycardia which is caused by an accessory conducting pathway (e.g. Wolff-Parkinson White syndrome). Symptoms are similar to this patient's presentation with palpitations, shortness of breath and chest pain that may be exacerbated by caffeine or alcohol. However, the presence of a delta wave is more suggestive of an accessory conducting pathway, making supraventricular tachycardia.

Question 108 of 129

You are asked to see a 64-year-old woman in the emergency department who has presented with a 3-day history of pleuritic chest pain and smallvolume haemoptysis.

She has a past medical history of hypertension, stage 4 chronic kidney disease, previous deep venous thrombosis (DVT), and a newly diagnosed primary breast cancer awaiting multidisciplinary team discussion.

You note crackles at the right lung base. There are no added heart sounds and no limb swelling is noted.

Her observations are as follows:

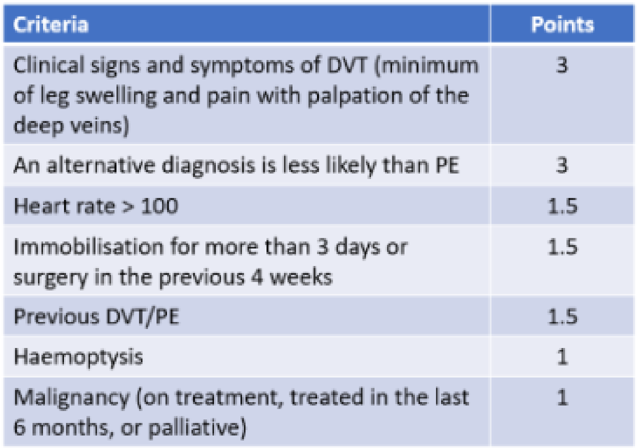
- Oxygen saturation: 98% on room air
- Respiratory rate: 18 per minute
- Heart rate: 104bpm
- Blood pressure 110/57 mmHg

Her blood results return as below.

Hb	116 g/L	Male: (135-180)Female: (115 - 160)
Platelets	181 * 10 /L	(150 - 400)
WBC	6 * 10 /L	(4.0 - 11.0)
Na	137 mmol/L	(135 - 145)
K	6.1 mmol/L	(3.5 - 5.0)
Urea	13.6 mmol/L	(2.0 - 7.0)
Creatinine	382 µmol/L	(55 - 120)
CRP	34 mg/L	(< 5)

Chest x-ray shows no obvious abnormality.

2-level PE Wells test



Criteria	Points
Clinical signs and symptoms of DVT (minimum of leg swelling and pain with palpation of the deep veins)	3
An alternative diagnosis is less likely than PE	3
Heart rate > 100	1.5
Immobilisation for more than 3 days or surgery in the previous 4 weeks	1.5
Previous DVT/PE	1.5
Haemoptysis	1
Malignancy (on treatment, treated in the last 6 months, or palliative)	1

Of the options given, which is the most appropriate next step?

A. Arrange for CT pulmonary angiogram
B. Arrange for Doppler ultrasound
C. Arrange for V/Q Scan
D. Arrange for d-dimer
E. Arrange for echocardiography

C. Arrange for V/Q Scan

This patient has a Wells score of 8, given that it is the most likely diagnosis, she has active malignancy, a history of previous DVT, a heart rate >100, and is presenting with haemoptysis. As they have scored more than four, pulmonary embolism (PE) would be considered likely in this patient, and therefore merits further imaging to further evaluate this, without the need for a d-dimer.

A **V/Q Scan** would be the investigation of choice for confirming a diagnosis of PE given this patient's renal function.

A **CT pulmonary angiogram** would have been the recommended initial lung-imaging modality for non-massive PE if not for being contraindicated by the patient's renal function.

Echocardiogram can be used in patients who are haemodynamically unstable, but as this is not the case here, it is not the initial investigation of choice.

Arranging for Doppler ultrasound is incorrect given there are no symptoms suggesting a current DVT.

D-dimer would be the appropriate next step for risk stratification if the patient had a Wells score of less than four.

Question 109 of 129

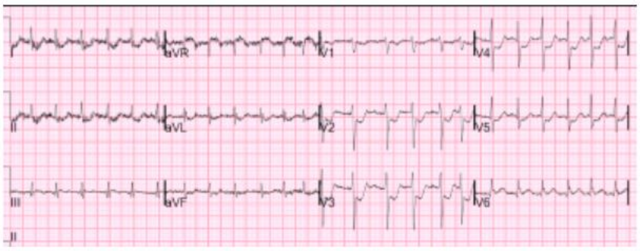
A 34-year-old male is brought in from a nearby bar having collapsed. His friends who accompany him tell you that the patient had been moderately intoxicated but had not reported any symptoms prior to his collapse.

On arrival, the patient is conscious but reports feeling generally weak and light-headed. He has never had an episode like this before but has had episodes of palpitation with associated shortness of breath previously.

The patient states he smokes 20 cigarettes/day, often binge drinks consuming 10 plus pints of beer most weekends and occasionally uses cocaine.

On examination, the patient is conscious and reports some lightheadedness and palpitation but feels otherwise ok when lying down. He has a fast, thready pulse between 130-150bpm and a blood pressure of 89/65mmHg. He has a mildly raised respiratory rate but his chest is clear and peripheral saturations are 94% on room air. Heart sounds are normal and examination is otherwise unremarkable.

An ECG is performed as below



What is the most appropriate management?

A. IV adenosine
B. IV metoprolol
C. Cardioversion
D. Pacemaker insertion
E. Percutaneous coronary intervention

C. Cardioversion

This patient has presented with a tachyarrhythmia and is haemodynamically unstable with an acute collapse, most likely syncope induced, and shock, demonstrated by the hypotension and cool extremities, etc. As seen on the ECG the patient's rhythm is fast and irregular with a varied rate of between 100 and 150 bpm. The QRS complexes are narrow and clear 'p' waves cannot be seen before each complex. This is in keeping with a tachyarrhythmia, most likely secondary to atrial fibrillation however the specific cause is not relevant as urgent synchronised cardioversion is required in any unstable patient presenting with a tachyarrhythmia. Cardioversion should be carried out in a resuscitation setting, with procedural sedation if possible. Other indications of instability in arrhythmias include evidence of myocardial ischaemia and heart failure. Following cardioversion, further management is based on the QRS complexes (narrow vs broad) and the rhythm (regular vs irregular).

Adenosine is the treatment of choice in patients presenting with supraventricular tachycardias (SVT), which may be the underlying diagnosis in this case, however, it should be used in hemodynamically stable patients only. As the patient is unstable cardioversion is required.

Metoprolol is an IV beta-blocker and, although it can be used in some tachyarrhythmias, this patient is unstable and requires urgent electrical cardioversion. IV beta-blockers can be used in stable patients with tachyarrhythmias (e.g. atrial fibrillation) for rate control as so long as they have an adequate systolic blood pressure and there are no other contraindications (e.g. asthma).

Pacemaker insertion is used in the management of bradycardia arrhythmias and does not have a role in acute arrhythmias causing compromise.

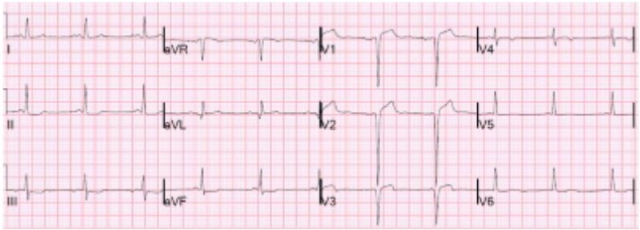
Percutaneous coronary intervention is used in the management of myocardial infarction (MI). Although the patient has some risk factors, and cocaine use can induce an MI, it is unlikely to be the underlying issue in this case with the patient not reporting chest pain **symptoms** and no clear evidence of ST-elevation seen on the ECG.

Question 110 of 129

A 55-year-old man presents to the emergency department with a 1-day history of central chest pain. He also complains of progressive shortness of breath on exertion over the last 2 weeks. He was discharged from the cardiology ward 21 days ago after having a drug-eluting stent inserted following an anterolateral myocardial infarction. His past medical history includes diet-controlled type 2 diabetes mellitus and hypertension.

On examination, he is afebrile with a temperature of 36.4ºC. His heart rate is 88 beats/min and regular with a blood pressure of 139/82mmHg. On auscultation, there are bibasal crepitations with saturations of 90% on air. There is pitting oedema to his mid-shins with soft and non-tender calves.

An ECG is taken, as shown below:



What is the most likely diagnosis?

A. Anterior myocardial infarction
B. Left ventricular aneurysm
C. Papillary muscle rupture
D. Pericarditis
E. Pulmonary embolism

B. Left ventricular aneurysm

This patient has a diagnosis of a **left ventricular aneurysm**, a rare but serious complication of an anterior myocardial infarction. The complication tends to present more than 2 weeks after a myocardial infarction and symptoms may include chest pain or congestive heart failure (e.g. shortness of breath and fluid overload). This patient's ECG shows ST elevation in V1-V3 with QS waves and upright T waves. Persistent ST elevation after a myocardial infarction is suggestive of a left ventricular aneurysm. However, in a patient with a recent history of ischaemia, it is also important to exclude another ischaemic event. A left ventricular aneurysm is much more likely if there are absent reciprocal ST changes, dynamic ST changes and well-formed Q or QS waves.

Acute coronary syndrome is an important diagnosis to consider following a myocardial infarction. However, ECG findings that are more typical of acute coronary ischaemia include new ST changes with dynamic and reciprocal changes, which are not seen here. Furthermore, the more gradual onset of symptoms is more suggestive of an alternative diagnosis such as a left ventricular aneurysm.

Papillary muscle rupture is incorrect. An anterolateral myocardial infarction causes rupture of the anterolateral papillary muscle, producing symptoms of mitral regurgitation. Although symptoms of congestive cardiac failure are evident in such cases, the onset of symptoms is usually more acute compared to a left ventricular aneurysm. Furthermore, it does not explain this patient's ECG changes or normal heart sounds (where a pansystolic murmur of mitral regurgitation may be seen).

Pericarditis is incorrect. Unlike Dressler's syndrome, post-MI pericarditis is an acute complication within the first 48 hours. The presentation is also similar to acute pericarditis with pleuritic chest pain worse on lying flat.

Pulmonary embolism is incorrect. ECG findings consistent with a pulmonary embolism include sinus tachycardia, RAD, RBBB or S1Q3T3. It does not explain this patient's ST elevation.

Question 111 of 129

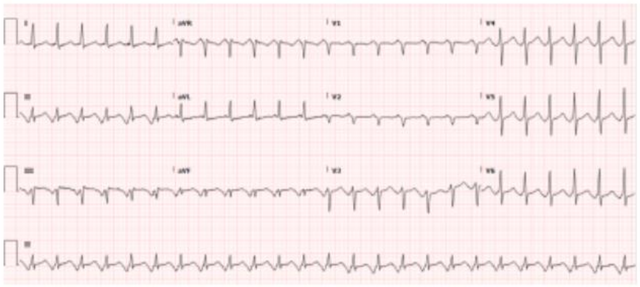
A 26-year-old woman has attended the emergency department with palpitations and shortness of breath. She has a background of asthma for which she has beclometasone and salbutamol inhalers.

On further questioning, she denies chest pain, dizziness, and loss of consciousness.

Observations are as follows:

Heart rate	200 bpm
Blood pressure	102/56 mmHg
Oxygen saturations	98%
Respiratory rate	28 /min
Temperature	36.8ºC

Her ECG is shown below:



Of the following options, what is the most appropriate management?

A. Direct current (DC) cardioversion
B. Intravenous adenosine 12mg
C. Intravenous adenosine 6mg
D. Intravenous metoprolol
E. Intravenous verapamil

E. Intravenous verapamil

The above ECG shows a narrow complex tachycardia of approximately 200bpm. This is also associated with an absence of P waves, except for P wave inversion in the inferior leads, otherwise known as retrograde conduction. These findings are in keeping with atrioventricular nodal reentry tachycardia, or AVNRT, the most common form of supraventricular tachycardia.

The correct management option in the above scenario is **intravenous verapamil**. All five of the above options can be used in patients with supraventricular tachycardia, however, verapamil is the most appropriate given the patient's clinical state and past medical history of asthma.

Following advanced life support algorithms, **DC cardioversion** should be used in narrow complex tachyarrhythmias when the patient is unstable. The above scenario does not describe evidence of heart failure, syncope, chest pain, or haemodynamic instability (shock). This option is therefore incorrect.

Intravenous adenosine 12mg is used in chemical cardioversion of narrow complex tachyarrhythmias, however, should only be used when 6mg of adenosine has failed to cardiovert. Furthermore, adenosine is contraindicated in patients with asthma. For these reasons, this option is incorrect.

Intravenous adenosine 6mg is often the initial medical management in narrow complex tachyarrhythmias. Although this would normally be the next option in management, adenosine is contraindicated in patients with asthma, making this option incorrect.

Intravenous metoprolol is also an option in the management of supraventricular tachycardias. Cardioversion is caused by heart rate reduction leading to self-termination of the tachyarrhythmia. However, due to the patient's asthma history, beta-blockers should be avoided.

Question 112 of 129

A 55-year-old Afro-Caribbean man visits his general practitioner for a follow-up of his hypertension management. He was diagnosed with hypertension 5 years ago, and his past medical history is significant for hypercholesterolemia. His hypertension has been treated with ramipril and amlodipine. Over the past 6 months, his blood pressure chart shows consistently high readings. He has already tried decreasing salt intake and exercising more. He does not smoke or consume alcohol.

On examination, his blood pressure is 156/97 mmHg, and pulse is 80/min. His BMI is 32 kg/m² with an increased waist-to-hip ratio.

Which of the following is the next best step in the management of this patient's hypertension?

A. Add benazepril
B. Add chlortalidone
C. Change ramipril to losartan
D. Refer to bariatric surgery
E. Advise lifestyle changes and reassess in 6 months

B. Add chlortalidone

In black individuals who are already taking a calcium channel blocker and ACE-inhibitor, and who continue to have blood pressure that is difficult to control, adding a thiazide-like diuretic is the next most appropriate step

This patient has primary hypertension that is not controlled by an ACEinhibitor and calcium channel blocker. The addition of another antihypertensive drug is the most appropriate management in this case. The choice of drug depends on the race of the patient. For black individuals, the recommendation is to add a thiazide-like diuretic, like chlortalidone or indapamide.

This patient is already taking an ACE inhibitor, ramipril. The guidelines recommend adding a drug with a different mechanism of action. Adding another ACE inhibitor, like benazepril, is inappropriate.

In some patients, ACE inhibitors may cause side effects. In such patients, it is suggested to replace the ACE-inhibitor with angiotensin-receptor blockers, like losartan. This patient does not report any side effects associated with ACE inhibitors.

Bariatric surgery is a treatment option for people with obesity if all of the following criteria are fulfilled:

- A BMI of 40 kg/m or more, or
- BMI between 35 kg/m and 40 kg/m and another significant disease (for example, type 2 diabetes or high blood pressure) that could be improved if they lost weight.

With a BMI of 32 kg/m , this patient does not fulfil the criteria for bariatric surgery.

This patient has already tried lifestyle changes. Although he should continue these lifestyle changes, he also needs a change in his drug regimen.

Question 113 of 129

A 32-year-old woman presents to the emergency department with palpitations. Her past medical history includes type 2 diabetes mellitus and acne vulgaris. She is currently taking Yasmin (a combined oral contraceptive pill) and has no known allergies.

Examination reveals a strong radial pulse, which is tachycardic and regular. She has a raised body mass index. Her cardiopulmonary system is unremarkable.

Observations:

- Heart rate: 168/min
- Blood pressure: 138/88 mmHg
- Oxygen saturations: 98% on room air
- Temperature: 37ºC

An ECG shows a supraventricular tachycardia.

The foundation year two doctor has already tried vagal manoeuvres and has just given 6mg of adenosine to no effect.

What is the most appropriate next step in management?

A. Amiodarone IV infusion
B. 12mg adenosine IV
C. Beta blocker IV
D. Cardioversion
E. Further 6mg adenosine IV

B. 12mg adenosine IV

Supraventricular tachycardia follows the narrow complex, regular tachycardia ALS guidelines.

If the patient is haemodynamically stable with no adverse features, such as shock, myocardial ischaemia or heart failure:

- Start with vagal manoeuvres. Carotid sinus massage or the Valsalva manoeuvre
- 2. Adenosine 6 mg rapid IV bolus; if unsuccessful give 12 mg; if unsuccessful give further 12 mg
- 3. If unsuccessful, seek specialist help

Use a large cannula to administer the adenosine and give a rapid bolus. A history of asthma is an important contraindication to adenosine, as it can cause bronchospasm. Instead, a calcium channel blocker should be used.

If the patient in unstable, they should the electrically cardioverted start with 70-120 J biphasic (100 J monophasic).

Question 114 of 129

A 72-year-old man presents to the emergency department with dizziness. There is no chest pain, palpitations or shortness of breath. He has a past medical history of hypertension. He takes amlodipine. He does not smoke or drink alcohol.

His observations are as follows:

- Heart rate 48 beats per minute
- Blood pressure 125/84 mmHg
- Respiratory rate 18/minute
- Oxygen saturations 96% on room air
- Temperature 37.1C

On examination, he is now asymptomatic. His heart rate is irregular. There are no murmurs. Chest auscultation is normal. His jugular venous pulse is not elevated. His Glasgow coma scale is 15/15.

An ECG demonstrates intermittently non-conducted P waves associated with a prolonged PR interval that is constant and not progressive. The QRS is broad. There are no ischaemic changes.

Bedside echocardiography reveals an approximately normal ejection fraction.

Given the likely diagnosis, what is the most appropriate management?

A. Atropine
B. Biventricular implantable cardioverter defibrillator (ICD) insertion
C. Dual chamber pacemaker insertion
D. Monitoring
E. Single chamber pacemaker insertion

C. Dual chamber pacemaker insertion

In patients with Mobitz type II AV block, or complete heart block, a DDD or DDDR pacemaker is indicated

Dual chamber pacemaker insertion is the correct answer. The patient has high grade atrioventricular (AV) block (2nd degree, Mobitz type II). This is characterised by intermittent non-conduction of P waves associated with a prolonged PR interval. In contrast to Mobitz type I, the PR interval is constantly prolonged and does not progressively lengthen. Mobitz type II is typically caused by significant structural damage to the conducting system. Mobitz II is much more likely than Mobitz I to degenerate to haemodynamic compromise and progress to complete heart block. Pacemaker insertion is warranted. It should be a dual chamber pacemaker on setting DDD or DDDR. DDD pacing means that the pacemaker can record both atrial and ventricular rates and can pace either chamber as necessary. DDDR is the same but can adjust the heart rate to account for the need for higher cardiac output.

Atropine is incorrect. 2nd degree AV block is typically not responsive to atropine. Additionally, while this patient certainly needs a pacemaker, he is now asymptomatic with no adverse features and therefore does not need atropine urgently.

Biventricular implantable cardioverter defibrillator (ICD) insertion is incorrect. This is typically indicated in patients with an ejection fraction of < 35% and symptomatic heart failure. It is not strictly inserted for high risk bradyarrhythmias.

Monitoring is incorrect. He is at risk of sudden deterioration and needs definitive management.

Single chamber pacemaker insertion is incorrect. Single chamber pacemakers are predominantly used for the management of atrial arrhythmias and are not indicated in those with AV block.

Question 115 of 129

A 58-year-old woman was referred to the acute medical unit with headaches and hypertension. She had no history of fever, neck stiffness, limb weakness, seizures or blurring of her vision. Her pulse rate was 70 beats per minute and blood pressure was 200/110 mmHg. Her heart sounds, chest examination, abdominal and neurological examination were normal. Fundoscopy was also normal.

Hb	138g/l
Platelets	238 * 10 /l9
WBC	6.2 * 10 /l9
Na +	135 mmol/l
K +	3.8 mmol/l
Urea	6.4 mmol/l
Creatinine	75 µmol/l
ECG	normal sinus rhythm
Chest x-ray	normal
CT Head	normal
Urinalysis	normal

What is the best initial management?

A. Intravenous labetalol
B. Intravenous glyceryl trinitrate
C. Oral amlodipine
D. Intravenous sodium nitroprusside
E. Sublingual nifedipine

C. Oral amlodipine

In an hypertensive urgency, treatment aims to lower blood pressure with the use of oral anti-hypertensive medication like a calcium channel blocker

This woman has hypertensive urgency, which is a severe increase in blood pressure (>180 mmHg systolic or >110 mmHg diastolic) not associated with target organ damage. This may be associated with headache, shortness of breath, or nosebleeds. The treatment is to lower the blood pressure within 24-48 hours and oral antihypertensive medications are recommended. Hospitalisation is not required. Amlodipine is a dihydropyridine calcium channel antagonist and a potent antihypertensive that can be used in this situation.

Intravenous antihypertensives like labetalol, glyceryltrinitrate are used in hypertensive emergencies which require lowering of blood pressure within minutes to hours e.g hypertensive encephalopathy, aortic dissection. Sublingual nifedipine is not recommended for treatment of hypertensive crises.

Question 116 of 129

A 72-year-old female presents with a 6-month history of gradual onset, progressive exertional dyspnoea associated bilateral increased lower limb pitting oedema. She has no previous cardiac history and prior to 6 months ago, had no limitations to exercise tolerance. Her past medical history includes B cell lymphoma diagnosed four years ago and in remission one course of chemotherapy. On examination, the jugular venous pulse is raised at 6cm above the angle of Louis. A soft systolic murmur and bibasal inspiratory crackles can be heard on auscultation. Abdominal examination demonstrates a pulsatile 3cm liver edge. Her blood pressure is 125/77 mmHg, heart rate 68/min and sinus, saturations 92% on 2l, respiratory rate 22/min. ECG reveals sinus rhythm of low voltage. Chest radiography demonstrates bibasal alveolar shadowing with prominent upper lobe vasculature.

Urine dip is as follows: blood -ve, leucocytes -ve, nitrates -ve protein 3+ pH 5.5.

Blood tests are as follows:

Hb	111 g/l
Platelets	275 * 10 /l
WBC	8.3 * 10 /l
Na	139 mmol/l
K	4.9 mmol/l
Urea	7.4 mmol/l
Creatinine	140 µmol/l
CRP	3 mg/l
Trop	<0.03 (normal range < 0.03)
Bilirubin	6 µmol/l
ALP	32 u/l
ALT	260 u/l

A transthoracic echocardiogram is performed, reported as significant increase in echogenicity, symmetrical left ventricular wall thickening, impaired bilateral diastolic ventricular dysfunction, bilaterally enlarged atria, pulmonary artery systolic pressure 45 mmHg, a 5mm pericardial effusion

What is the most likely underlying diagnosis for this patient?

A. Amyloid cardiomyopathy
B. Non-ST elevation myocardial infarction (NSTEMI)
C. Idiopathic dilated cardiomyopathy
D. Chemotherapy induced cardiomyopathy
E. Viral pericarditis leading to tamponade

A. Amyloid cardiomyopathy

A clinical picture of decompensated congestive cardiac failure is described with subtle details of hepatomegaly (possible congested, possibly related to the underlying diagnosis), associated heavy proteinuria, echogenic TTE and low voltage criteria ECG, on a background of previous B-cell lymphoma. The unifying diagnosis is amyloid cardiomyopathy, likely secondary to AL amyloid from B-cell dyscrasia, with cardiac, renal and possibly liver involvement. There is no evidence of a sudden onset cardiac event suggestive of an ischaemic cause. Echocardiogram does not demonstrate ventricular dilation, only impaired function. A 5mm pericardial effusion is not sufficiently significant to cause tamponade. Although chemotherapy-induced cardiomyopathy is possible, it is difficult to tie in the delayed onset from the end of treatment with renal involvement. It also does not explain the echogenic pictures on transthoracic echo.

Question 117 of 129

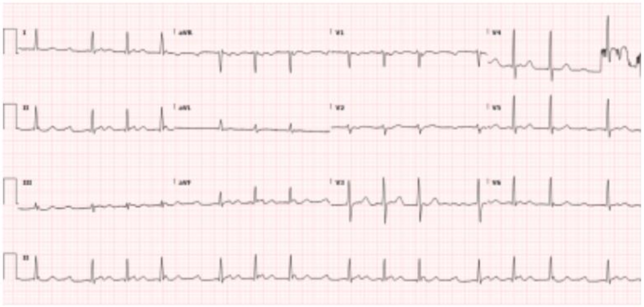
A 68-year-old man presents with occasional lightheadedness over the past few weeks. During his morning walk today, he experienced a brief spell of dizziness. He denies experiencing chest pain, breathlessness, or any other significant complaints.

His medical history includes diabetes, hypertension, and myocardial infarction for which he underwent coronary artery bypass grafting (CABG) seven years ago. He is currently on medications including aspirin, bisoprolol, lisinopril, atorvastatin, and vildagliptin.

Observations are as below:

- BP 140/85 mmHg
- Heart rate 74 bpm
- Respiratory rate 16/min
- Oxygen saturation 98% on room air

An ECG was subsequently carried out.



What is the most appropriate management option?

A. Discharge him and recommend a follow up ECG
B. Give atropine
C. Intravenous infusion of isoproterenol
D. Refer for cardiac resynchronisation therapy
E. Review his medications

E. Review his medications

The correct answer is **review his medications**.

The ECG findings demonstrate a progressive prolongation of the PR interval until a beat is dropped. This is characteristic of a 2nd-degree Mobitz 1 or Wenckebach-type block. This is usually a relatively benign rhythm with a low risk of progression to higher-degree blocks. This patient has symptoms of lightheadedness and dizziness, coupled with stable vital signs. As long as there is no hemodynamic compromise, there is no need for urgent intervention. Searching for a reversible cause should be the first step. This patient is on bisoprolol, a known cause of AVN block, and should be reviewed.

Discharge him and recommend a follow up ECG is incorrect. It is indicated if the patient is asymptomatic with a first or 2nd-degree Mobitz 1 heart block. This patient has symptoms of light-headedness and dizziness, which require intervention.

Give atropine is incorrect. Atropine is generally indicated for acute symptomatic AVN block with bradycardia and adverse features (heart failure, shock, syncope, myocardial ischaemia). There are no adverse features in this case.

Intravenous infusion of isoproterenol is also an incorrect answer. Reviewing any potential causes should be the initial approach for symptomatic hemodynamically stable patients. IV isoproterenol is indicated for hemodynamically unstable patients who fail to respond to transcutaneous pacing. Given the patient's history of coronary artery disease, it is important to avoid it if possible, due to the potential development of ischemic chest pain.

Refer for cardiac resynchronisation therapy is incorrect. Cardiac resynchronisation can be beneficial if there is heart failure with an ejection fraction of less than or equal to 35% and a widened QRS (e.g. left bundle branch block) complex on the ECG. It has no role in the management of the AVN block.

Question 118 of 129

A 23-year-old man presents with 2 days of chest pain to the emergency department. The pain is moderate and made worse if he breathes in. He is usually well but did have a few days off work for 'flu' last week.

On examination, he is sitting forward and groans in pain when asked to lie flat. His heart sounds are normal and his chest is clear. He is tender in his trapezius and scapular muscles but able to flex his neck .

Na +	135 mmol/l
K +	4.9 mmol/l
Urea	4.2 mmol/l
Creatinine	86 µmol/l
Hb	130 g/l
Platelets	354 * 10 /l9
WBC	4.5 * 10 /l9
Lymphocytes	0.1* 10 /l9
Chest X-ray	clear lung fields, normal heart contour

What ECG finding would be most specific in the likely diagnosis?

A. S1Q3T3
B. ST elevation
C. Sinus arrhythmia
D. Sinus tachycardia
E. PR depression

E. PR depression

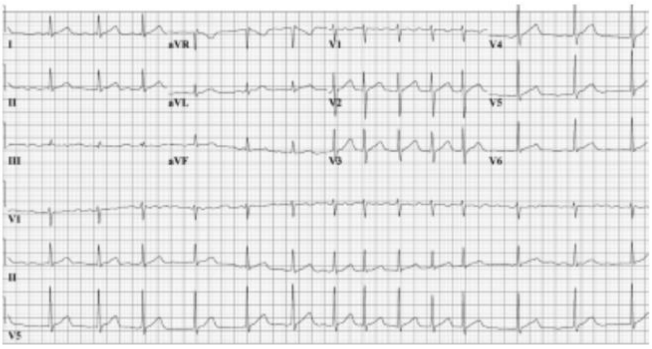
The most specific ECG finding in acute pericarditis is PR depression

The chest pain that is worse on lying flat in a person with a recent viral illness is due to pericarditis. ST elevation and PR depression are seen with the latter being the most specific. Sinus tachycardia and S1Q3T3 pattern are more commonly seen in pulmonary embolism. Sinus arrhythmia is a normal phenomenon in which the heart rate varies with the respiratory cycle.

Question 119 of 129

A 68-year-old man presents to hospital with left-sided upper and lower limb weakness that was present on waking that morning. The symptoms continue to persist in hospital. On examination, he has homonymous hemianopia. He has no significant medical history and is not on any regular medications.

A CT head is performed which is unremarkable. An ECG is performed, which is shown below.



How would you manage this patient's anti-thrombotic medication?

A. Apixaban for life
B. Aspirin 300mg once daily for 14 days, then apixaban for life
C. Aspirin 300mg once daily for 14 days, then clopidogrel 75mgonce daily for life
D. Clopidogrel 75mg once daily for life
E. Thrombolysis with alteplase, then apixaban for life

B. Aspirin 300mg once daily for 14 days, then apixaban for life

Aspirin 300mg once daily for 14 days, then apixaban for life is the correct answer. This patient has symptoms in keeping with a partial anterior circulation stroke. The CT head excludes an intracranial haemorrhage. Note, a normal scan is not sufficient to exclude a stroke, and stroke diagnosis should be based on clinical signs. The ECG shows atrial fibrillation which is the likely cause of the stroke. His CHA DS - VASc score is 3 due to age (+1) and stroke (+2). As such, he should receive two weeks of single anti-platelet treatment (aspirin 300mg once daily) as with all ischaemic strokes, followed by lifelong anticoagulation with a direct-acting oral anticoagulant (DOAC) such as apixaban, to prevent further strokes secondary to atrial fibrillation. Initiation of apixaban is delayed by two weeks post-stroke to prevent haemorrhagic transformation.

Apixaban for life is the incorrect answer. Therapeutic anticoagulation with a DOAC should not be initiated until after two weeks to avoid hemorrhagic transformation of an ischaemic stroke.

Aspirin 300mg once daily for 14 days, then clopidogrel 75mg once daily for life is the incorrect answer. This would be the correct answer if this patient did not have atrial fibrillation. Clopidogrel is insufficiently anti-thrombotic to prevent strokes secondary to atrial fibrillation and therefore a lifelong DOAC is required.

Clopidogrel 75mg once daily for life is the incorrect answer. A higher dose of antiplatelet is required to initially treat an ischaemic stroke (aspirin 300mg) and a DOAC is required long-term to reduce the atrial fibrillation-associated stroke risk.

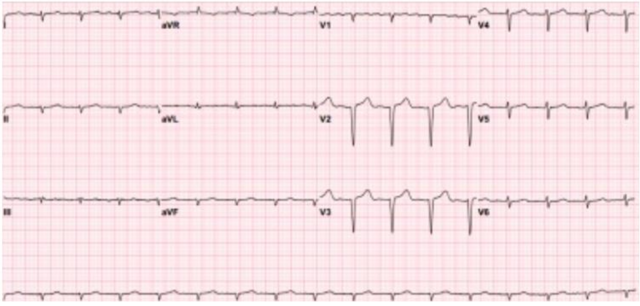
Thrombolysis with alteplase, then apixaban for life is the incorrect answer. This patient does not meet the criteria for thrombolysis as he awoke with the symptoms - thrombolysis requires symptom onset within the past four hours.

Question 120 of 129

An 81-year-old man is referred to the cardiology clinic due to worsening heart failure. Over the past 12 months he has become increasingly shortof-breath, particularly upon exertion or when lying flat. He has also suffered from ankle swelling which his GP has treated with large doses of furosemide. Around 6 months ago a clinical diagnosis of heart failure was made and he is already taking bisoprolol, ramipril and atorvastatin. He was diagnosed with multiple myeloma around 3 years and has been treated with melphalan to date.

On examination his pulse is 72/min and blood pressure 98/62 mmHg. The JVP is elevated at 6cm above the angle of Louis. There are crackles in both bases and pitting oedema to his knees.

An ECG accompanies the referral:



What is the most likely diagnosis?

A. Constrictive pericarditis
B. Infective endocarditis
C. Chronic pulmonary embolism
D. Cardiac amyloidosis
E. Hypercalcaemia-induced heart failure

D. Cardiac amyloidosis

This patient is likely to have AL amyloidosis secondary to his myeloma. This has resulted in cardiac amyloidosis as evidenced by the low voltage QRS complexes asssociated with poor R wave progression in the chest leads (a pseudoinfarction pattern).

Question 121 of 129

A 52-year-old lady was admitted with sudden onset central chest pain and breathlessness. Her past medical history included hypertension, atrial fibrillation and she was a smoker of 20 cigarettes a day. She lived alone after having recently split up with her husband which had been a difficult few weeks. Her friend had been staying with her occasionally during this difficult period.

She had a family history of ischaemic heart disease. She had recently complained to her friend of a cold that was now passing. Medications included aspirin 75mg OD and bisoprolol 5mg OD. Her friend called 999 and she was admitted directly to the angiogram suite as the paramedics noticed that her ECG showed ST elevation in the anterior chest leads.

Her angiogram showed mild coronary atherosclerosis but an akinetic left ventricle. Her troponin T was significantly elevated at 7800ng/L (normal < 14).

What is the most likely diagnosis?

A. Pericarditis
B. Myocardial infarction
C. Embolic event
D. Takotsubo cardiomyopathy
E. Pneumothorax

D. Takotsubo cardiomyopathy

Takotsubo cardiomyopathy (also known as broken heart syndrome, apical ballooning syndrome, acute stress cardiomyopathy) is nonischaemic cardiomyopathy where there is a temporary weakening of the heart muscle. This is often triggered by emotional stress or physical stress. Takotsubo cardiomyopathy is more commonly seen in postmenopausal women Patients most commonly present with chest pain and shortness of breath. Sudden onset congestive cardiac failure may also occur.

On initial presentation diagnosis of Takotsubo cardiomyopathy may be difficult and patients are often treated as an acute myocardial infarction. ECGs classically mimics ST-segment elevation myocardial infarction. Patients often proceed acutely to an angiogram which shows the absence of significant heart disease. Cardiac enzymes are significantly raised.

An echocardiogram may demonstrate pathognomonic wall motion abnormalities, in which the base of the left ventricle is contracting normally or is hyperkinetic while the remainder of the left ventricle is akinetic or dyskinetic. This coupled with the lack of significant coronary artery disease that would explain the wall motion abnormalities allows the diagnosis to be made.

Treatment of Takotsubo cardiomyopathy is generally supportive. Patients may be hypotensive, however, treatment with inotropes often worsens the disease. Patients may require intra-aortic balloon pump, fluids, beta blockers or calcium channel blockers. Aspirin is also commonly used in the management. Once over the initial event outcomes are favourable. Even when ventricular systolic function is heavily compromised at presentation, it typically improves within the first few days and normalises within the first few months

Question 122 of 129

A 71-year-old gentleman is reviewed in hospital prior to discharge after having received treatment for an exacerbation of COPD. His systolic blood pressure has been recorded as measuring between 138mmHg to 156mmHg. He has a background of ischaemic heart disease, gout, gallstones and a fractured neck of femur which was repaired with a dynamic hip screw. He is found to have ACR of 31mg/mmol. He normally takes allopurinol, aspirin, tiotropium, Symbicort, salbutamol.

What is the most appropriate pharmacological management for his hypertension?

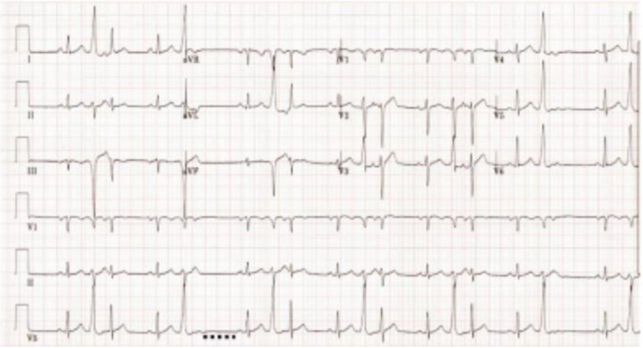
A. ACE-inhibitor
B. Calcium channel blocker
C. Thiazide-like diuretic
D. Beta-blocker
E. Alpha-blocker

A. ACE-inhibitor

The correct answer is ACE-inhibitor. This is an elderly man with hypertension and proteinuria. A calcium channel blocker would have been appropriate if he did not have proteinuria, whilst a thiazide-like diuretic and alpha-blocker feature at later stages in the NICE guidelines. Beta-blockers can provoke bronchospasms in COPD.

Question 123 of 129

A 58-year-old woman presents to the emergency department complaining of palpitations for half an hour. She has been diagnosed with diabetes, heart failure and depression and is on metformin, dapagliflozin, losartan, digoxin and sertraline. She reports experiencing abdominal cramps and loose stools over the last two days, which began following dinner at a restaurant. Her heart rate is 103 beats per minute, her BP is 100/70 mmHg, and respiratory rate is 20 breaths/min. An ECG was taken:



What is the most likely cause of the findings on the ECG?

A. Anxiety
B. Dapagliflozin
C. Digoxin
D. Metformin
E. Sertraline

C. Digoxin

The correct answer is **digoxin**. The electrocardiogram (ECG) presented demonstrates premature ventricular complexes (PVCs), which are characterised by abnormally large QRS complexes that disrupt the underlying sinus rhythm. A typical PVC is identifiable on an ECG as a broad QRS complex, exceeding 120 milliseconds in duration, with an abnormal morphology. It presents prematurely and is associated with discordant ST segment and T wave changes, followed by a compensatory pause prior to the next normal sinus impulse. Digoxin toxicity is recognised for inducing a spectrum of arrhythmias that may be lifethreatening. Arrhythmias associated with digoxin toxicity include paroxysmal atrial tachycardia, atrioventricular (AV) blocks, junctional rhythms, and ventricular arrhythmias such as PVCs and ventricular tachycardia. Notably, PVCs are often the initial indicator of digoxin toxicity and represent the most frequent arrhythmia linked to elevated serum digoxin levels. In this case, it is probable that hypokalaemia induced by diarrhoea has precipitated the digoxin toxicity.

Anxiety can lead to the emergence of PVCs; however, it is not deemed the primary cause in this clinical scenario. Anxiety-related PVCs are thought to originate from sympathetic nervous system overactivity. Nonetheless, given the circumstances described above, digoxin toxicity remains a more plausible explanation for this patient's presentation.

Metformin is not associated with arrhythmias when used within therapeutic dosages. Metformin toxicity might arise in contexts such as renal impairment or intentional overdose and could result in severe lactic acidosis that may precipitate arrhythmias. Despite this possibility, hypokalaemia secondary to diarrhoea resulting in digoxin toxicity appears to be a more convincing cause for the observed PVCs.

Sertraline, an selective serotonin reuptake inhibitor (SSRI), is utilised primarily in managing depression and does not align with this clinical picture as a causative agent for arrhythmia. Although sertraline is among the preferred antidepressants following myocardial infarction (MI), there exists no direct association between sertraline use and arrhythmic events. It should be noted that certain psychiatric medications such as clozapine, chlorpromazine and thioridazine have been implicated in arrhythmic side effects.

Dapagliflozin stands incorrect for this question. As an SGLT-2 inhibitor prescribed for heart failure and diabetes management, dapagliflozin has been reported to reduce incidents of significant ventricular arrhythmias including cardiac arrest or sudden death; thus it would be improbable for dapagliflozin to account for the clinical findings described here.

Question 124 of 129

An 86-year-old female presents to the diabetic ulcer clinic for regular foot care when regular observations note her heart rate to be 42 beats per minute. Her blood pressure is 142/55 mmHg and she reports no recent episodes of syncope. A 12 lead ECG demonstrates Mobitz type 1 rhythm at 42 beats/ minute. A 24 hours tape performed as an outpatient demonstrates bradycardia with up to 2.7 second pauses. Her past medical history includes type 2 diabetes mellitus, hypertension and angina. Her medications include ramipril, furosemide, verapamil, metformin and GTN on an as-required basis. During this second encounter, her heart rate is 41 beats/min and her blood pressure is 115/ 57 mmHg. She informs you that she is guided by you in terms of the most appropriate treatment.

What do you advise?

A. Temporary pacing wire
B. Permanent pacemaker insertion
C. Stop verapamil
D. Regular digoxin
E. Stop furosemide

C. Stop verapamil

The question focuses on the management of bradycardia and indications for pacemaker insertion. The patient is asymptomatic and sustains a reasonable blood pressure, there is no indication for a temporary pacing wire. Digoxin would only further the atrioventricular node block. Stopping furosemide is unlikely to affect the heart rate. Indications for pacemaker insertion is dependent on whether the patient has recently suffered a myocardial infarction. For patients post-MI, complete heart block, Mobitz type II, trifascicular block, symptomatic bradycardia or bilateral bundle branch block. For a patient not post-MI, symptomatic second and third-degree heart block or with haemodynamic compromise or VT secondary to bradycardia are indications. In this case, the patient is not post-MI and asymptomatic with no haemodynamic compromise. There are no indications for a pacemaker, stopping verapamil, an AV node blocker should be sufficient.

Question 125 of 129

A 50-year-old gentleman presented feeling dizzy and generally unwell for the last 3 days. He denied any chest pain, cough or fever but complained of increased fatigue and breathlessness. His past medical history included ischaemic heart disease, heart transplant, hypertension and diabetes. He takes the following medications: ramipril 5mg od, metformin 500mg bd, simvastatin 40mg od, amlodipine 5mg od and various immunosuppressive agents.

On examination his heart sounds were normal and chest was clear. There was only mild leg oedema. His vital signs show heart rate = 35 beats per minute, blood pressure = 90/40, SaO2 = 95% on air, T= 36.5ºC and respiratory rate = 20 breaths per minute. His ECG shows sinus bradycardia and CXR is clear. The following blood tests were obtained:

Hb	10.5 g/dl
Platelets	190 * 10 /l9
WBC	10.4 * 10 /l9
Na +	135 mmol/l
K +	3.6 mmol/l
Urea	8 mmol/l
Creatinine	100 µmol/l
CRP	15 mg/l

What is the next best management plan?

A. Glucagon intravenous
B. Atropine intravenous
C. Theophyline intravenous
D. Adrenaline 0.5mg intravenous
E. Adrenaline 0.5mg intramuscular

C. Theophyline intravenous

This question aims to raise awareness about the treatment of bradycardia and some potential pitfalls.

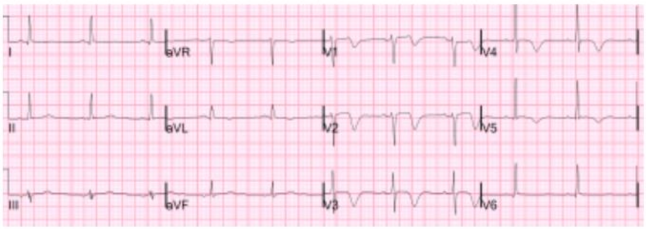
According to ALS protocol any bradycardia has to be assessed according to the ABCDE principle. In the presence of shock, syncope, myocardial ischaemia or heart failure further treatment is needed as soon as possible. Atropine is usually the first line treatment for bradycardia and if that fails then consider transcutaneous or transvenous pacing. However, in patients with heart transplant atropine is contraindicated. The hearts of these patients are denervated and do not respond to vagal blockade by atropine, which might precipitate paradoxical sinus arrest of highgrade block. In these cases, theophyline intravenously is indicated.

Glucagon is indicated if the bradycardia is due to beta-blockers or calcium channel blockers. Adrenaline is a potential option but the dose mentioned is wrong and certainly use of intravenous adrenaline outside the cardiac arrest scenario requires expert help (intramuscular in the anaphylaxis scenario.

Question 126 of 129

A 55-year-old man presents to the emergency department with a 2-hour history of central chest pain and dizziness. He has tried taking paracetamol and ibuprofen but has seen little improvement. His past medical history includes hypertension, for which he takes amlodipine 10mg once daily. He smokes 20 cigarettes/day and drinks 12 units of alcohol per week.

An ECG is taken (as shown below):



What is the most likely diagnosis?

A. Brugada syndrome
B. Hypertrophic obstructive cardiomyopathy
C. Posterior myocardial infarction
D. Pulmonary embolus with right heart strain
E. Wellen's syndrome

E. Wellen's syndrome

This patient has **Wellen's syndrome**. Wellen's syndrome is the critical ischaemia of the left anterior descending artery. Patients typically have a history of chest pain and ECG findings include biphasic T waves in the anterior leads or deep symmetrical T wave inversion in leads I and aVL associated with 1mm ST elevation in the chest leads. These can be seen in this patient's ECG.

Brugada syndrome is incorrect. Brugada syndrome is an autosomal dominant inherited cardiovascular disease. It can be asymptomatic and lead to sudden cardiac death. ECG changes consistent with Brugada syndrome include convex ST-segment elevation > 2mm in at least 1 of V1-V3 that is followed by T wave inversion. A partial right bundle branch block may also be seen. Sometimes, a patient may have an appearance of a normal electrocardiogram (ECG). However, following the administration of flecainide, these ST-segment changes may appear.

Hypertrophic obstructive cardiomyopathy (HOCM) is incorrect. HOCM is an autosomal dominant inherited condition resulting in left ventricular hypertrophy with decreased cardiac compliance and ultimately decreased cardiac output. Most cases can be asymptomatic. However, symptoms include exertional dyspnoea or chest pain, syncope and sudden cardiac death. Given the family history of sudden cardiac death, HOCM is a reasonable differential. However, ECG features of HOCM include features of left ventricular hypertrophy (eg increased R wave height), deep Q waves and non-specific ST segment changes that are not seen in this patient's ECG.

Posterior myocardial infarction is incorrect. A posterior myocardial infarction causes ECG findings such as a dominant R wave in V2, and horizontal ST depression and broad R waves in V1-V3. Although the patient has cardiac-sounding chest pain, their ECG findings do not match those that we see here, making this answer incorrect.

Pulmonary embolus with right heart strain is incorrect. Classic findings of a pulmonary embolus include sinus tachycardia and the less common S1Q3T3 finding. However, in more significant disease, patients may exhibit features of **right heart strain** secondary to pulmonary hypertension. Right heart strain appears as ischaemic changes (including deep T wave inversion) in the right-sided leads of the heart. The history here is not suggestive of a pulmonary emboli and does not explain the biphasic T waves seen in V1 and V2.

Question 127 of 129

A 48-year-old man is brought in by ambulance to the Emergency Department after experiencing sudden onset crushing central chest pain radiating down his arm. His ECG showed an inferior STEMI and he undergoes successful PCI with 2 stents inserted into his right coronary artery. He is transferred to CCU and started on secondary prevention medication.

24 hours after his presentation he becomes bradycardic at 34bpm and his blood pressure falls to 80/47 mmHg. He denies chest pain but feels dizzy and light headed. His cardiac monitor shows 3rd-degree heart block. He is given atropine but his heart rate does not raise and his blood pressure remains low.

What is the most appropriate next step in his management?

A. Isoprenaline infusion
B. Temporary pacing wire
C. Permanent pacemaker
D. Repeat angiography
E. Adrenaline

B. Temporary pacing wire

This gentleman has suffered an inferior STEMI. Complete heart block is a recognised complication of both inferior and anterior STEMI. Inferior STEMI CHB is usually more transient and they only require treatment if they become symptomatic. Anterior STEMI CHB is a more serious complication and they are more likely to need a permanent pacing system. In this case, as he has not responded to atropine then isoprenaline infusion is unlikely to work. There is no suggestion of repeat infarction so angiography is not indicated. He will require a temporary pacing wire to be placed.

Question 128 of 129

A 70-year-old woman with a history of type 2 diabetes mellitus and hypertension is reviewed in clinic. You can see from the records their is no evidence of diabetic retinopathy, chronic kidney disease or cardiovascular disease.

Her current medication is as follows:

- simvastatin 40mg on
- ramipril 10mg od
- amlodipine 5mg od
- metformin 1g bd

Recent blood results are shown below:

Na +	142 mmol/l
K +	4.4 mmol/l
Urea	7.2 mmol/l
Creatinine	86 µmol/l
HbA1c	45 mmol/mol (6.3%)

Urine dipstick shows no proteinuria. Her blood pressure today in clinic is 134/76 mmHg.

What is the most appropriate course of action?

A. Add gliclazide
B. Increase amlodipine
C. Increase ramipril
D. Add losartan
E. No changes to medication required

E. No changes to medication required

Newly diagnosed patient of black African or African-Caribbean origin with hypertension - add a calcium channel blocker

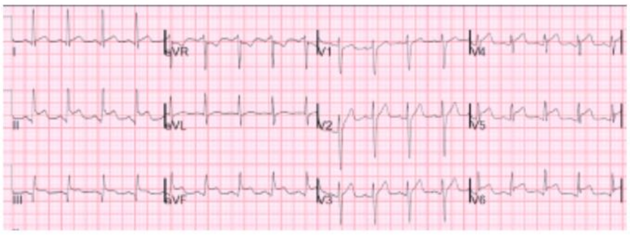
Her diabetic control is good - NICE do not advocate changing treatment at this stage unless the HbA1c is >= 6.5%.

As she has no complications from her diabetes the blood pressure target is < 140/80 mmHg. No changes are therefore required to her antihypertensive regime.

Question 129 of 129

A 45-year-old man is referred to the ambulatory care team with a 4-day history of left-sided chest pain and dyspnoea. He describes the chest pain as feeling sharp in nature and is associated with occasional shortness of breath and palpitations, particularly on exertion. He reports that his daughter has just recovered from scarlet fever and he has also been affected by coryzal symptoms over the past 2 weeks. His past medical history includes hypertension for which he takes ramipril 10mg OD.

An ECG is taken, as shown below:



Given the likely diagnosis, what is the most appropriate management?

- A. Calcium gluconate
- B. Colchicine
- C. Dual antiplatelet therapy
- D. Intravenous broad-spectrum antibiotics
- E. Simple analgesia and reassurance

B. Colchicine

Colchicine is correct. This patient has a likely diagnosis of pericarditis given the history of pleuritic chest pain after a recent coryzal illness. This patient's ECG shows widespread ST elevation with PR depression, both of which are characteristic signs of pericarditis. The treatment of pericarditis is with NSAIDs and colchicine, particularly for idiopathic or viral pericarditis.

Calcium gluconate is incorrect. ECG findings consistent with hyperkalaemia include tall tented T waves, PR interval prolongation and QRS widening. It does not cause widespread ST elevation or PR depression, as shown here.

Dual antiplatelet therapy is incorrect. Although this patient's ECG does demonstrate ST elevation, the ST elevation is widespread and not specific to one coronary artery territory. Furthermore, the nature of the ST depression is concave and is associated with PR depression, making pericarditis much more likely.

Intravenous broad-spectrum antibiotics is incorrect. This would be indicated for infective endocarditis which is a condition that typically presents with fevers, constitutional symptoms (e.g. malaise, weight loss, night sweats) and fatigue as the predominant features, in contrast to pleuritic chest pain that is being described here. Infective endocarditis does not explain this patient's ECG findings.

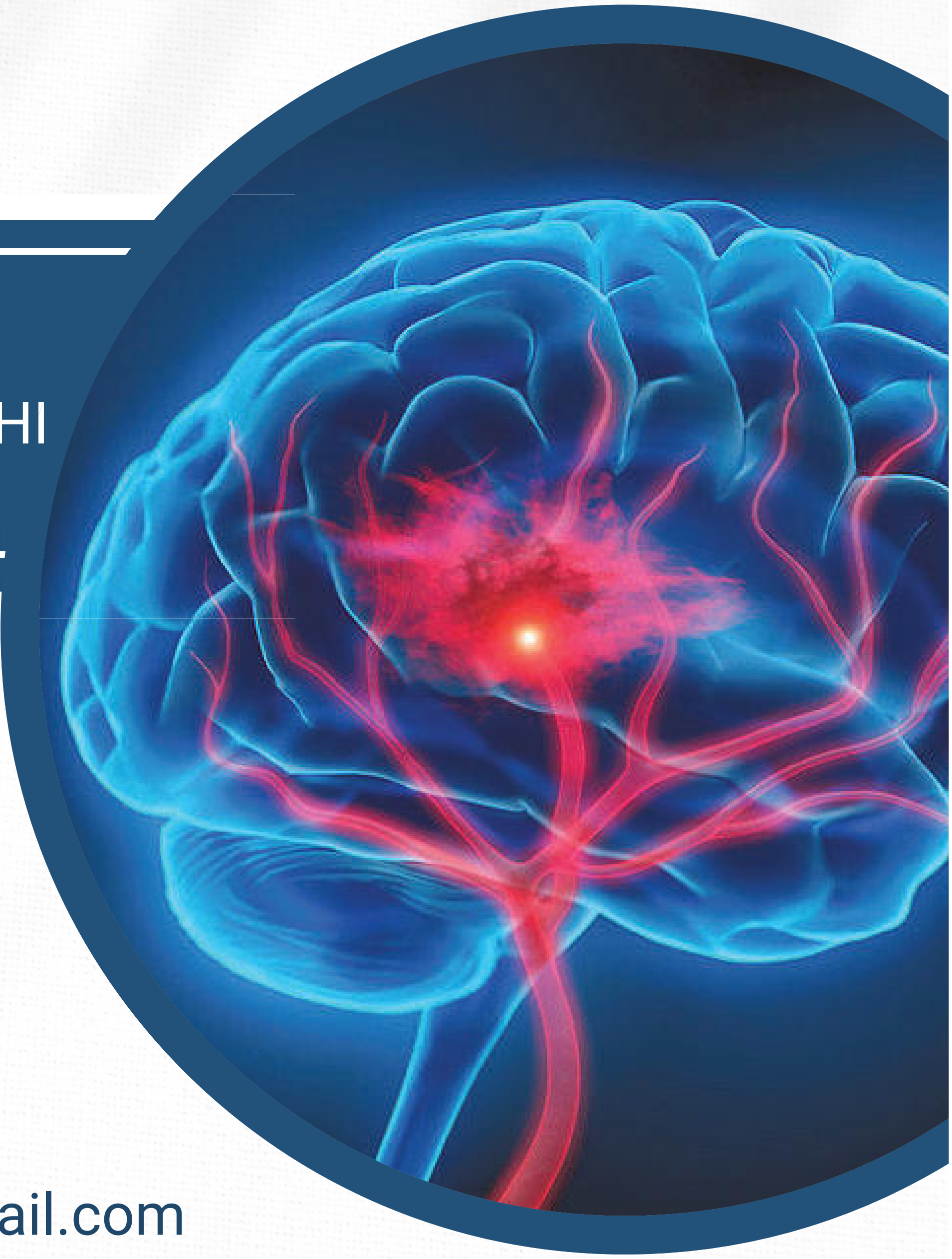
Simple analgesia and reassurance is incorrect. This would be indicated for musculoskeletal chest pain or perhaps even costochondritis which is characterised by pleuritic chest pain that may follow a recent viral illness. However, unlike pericarditis, costochondritis is the inflammation of the costochondral joints and does not produce ECG changes, as seen in this presentation.

Alrajhi Jad

PASSMEDICINE

NEUROLOGY, PSYCHIATRY AND OPHTHALMOLOGY

Internal medicine



EDITED BY
DR. JAMIL ALRAJHI

2024

773-555-493
jameelalrajhi@gmail.com

PASSMEDICINE PART 2 NEUROLOGY OPHTHALMOLOGY, and PSYCHIATRY 2024

Question 1 of 409

A 68-year-old man is admitted to the unit with an exacerbation of severe pain around his abdomen. He describes it as a tight band like pain which is burning in character. He has a past medical history of metastatic prostate cancer. His drug history includes morphine modified release (100mg, twice daily), duloxetine (60mg, twice daily), pregabalin (200mg, three times per day). He states that the pain initially responded well to the analgesics he is on however over the past few weeks it has got progressively worse and culminated in a severe attack of pain today.

After discussion with the palliative care consultant, you elect to start the patient on an NMDA receptor antagonist.

What medication will you start?

A. Fentanyl
B. Codeine
C. Paracetamol
D. Gabapentin
E. Ketamin

E. Ketamin

Ketamine can be used as a third line agent in the management of neuropathic pain which is poorly responsive to titrated opioids and oral adjuvant analgesics

The patient describes classic radicular pain associated with thoracic radiculopathy. It is described as a tight band like pain which may be burning, tingling, or shooting in character. He is on a large dose of opioids and two neuropathic agents which act on different pathways.

He is already on a strong opioid so it would be inappropriate to use a middle strength opioid (e.g. codeine, dihydrocodeine, or buprenorphine). Fentanyl would only be used in stable pain, or with specific other indications such as renal disease. In this case it is unlikely to control his pain.

He is already on two neuropathic agents so it would be inappropriate to start gabapentin.

Ketamine can be used for neuropathic pain poorly responsive to titrated opioids and oral adjuvant analgesics (e.g. antidepressant and/or anticonvulsant) particularly when there is abnormal pain sensitivity (e.g. allodynia, hyperalgesia or hyperpathia). Ketamine is an NMDA receptor antagonists.Side effects to be aware of include hypertension, raised intracranial pressure and hallucinations.

Gabapentin was designed to mimic the neurotransmitter GABA. It does not, however, bind to GABA receptors. Its mechanism of action is to block voltage-gated calcium channels.

Question 2 of 409

A 34-year-old female presents to the clinic. She states that she sometimes has difficulties leaving the house due to fear of being in situations where she cannot escape. Some examples she gives includes being in busy places such as shopping malls and public transport. She states that when she he is at home and in her place of work she functions well, with regards to both productivity and interpersonal relationships.

What is the most likely diagnosis?

A. Panic disorder
B. Generalized anxiety disorder
C. Agoraphobia
D. Depression
E. Social phobia

C. Agoraphobia

Agoraphobia describes a fear of open spaces. It may or may not be associated with panic disorder

The clinical features are suggestive of agoraphobia which is described as fear of open spaces and being trapped in situations which are difficult to escape from.

Agoraphobia may or may not be associated with panic disorder. However, there is no evidence that this patient is having panic attacks.

Generalized anxiety disorder describes a syndrome where patients feel anxiety in a range of different situations including work and personal life. Social phobia describes a condition in which patients have a fear of being in social situations e.g. a fear of embarrassing themselves. The patient functions well with others both at work and home suggesting an alternative diagnosis.

There is nothing to suggest that the patient is depressed.

Question 3 of 409

A 64-year-old man presents to the endocrinology clinic with a palpable neck mass. He has noticed this incidentally. He denies any problems in swallowing, speaking or breathing. On more detailed history he does admit to a 3kg weight loss over the last 4 months which he cannot explain but is happy with as he has always been overweight. He has a past medical history of appendicectomy, hypertension, high cholesterol. He takes aspirin and atorvastatin. Thyroid function tests are normal. On examination, he has a roughly 3cm mass in the anterior neck triangle on the left side. It is firm and non-tender.

What is the most appropriate next investigation?

A. CT neck
B. US neck

Question 6 of 409

A 37-year-old man presents to the emergency department with a six-hour history of an acutely painful right eye associated with photophobia and reduced visual acuity. He has had no previous episodes. He has no relevant past medical history and occasionally takes codeine for back pain.

On examination, diffuse redness is noted throughout the right eye. The right pupil is smaller than the left and he is unable to count fingers in the right eye although visual acuity in the left eye is unimpaired.

What is the most likely diagnosis?

A. Acute angle closure glaucoma
B. Anterior uveitis
C. Bacterial conjunctivitis
D. Cluster headache
E. Scleritis

B. Anterior uveitis

Anterior uveitis presents with acutely painful red eye, photophobia, small pupil, reduced visual acuity. It is often associated with pus in the anterior chamber (a hypopyon)

This man has presented with a case of anterior uveitis, as confirmed by the typical findings on examination. Note that anterior uveitis can be both unilateral or bilateral, largely dependant on the cause. Although this condition is associated with a hypopyon, this is only sometimes present. A further clue is the presence of back pain in a young man which points towards a history of ankylosing spondylitis, a condition closely associated with anterior uveitis.

Acute angle-closure glaucoma does present with an acutely painful red eye however is commonly associated with a headache, nausea, and vomiting, and seeing halos. It is also more likely in the older population and particularly people with a history of presbyopia or chronic open-angle glaucoma.

Bacterial conjunctivitis does cause redness but is usually associated with itching and purulent discharge. Additionally, it is unlikely to be associated with the additional features this man has exhibited (miosis, photosensitivity, and reduced visual acuity).

Scleritis again is another cause of an acutely painful red eye however is unlikely to cause photophobia and miosis. Although scleritis is related to autoimmune conditions (such as systemic lupus erythematosus and rheumatoid arthritis), it is less associated with ankylosing spondylitis.

A cluster headache causes pain behind the eye and can be associated with redness and watering of the eye as well as miosis however attacks are more likely to be frequent and short-lived rather than persisting for six hours.

Question 7 of 409

A 20-year-old woman presents to a general neurology clinic. Over the last year, she has noted that she is becoming increasingly clumsy with heavy objects. She describes struggling to wash her hair and complete certain house hold chores such as hanging washing up.

Neurological examination findings of the upper limbs have been summarised below. Power has been recorded as per the Medical Research Council (MRC) grading system.

	Left arm	**Right arm**
Tone	normal	normal
Power:		
shoulder abduction	3/5	3/5
shoulder adduction	4/5	4/5
elbow flexion	4/5	5/5
elbow extension	5/5	5/5
wrist flexion	5/5	5/5
wrist extension	5/5	5/5
Reflexes:		
Biceps	normal	normal
Triceps	normal	normal
Supinator	normal	unable to elicit
Sensation	intact	intact

When examining her cranial nerves it is noted there is drooping of her eyelids and decreased facial expression. Tests for fatigability were unremarkable. There were no other abnormal findings.

What is the most likely diagnosis?

A. Myasthenia gravis
B. Facioscapulohumeral dystrophy
C. Multiple sclerosis
D. Miller-Fisher syndrome
E. Becker's muscular dystrophy

B. Facioscapulohumeral dystrophy

Facioscapulohumeral muscular dystrophy: facial weakness (e.g. difficulty closing eyes, smiling, blowing), winged scapula and weakness of the shoulderm upper arm and hip girdle muscles

The history here is typical of facioscapulohumeral dystrophy, the diagnosis is confirmed by the examination findings which confirm a proximal weakness affecting the limb girdle and subtle signs of weakness in the face.

The absence of fatigability in muscle power excludes myasthenia gravis. The clinical picture is not of Miller-Fisher syndrome which often presents as a triad of ophthalmoplegia, ataxia, and areflexia. Becker's muscular dystrophy is excluded as it typically affects the limb girdles in males who carry the X-linked gene. The presentation is also atypical of MS.

Question 8 of 409

A 43-year-old woman presents to the emergency department with fevers and confusion. She has no past medical history and does not take any regular medications.

On examination, her Glasgow coma scale is 13/15 (E3V4M5). There is no localizing neurological signs. Cardiovascular, respiratory and abdominal assessment is unremarkable. Her temperature is 38.8ºC.

An urgent MRI brain is organized, which demonstrates oedema of both temporal lobes. A lumbar puncture is arranged.

What set of results is most likely to be found?

A. Normal white cell count / elevated protein
B. Normal white cell count / normal protein
C. Raised lymphocytes / elevated protein
D. Raised lymphocytes / normal protein
E. Raised neutrophils / elevated protein

A. Raised lymphocytes / elevated protein

Herpes simplex encephalitis - the CSF usually shows a lymphocytosis amd elevated protein

Raised lymphocytes / elevated protein is the correct answer. The patient presents with fever and confusion and is pyrexial with a low GCS on examination. In this context, the MRI findings of bilateral temporal lobe oedema are highly suggestive of herpes simplex encephalitis. A lumbar puncture would typically demonstrate lymphocytosis and elevated protein.

Normal white cell count / elevated protein is incorrect. The normal white cell count points away from infection. Protein content in the CSF is usually raised either due to either an increased permeability of the blood brain barrier or synthesis of proteins within the spinal canal due to inflammatory cells. The differential for this set of results is broad and includes numerous inflammatory causes. It is however, a less likely set of results in HSV encephalitis.Raised lymphocytes / normal protein is incorrect. This set of results may be compatible with a viral infection but in HSV encephalitis the protein is more likely raised than normal.

Raised neutrophils / elevated protein is incorrect. This set of results is more consistent with a bacterial infection.

Normal white cell count / normal protein is incorrect. This is a normal result and unlikely in HSV encephalitis.

Question 9 of 409

A 47-year-old woman was admitted from a rehabilitation unit after appearing unwell. She has a past medical history of a road traffic accident 1 year prior and spinal cord injury at the C5 level which has left her quadriplegic.

Her regular medications include laxido, codeine and paracetamol.

On examination from the end of the bed, there is marked flushing of the skin across the face and shoulders and she appears diaphoretic and extremely agitated. Her temperature is 37.6ºC, oxygen saturation 97% on air, heart rate 102 beats per minute, respiratory rate 21 breaths per minute and blood pressure 206/130 mmHg. Auscultation of the chest reveals good bilateral air entry and vesicular breath sounds. Her heart sounds are normal with no added murmurs. There is a bounding pulse peripherally and central capillary refill time of 2 seconds. There is no swelling of the lower legs and the abdomen is soft with a rounded supra-pubic mass. There is a long term catheter in-situ connected to an empty catheter bag.

Initial investigations are displayed below:

ECG	Sinus tachycardia

Hb	126 g/L	Male: (135-180) Female: (115 - 160)
Platelets	368 * 10 /L	(150 - 400)
WBC	9.3 * 10 /L	(4.0 - 11.0)
Na	141 mmol/L	(135 - 145)
K	4.3 mmol/L	(3.5 - 5.0)
Urea	5.3 mmol/L	(2.0 - 7.0)
Creatinine	84 µmol/L	(55 - 120)
CRP	4 mg/L	(< 5)
Troponin T	5ng/L	(<14)

Chest X-ray	no focal consolidation or collapse

What would be the next most appropriate management step given the likely diagnosis?

A. Treatment dose of low molecular weight heparin followed by a CTPA
B. IV co-amoxiclav and gentamicin
C. Antihypertensives
D. Abdominal X-ray
E. Antihypertensives and bladder scan

E. Antihypertensives and bladder scan

Management of autonomic dysreflexia involves removal/control of the stimulus and treatment of any life-threatening hypertension and/or bradycardia

Patients with a high cervical spinal cord injury are at risk of autonomic dysreflexia which can be distressing and potentially cause life-threatening hypertension. The management of autonomic dysreflexia would be to identify and remove the offending stimulus - in this case, a blocked catheter causing urinary retention - while treating extreme hypertension.

Although the patient is at risk of venous thromboembolism given her immobility. The clinical picture is more in keeping with autonomic dysreflexia secondary to urinary retention and therefore option 1 is incorrect.

The clinical picture and investigation results are not consistent with urinary sepsis and therefore option 2 is incorrect.

Antihypertensives alone do not address the underlying cause and therefore option 3 is incorrect.

An abdominal X-ray may be an appropriate further down the line if you are suspecting faecal impaction as a cause for the urinary retention or for the autonomic dysreflexia. In this case, the clinical picture is more suggestive of acute urinary retention due to a blocked catheter. To confirm and manage this retention would be the immediate priority and therefore option 4 is incorrect

Question 10 of 409

A 60-year-old female presents to the Emergency Department with altered sensation in both hands and reduced visual acuity in the left eye. She states that these symptoms have slowly worsened over a week. She has no issues with bladder or bowel activity.

On examination, she is haemodynamically stable. Of note, she has a sensory loss throughout her arms and legs, but no clear sensory level. Additionally, she has brisk reflexes in both the upper and lower limbs. She also has a visual acuity of 6/36 in the left eye, but normal vision in the right eye, with normal visual fields.

Her MRI scan reveals multiple continuous segments of inflamed spinal cord throughout the cervical region and left optic nerve inflammation. There are no cerebral lesions. Her cerebrospinal fluid (CSF) studies are negative for oligoclonal bands.

Which of the following tests will confirm the diagnosis?

A. dsDNA antibodies
B. Aquaporin-4 antibodies
C. ACh receptor antibodies
D. NMDA receptor antibodies
E. Neuronal antibodies

B. Aquaporin-4 antibodies

Neuromyelitis optica is associated with aquaporin 4 positive serum antibody

The description of a patient with optic neuritis and longitudinal extensive cervical transverse myelitis with a CSF negative for oligoclonal bands is highly suggestive of neuromyelitis optica (NMO). The classic antibody associated with this disease is NMO-IgG or antibodies against aquaporin-4.

The 2006 diagnostic criteria for NMO are as follows:

Transverse myelitis and optic neuritis with at least 2 of the following features:

- MRI brain negative/nondiagnostic for multiple sclerosis
- MRI spinal cord lesion extending over 3 vertebral segments (LETM)
- NMO-IgG/Aquaporin-4 antibody seropositivity.

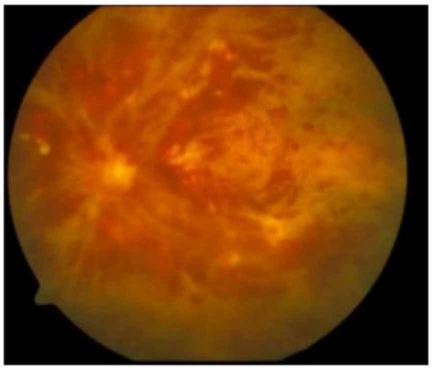
Question 11 of 409

A 62-year-old woman presents with sudden loss of vision in her left eye. Fundoscopy reveals the following:-

What is the diagnosis?

A. Retinal detachment
B. Ischaemic optic neuropathy
C. Vitreous haemorrhage
D. Central retinal vein occlusion
E. Central retinal artery occlusion

D. Central retinal vein occlusion



Central retinal vein occlusion - sudden painless loss of vision, severe retinal haemorrhages on fundoscopy

This appearance is sometimes compared to a cheese and tomato pizza.

Question 12 of 409

A 74-year-old man presents to the emergency department via ambulance with a sudden onset of speech difficulty, numbness and weakness. The symptoms started eight hours previously but he was initially reluctant to come to hospital due to fears regarding the coronavirus pandemic. He has a past medical history of hypertension, hypercholesterolemia and ischaemic heart disease. He is on regular aspirin, atorvastatin, bisoprolol and amlodipine. He does not smoke and drinks minimal alcohol. He is usually independent and lives with his wife.

His observations are heart rate 88 beats per minute, blood pressure 158/64 mmHg, respiratory rate 18/minute, oxygen saturations 96% on room air and temperature 37.1ºC.

On examination, an expressive dysphasia is noted in addition to right-sided hemisensory loss affecting his face, arm and leg. There is evidence of facial weakness and power is 3/5 in his right upper limb and 4/5 in his right lower limb. Chest auscultation is normal. Heart sounds are audible with no murmurs and his pulse is irregular. There are no carotid bruits.

Urinalysis is unremarkable and bedside testing demonstrates a glucose level of 7.1 mmol/L. An ECG demonstrates atrial fibrillation.

Urgent CT angiography demonstrates occlusion of the proximal anterior circulation.

Approximately one hour later, the patient undergoes a diffusion-weighted MRI that shows limited core infarct volume.

What is the most appropriate management?

A. Anticoagulation
B. High dose aspirin
C. Intravenous thrombolysis
D. Intravenous thrombolysis and thrombectomy
E. Thrombectomy

A. Thrombectomy

For thrombectomy in acute ischaemic stroke, an extended target time of 6-24 hours may be considered if there is the potential to salvage brain tissue, as shown by imaging such as CT perfusion or diffusion-weighted MRI sequences showing limited infarct core volume.

Anticoagulation is incorrect. This patient has had an ischaemic stroke likely secondary to new atrial fibrillation and will need anticoagulation in due course. However, anticoagulation is not a treatment for acute stroke and is usually delayed for two weeks after the initial event.

High dose aspirin is incorrect. This patient is outside the typical thrombolysis window of 4.5 hours but is already established on aspirin treatment and there is another more suitable option in this circumstance i.e. thrombectomy.

Intravenous thrombolysis is incorrect. This treatment needs to be administered with 4.5 hours of the onset of an ischaemic stroke. We are outside the thrombolysis window and therefore this is not an appropriate option.

Intravenous thrombolysis and thrombectomy is not the right answer. The combination of thrombectomy and intravenous thrombolysis is still time-limited by the thrombolysis window of 4.5 hours. The decision to offer thrombectomy alone is dependent on different criteria. This patient is outside the thrombolysis window.

Thrombectomy is the correct answer. An extended target time of 6-24 hours for thrombectomy alone may be considered if there is confirmed proximal occlusion of the anterior circulation by CT angiography and the potential to salvage brain tissue, as shown by imaging such as CT perfusion or diffusion-weighted MRI sequences showing limited infarct core volume. Our patient underwent a diffusion-weighted MRI demonstrating limited infarct core volume within 9 hours of symptom onset and has a confirmed proximal anterior circulation occlusion and therefore is an eligible candidate.

Question 13 of 409

A previously well 69 year old patient presents to the A+E department with a sudden onset of weakness. This was noticed by his wife who immediately called 999.

Neurological examination: CN I-IV normal

CN V ophthalmic and maxillary divisions normal with reduced sensation in mandibular division on left side

CN VI normal

CN VII reduced power to left lower facial musculature

CN VIII normal

CN IX, X and XII weakness in swallow

CN XI weakness on turning the head to the left.

Right upper limb normal tone, power 5/5, normal reflexes and sensation. Normal finger pointing. Left upper limb markedly increased tone, power 1/5 globally in all muscle groups, brisk reflexes and reduced sensation to light touch. Unable to move limb to determine ability to finger pointing.

Right lower limb normal tone, power 5/5, normal reflexes and sensation. Heel-knee-shin test normal.

Left lower limb slightly increased tone, power 3/5 globally, brisk reflexes and reduced sensations. Reduced ability to heel-knee-shin test when compared to right side.

Gait - not assessed due to weakness. Romberg's test - not done due to weakness.

Where is the lesion most likely to be?

A. Left middle cerebral artery
B. Right middle cerebral artery
C. Posterior cerebral artery
D. Left anterior cerebral artery
E. Right anterior cerebral artery

A. Right middle cerebral artery

Contralateral hemiparesis and sensory loss with the upper extremity being more affected than the lower, contralateral homonymous hemianopia and aphasia - middle cerebral artery

The understanding of stroke territories is important both in clinical practice but also in the MRCP! In terms of limb weakness it must be remembered that an ischaemic event in the anterior cerebral territory caused contralateral hemiparesis (leg>arm) and the middle cerebral territory causes contralateral hemiparesis (face/arm>leg).

A posterior cerebral artery lesion causes contralateral homonymous hemianopia with amnesia and sensory loss.

Question 14 of 409

A 56-year-old man presents to the neurology clinic with a 6-month history of loss of dexterity and new-onset dysphagia. The patient reports a history of hypertension for which he takes amlodipine 5mg once daily with no family history of note, he is a non-smoker and drinks 3 glasses of wine per week. Systems review reveals no history of rashes, joint pain, swelling or stiffness, no fevers, and no weight loss. A recent endoscopy showed no obvious abnormalities to explain the patient's dysphagia.

Physical examination demonstrates asymmetrical wasting of the deltoids with noticeable weakness of wrist flexion bilaterally (MRC grading 4/5).

Co-ordination is normal on the assessment of the upper and lower limbs, which decreased ankle jerk relaxes bilaterally. Plantars are downgoing bilaterally. Cranial nerve assessment is unremarkable, however, a bedside swallow assessment is suggestive of a delayed oropharyngeal phase.

Bloods tests ordered by the neurologist demonstrate:

Hb 140 g/L	Male: (135-180) Female: (115 - 160)
Platelets 220 * 10 /L	(150 - 400)
WBC 5.0 * 10 /L	(4.0 - 11.0)

Calcium 2.26 mmol/L	(2.1-2.6)
Phosphate 1.04 mmol/L	(0.8-1.4)

Magnesium 0.9 mmol/L	(0.7-1.0)
Thyroid stimulating hormone (TSH) 0.6 mU/L	(0.5-5.5)
Free thyroxine (T4) 10.1 pmol/L	(9.0 - 18)
Creatine kinase 1000 U/L	(35 - 250)

What is the most likely diagnosis?

- A. Inclusion body myositis
- B. Dermatomyositis
- C. Motor neuron disease
- D. Multiple sclerosis
- E. Myasthenia gravis

A. Inclusion body myositis

Older man + quadriceps and finger/wrist flexors weakness + raised CK → inclusion body myositis

Inclusion body myositis is characterized by frequently asymmetrical muscle weakness, with a mixed proximal and distal distribution of weakness. Dysphagia affects approximately 9% of patients at presentation.

Dermatomyositis usually features symmetrical muscle weakness with a characteristic rash. This patient has neither.

Motor neuron disease presents with upper motor neuron signs, with hyperreflexia and extensor plantars. This patient has neither.

Multiple sclerosis could possibly explain the patient's symptoms, however, it would be very unlikely in a male patient of late middle age.

Myasthenia gravis usually presents with symmetrical muscle weakness which is fatiguable. This patient has an asymmetrical weakness with no mention of fatiguability.

Question 15 of 409

A 24-year-old male presents to the acute medical unit with confusion. His mother states that over the past day he has been behaving very strangely. His past medical history includes epilepsy, asthma, and depression. His drug history includes salbutamol, sertraline, and sodium valproate. On examination, you note myoclonus and his Glasgow coma score is reduced at 13 (M6, V4, E3).

Blood results are as follows:

Hb	132 g/l	Na	142 mmol/l	Bilirubin	18 µmol/l
Platelets	328 * 10 /l	K	4.1 mmol/l	ALP	72 u/l
WBC	8.7 * 10 /l	Urea	6.8 mmol/l	ALT	184 u/l
Neuts	7.4 * 10 /l	Creatinine	72 µmol/l	INR	1.1
Lymphs	1.2 * 10 /l	Albumin	42 g/l	Plasma ammonia	72 µmol/L (normal range 11-32 µmol/L)

What is the most likely cause of this presentation?

- A. Hepatic encephalopathy
- B. Sertraline
- C. Small intestinal bacterial overgrowth
- D. Sodium valproate
- E. Intracerebral hemorrhage

D. Sodium valproate

Sodium valproate is a well known cause of hyperammonemic encephalopathy

The patient has features of encephalopathy and raised serum ammonia levels. Valproate-induced hyperammonaemic encephalopathy (VHE) is an unusual complication characterised by a decreasing level of consciousness, focal neurological deficits, cognitive slowing, vomiting, drowsiness, and lethargy. Asymptomatic hyperammonaemia occurs in 15-50% of patients on valproate.

Treatment with L-carnitine may be beneficial in reducing ammonia levels.Although the ALT is raised, this is a fairly common finding and may be entirely explained as a side effect of valproate therapy. The true liver function is normal as demonstrated by normal albumin, INR, and bilirubin. Therefore hepatic encephalopathy is an incorrect answer.

Small intestinal bacterial overgrowth can lead to acquired hyperammonaemia, however, there is nothing to suggest that this is the diagnosis in this case.

Question 16 of 409

A 48-year-old man is reviewed in psychiatry clinic after a referral from his general practitioner with eccentric behaviour. He has no past medical history and does not take any regular medications. There is no history of drug or alcohol abuse.

On examination, he appeared unkempt and dressed exclusively in black clothing. He was accompanied by his mother.

During the course of the interview, it transpires that others have always felt he was 'odd' and he has difficulty forming social relationships. He becomes anxious in social situations. He is obsessed with ghosts and has sought to purchase ghost-hunting equipment. He occasionally feels the spirits of ghosts in his home as a cold breeze.

His affect is inappropriate throughout, avoiding eye contact while laughing at unusual points in conversation.

What is the correct description of this type of personality disorder?

- A. Antisocial
- B. Avoidant
- C. Borderline
- D. Schizoid
- E. Schizotypal

E. Schizotypal

Schizotypal personality disorder is characterised by social isolation and odd beliefs

Schizotypal is the correct answer. This personality disorder is characterized by social isolation, odd or eccentric behaviour, inappropriate affect, unusual perceptual disturbances (the cold breeze in this scenario) and odd beliefs and magical thinking (ghost-hunting in this scenario). This patient demonstrates most of these characteristics.

Avoidant is incorrect. While this personality disorder can also result in social isolation, it is characterized by a craving for others that is absent in this context. Most of the avoidant behaviors in this disorder are as a consequence of fears of embarrassment or ridicule.

Borderline is incorrect. This is characterized by fear of abandonment, unstable interpersonal relationships, impulsivity and chronic feelings of emptiness. Patients often demonstrate recurrent suicidal behaviour.

Schizoid is incorrect. While there is also a preference for solitude in this condition and few friends or confidants other than family, abnormal perceptions and magical thinking are absent. Coldness is predominant rather than eccentricity.

Antisocial is incorrect. The key characteristics of this personality disorder are repeated failure to respect lawful behaviours, deception, irritability and aggressiveness, which are not mentioned in this case.

Question 17 of 409

A 16-year-old female is brought to the general practitioner by her mother, who is concerned about the difficulties her daughter has in forming successful friendships and personal relationships. The patient herself denies this is a problem and reports that she has many 'best friends'.

During the course of the consultation, the GP becomes aware that she is inappropriately sexually seductive and seems to use her physical appearance to seek attention. In addition, she tends to dramatize events and stories and always places herself as the central protagonist. Many of her stories seem to involve emotional overreaction her part.

What is the most accurate description of this personality disorder?

A. Antisocial
B. Borderline
C. Dependent
D. Histrionic
E. Narcissistic

D. Histrionic

Histrionic personality disorder is characterised by excessive attention-seeking and emotional overreaction

A histrionic personality disorder is correct. This personality disorder is characterized by inappropriate sexual seductiveness, self-dramatization, attention-seeking behaviour and emotional overreaction. Additionally, relationships are considered to be more intimate than they are in reality., In this case, the patient considered herself to have many 'best friends', while her mother disagrees with that assessment.

An antisocial personality disorder is incorrect. This personality disorder is characterized by repeatedly breaking the law, deception, irresponsibility and a lack of remorse.

A borderline personality disorder is incorrect. This personality disorder is characterized by an unstable self-image and efforts to avoid abandonment. Impulsivity is common, along withdifficulties in controlling temper and recurrent suicidal behaviour.

A dependent personality disorder is incorrect. People with this personality disorder have difficulty making everyday decisions without excessive reassurance from others and have unrealistic fears about being left to care for themselves. They often commence an urgent search for another relationship as a source of care when a close relationship ends.

A narcissistic personality disorder is incorrect. This personality disorder is characterized by a grandiose sense of self-importance and a sense of entitlement. People who exhibit these features often take advantage of others to achieve their own needs and lack empathy.

Question 18 of 409

A 25-year-old woman is reviewed in the neurology outpatient clinic. She has a past medical history of idiopathic intracranial hypertension and she reports a recurrence of headaches. She takes regular acetazolamide.

On examination, fundoscopy reveals papilloedema. Her body mass index is 31 kg/m². Her visual acuity is normal.

A decision is made to perform a therapeutic lumbar puncture in the left lateral position. The opening pressure is 29 €‰25 cm CSF and 10 ml of cerebrospinal fluid is removed. The patient subsequently develops a headache.

What intervention could have reduced the likelihood of developing this complication?

A. Bed rest following procedure
B. Decreased quantity of CSF removal
C. Increased fluid intake post procedure
D. Performing the procedure in a sitting up position
E. Re-insertion of the stylet prior to removing the needle

E. Re-insertion of the stylet prior to removing the needle

Lumbar puncture: Re-insertion of the stylet prior to removing the needle reduces the incidence of post-LP headaches

Re-insertion of the stylet prior to removing the needle is the correct answer. This is an intervention that has been shown to decrease the incidence of post LP headaches.

Bed rest following procedure is not the correct answer. This has not been shown to decrease the incidence of post-LP headaches.

Decreased quantity of CSF removal is incorrect. This patient has had a standard amount of CSF removed (10ml). It has not been shown that varying the amount of CSF removed has any effect on the incidence of post LP headaches.

Increased fluid intake post-procedure is incorrect. This has not been shown to reduce the incidence of post-LP headaches.

Performing the procedure in a sitting up position is incorrect. This has now been shown to reduce the incidence of post-LP headaches. Additionally, we cannot measure an opening pressure in this position so it would be unsuitable in this context.

Question 19 of 409

You have been referred a 45-year-old man by the Accident and Emergency doctors with a severe headache. The headache woke him up at 3am, and he describes it as the worst headache he's ever had. He has had two episodes like this over the past three days that have followed a very similar pattern, each lasting around 70 minutes before going. The pain is mainly around the left eye and temple and is sharp in nature. On examining him you note that his left eye is watering and swollen, and there's some redness and mild bruising just above the eye. When you ask him about this bruising he says that the pain was so bad he bashed his head against the fridge door to try and help take it away.

How would you treat his headache?

A. Oral triptan
B. Indometacin
C. 100% oxygen and indometacin
D. 100% oxygen
E. 100% oxygen and nasal triptan

E. 100% oxygen and nasal triptan

Cluster headache - acute treatment: subcutaneous sumatriptan + 100% O2

What is described above is the classical cluster headache. NICE guidelines characterise cluster headaches as one-sided headache in or around the eye or temporal region associated with signs of autonomic dysfunction on the same side.

Attacks of pain:

- Usually last for 15-180 minutes
- Almost always described as the most severe pain known
- Tend to recur at the same time each day
- Often wake the person shortly after falling asleep

Signs of symptoms of autonomic dysfunction include:

- Rhinorrhoea or nasal congestion
- Red eye and/or lacrimationFacial or forehead sweating or flushing
- Constriction of the pupil and/or ptosis
- Eyelid oedema
- A sense of aural fullness

The suggested first line treatment for cluster headaches is 100% oxygen at a flow rate of at least 12 litres per minute and a subcutaneous or nasal triptan. Evidence from one small study suggests that subcutaneous sumatriptan provides faster relief of symptoms than intranasal sumatriptan.

Indometacin is used in paroxysmal hemicrania which has similar characteristics to cluster headaches, but attacks tend to be shorter (2-45 minutes) and more frequent (up to 40/day) and more common in women.

Oral triptans have no evidence base in cluster headaches.

Question 20 of 409

A 16-year-old boy attends GP with their parents for a review of constipation. They have improved well and are taking regular movicol with a soft motion every 1-2 days. The parents asked about a rash across the face. He has a background of autism and learning difficulty.

On examination, the boy has raised papules across the nose and cheeks with a warty appearance that measure 1-2mm. This does not extend across the back or chest. He has 2 patches of hypopigmentation on his lower back that are 3cm x 2cm and 1cm x 5cm. There are three hyperpigmented patches over the abdomen.

These have not resolved despite using over the counter treatments for acne.

Hb	134	Na	139 mmol/l	Bilirubin	5 µmol/l
Platelets	456	K	4.2	ALP	100 u/l
WBC	4.5	Urea	4.21	ALT	24 u/l
Neuts	2.2 *	Creatinine	78	γGT	78 u/l
Lymphs	0.1	Albumin	35 g/l		

What treatment would you start for the facial lesions?

A. Sirolimus
B. Retinoid
C. Lymecycline
D. Benzoyl
E. peroxide Excision

A. Sirolimus

Acne and hyperpigmentation -> tuberous sclerosis

This patient has autism, unusual pigmented patches on the skin and acne. This collection of signs should prompt thoughts of neurocutaneous syndromes and particularly of tuberous sclerosis. The acne that occurs is called acne sebaceum and these are angiofibromas. Compared to acne vulgaris, these do not respond to typical therapies and the first line treatment is sirolimus cream. They aretoo small and numerous to be excised. This boy should be referred to genetics to confirm the diagnosis and screen for further malignancies that he will be at risk of.

Question 21 of 409

An 18-year-old female is admitted to hospital with worsening lower limb weakness. She states that it started in her ankles, but now she is unable to stand from a squatting position which is abnormal for her. On examination, she has reduced power in ankle plantar and dorsiflexion bilaterally as well as some mild weakness in her hip extensors. Her hand grip strength is normal.

A cerebrospinal fluid (CSF) study is carried out and based on the clinical syndrome and the result she is diagnosed with Guillain-Barre Syndrome (GBS).

Which of the following treatments should be commenced in the acute setting?

A. Intravenous methylprednisolone
B. Intravenous immunoglobulin
C. Oral prednisone
D. Rituximab
E. Pyridostigmine

B. Intravenous immunoglobulin

IVIG is generally used first-line for Guillain-Barre syndrome

Immunomodulatory treatment has been proven to hasten recovery in GBS. Intravenous immunoglobulin (IVIG) and plasma exchange have proved equally effective, however, IVIG is often the initial treatment for practical reasons.

Corticosteroids (oral and intravenous) have not been found to have a clinical benefit in GBS. Consequently, this class of drugs is not currently employed in the treatment of the syndrome.

Question 22 of 409

You are working in a neurology outpatient clinic seeing a patient referred from a local GP clinic. He's a 42-year-old man who has been troubled by severe headaches over the past half a year. These headaches are the worse that he's ever had in his life, describing them as far worse that the compound fracture he sustained three years ago. These headaches tend to happen most nights at around 2am just after he falls asleep. He often paces around his kitchen for a couple of hours and often resorts to bashing his head against the fridge the pain is so bad. When probed further he mentioned that he gets a sense of fullness in his right ear (which is the side that the headache most often occurs on). He remembers having a similar problem a couple of years ago that lasted a few months before resolving on their own.

What medication is most likely to prevent these headaches?

A. Oral triptan
B. Home oxygen
C. Pizotifen
D. Propranolol
E. Verapamil

E. Verapamil

Verapamil is used for long-term prophylaxis of cluster headaches

NICE guidelines suggest that one should diagnose a cluster headache if:

- Pain lasts 15 minutes to 3 hours and is associated with intense restlessness and agitation.
- Episodes occur with a frequency between one every other day and eight times a day.
- Commonly wakes the person from sleep within 2 hours of going to sleep.
- At least five episode of pain have occurred.
- Headache is associated with at least one autonomic feature occurring on the same side as the pain.

Acute treatment for cluster headaches:

- Subcutaneous or nasal triptan
- High flow oxygen 12L/min - this should be set up at home

Prophylactic treatment:

- Verapamil

Question 23 of 409

A 20-year-old man presented to his general practitioner following an episode of collapse. He reported that he was attending a comedy night when he suddenly collapsed to the floor. He denied loss of consciousness and had a quick recovery from the event. He is normally fit and well, though reports that he is having difficulty at university as he is struggling to stay awake.

On examination, his pulse was regular at 60bpm and ECG demonstrated normal sinus rhythm. Blood pressure was 134/70mmHg on sitting and 125/65mmHg on standing.

What is the most likely cause of his collapse?

A. Transient ischemic attack (TIA)
B. Neuromuscular dystrophy
C. Cataplexy
D. Conversion disorder
E. Stokes-Adams syndrome

C. Cataplexy

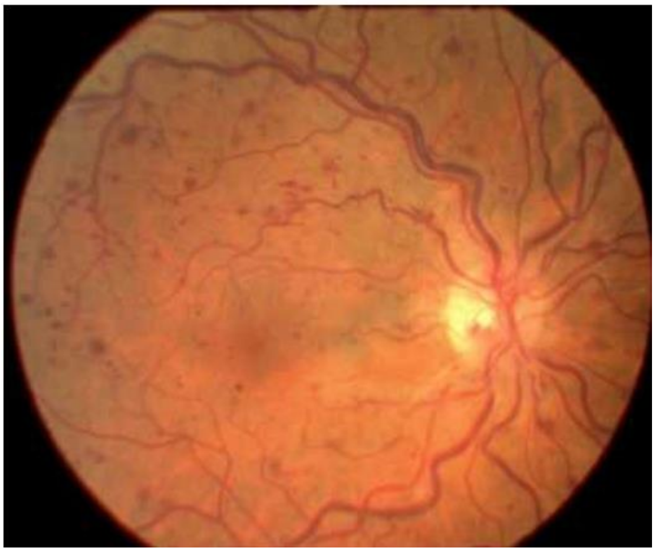
Cataplexy occurs during heightened emotion. Differential to consider with collapse of unknown cause

Cataplexy is a sudden, transient episode of muscular weakness without loss of consciousness. Typically it occurs during heightened emotions, most commonly laughter such as the comedy clue in the question. Often cataplexy co-occurs with narcolepsy which typically presents as patients recurrently falling asleep during the day, as with this patient.

Stokes-Adams syndrome is a transient AV block and results in loss of consciousness, a more progressive history would be expected with neuromuscular dystrophy and although TIAs do present with transient focal motor weakness the patient's age and the bilateral nature of his symptoms make this unlikely.

Question 24 of 409

A 73-year-old man complained of a one day history of blurred vision in the right eye. He has a past history of poorly controlled hypertension and polycythaemia but otherwise enjoys good health. Visual acuity is 6/6 in the left eye and 6/18 in the right eye. Fundoscopy of the right eye is as follows



What is the most likely diagnosis?

A. Ischaemic optic neuropathy
B. Central retinal artery occlusion
C. Papilloedema
D. Central retinal vein occlusion
E. Macular degeneration

D. Central retinal vein occlusion

Central retinal vein occlusion - sudden painless loss of vision, severe retinal haemorrhages on fundoscopy

This slide shows a non-ischaemic central retinal vein occlusion. There are dilated retinal veins and widespread multiple blot haemorrhages.

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A 43-year-old gentleman is admitted to the Emergency Department with a 2-week history of worsening drowsiness and confusion. His wife states that he had initially complained of increased tiredness and had started taking to his bed immediately after arriving home from work. One week ago he stopped going to work altogether and his wife reports that he has been sleeping for most of the day since then. This morning she had difficulty waking him, and when she did manage to rouse him he seemed confused with apparent slurring of his speech.

His past medical history is remarkable for epilepsy and bipolar disorder.

On examination, he is responsive only to pain. His vital signs are within normal limits and examination of the chest is unremarkable. His abdomen is soft, with no obvious tenderness and no organomegaly. Bowel sounds are present.

A set of blood tests are taken:

Hb	152 g/l	Na	139 mmol/l	Bilirubin	18 µmol/l
Platelets	278 * 10 /l	K	4.3 mmol/l	ALP	117 u/l
WBC	8.1 * 10 /l	Urea	5.2 mmol/l	ALT	19 u/l
Neuts	5.4 * 10 /l	Creatinine	93 µmol/l	γGT	48 u/l
Lymphs	1.8 * 10 /l	Albumin	41 g/l		
Eosin	0.2 * 10 /l	Ammonia	197 µmol/l		

His wife later remarks that his medications have recently been altered by his GP.

Which of the following is most likely to be responsible?

A. Lithium
B. Sodium valproate
C. Venlafaxine
D. Diazepam
E. Quetiapine

B. Sodium valproate

The patient's presenting symptoms are relatively non-specific and, taken in high enough doses, any of the medications listed could cause impairment of consciousness. The patient's grossly abnormal ammonia level is the critical detail in this case and confirms a diagnosis of valproate associated hyperammonaemic encephalopathy (VHE).

VHE is a well-documented complication of valproate therapy and can occur in patients with therapeutic as well supratherapeutic plasma valproate levels. Valproate inhibits carbamoyl phosphate synthetase I, an enzyme essential for ammonia metabolism in the liver, leading to elevated plasma ammonium levels. Interestingly, asymptomatic hyperammonaemia has been shown to occur in a large proportion of patients taking sodium valproate.

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A 23-year-old female has represented 2 days after discharge from the neurology team with a constant diffuse headache, worse on standing than lying down, without neck stiffness or photophobia, but associated with nausea and vomiting. 48 hours ago, she was an inpatient being investigated for intermittent headaches over the past 8 months, typically over the left side of her frontal and temporal regions, of sudden onset during any time of day, associated with double vision, lasting for 'hours' at a time. She was experiencing up to 5 episodes a week, with one episode witnessed during her admission, when the senior house officer noted bilateral restriction in vertical eye movements, adduction and unreactive pupils, spontaneously resolving after 3 hours. Her blood tests were unremarkable.

The patient underwent a CT head, demonstrating no intracranial lesions; and a lumbar puncture:

Opening pressure	12.3 cmH2O
WCC	2 /mm³
RBC	50 /mm³
Protein	0.45 g/l
Glucose	4.5 mmol/l (serum 6.7 mmol/l)
Oligoclonal bands	None present

The patient subsequently self-discharged, reporting no immediate headaches, back pain or lower limb paraesthesia after the lumbar puncture.

During this second admission, she undergoes an MRI head scan, demonstrating significant diffuse meningeal enhancement and bilateral shallow subdural haemorrhages.

What is the appropriate treatment?

A. Oral fluids, caffeine and blood patch
B. Repeat lumbar puncture I
C. ntravenous aciclovir
D. Intravenous ceftriaxone
E. Neurosurgical input

A. Oral fluids, caffeine and blood patch

There are two headaches here: the initial presentation appears to be an unusual migrainous phenomenon, possibly ophthalmoplegic migraine, while the second is clearly a low-pressure headache. The neurologists would have correct in initially ruling out any other possible causes of ocular paresis with a lumbar puncture but the CSF constituents are fairly unremarkable.

Do not be alarmed by the report of the MRI head: meningeal enhancement, thickening and shallow subdural haematoma are features of low-pressure headaches and do not represent meningitis or subdural haematomas regarding neurosurgical drainage. A repeat lumbar puncture would make exacerbate the problem and hence inappropriate. In view of the low-pressure headache occurring soon after a lumbar puncture, encouraging oral fluids, caffeine and a blood patch by the anaesthetists would be most appropriate.

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A 17-year-old Caucasian male with no past medical history presents with his first episode of sudden onset left leg weakness and numbness on his anterior left thigh, of sudden onset and persistent after 4 days. On examination, you note 3/5 weakness on flexion of his left hip and loss of sensation to light touch, pain and temperature on his anterior left thigh in the sensory nerve root L1 distribution. A contrasted MRI scan of the patient's spine reveals a hyperintense T2 signal partially within the left side of the cord at the L1, with corresponding enhancement with gadolinium. No masses were observed. Further imaging of the brain is awaited.

What is the most likely diagnosis at present?

- Multiple sclerosis
- Left spinal cord tumour
- Left anterior spinal ischaemic stroke
- Left transverse myelitis
- Guillain Barre syndrome

The history, examination and radiological evidence are strongly suggestive of partial L1 transverse myelitis. The key question is whether this represents a diagnosis of multiple sclerosis (MS) or not. The diagnosis of MS is determined by the McDonald criteria, first proposed in 2001 and most recently revised in 2010 . The lesions or clinical episodes suggestive of acute demyelinating episodes must be disseminated in both time and place. As a result, a patient presenting with only one symptomatic episode can be diagnosed with MS if their MRI scans demonstrate demyelinating plaques that are:

- 1 i) distinguished in age from the current symptomatic lesion
- ii) In at least two of 4 areas typical of MS (periventricular, juxtacortical, infratentorial, spinal cord white matter).

In this case, only one episode in time and space has been demonstrated so far. Although a risk of future MS diagnosis is acknowledged and may go on to be proven following MRI of the head, the diagnosis remains transverse myelitis at present.

It is often quoted that in patients diagnosed with transverse myelitis after their first symptomatic episode, only a small proportion go on to be diagnosed with MS later in their lives. However, more recent evidence suggests the pattern of transverse myelitis is important: only 5-10% of patientswith complete transverse myelitis will be diagnosed with MS, with a viral febrile illness the most likely underlying cause. Patients with incomplete transverse myelitis have up to 72% risk of MS consistent plaques found on MRI head, with 60-90% subsequently being diagnosed MS within 5 years .

Old MS plaques appear better defined on T1 and T2 imaging than acute lesions. Contrast studies with gadolinium are also helpful: acute plaques tend to enhance to a greater degree on T1 imaging, correlated to acute inflammation that persists for up to 8 weeks and diminishes on treatment with glucocorticoids.

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A 32-year-old male presents with his 4th episode of worst ever headache in one week. He describes the headache to always be of sudden onset on the left side of his head, of 10 out of 10 severity and that he finds bright lights extremely distressing during these periods. The episodes last for around 30 minutes, typically after dinner. He also describes redness and swelling of his left eye and a blocked left nostril during the headaches, associated with tearing of his left eye.

He has no past medical history and family history of migraines. He denies illicit drug use, is a nonsmoker and drinks two glasses of wine with dinner every night. Over the past 7 days, he has been self-medicating with paracetamol and ibuprofen. On examination, you notice no focal neurology, no meningism and fundoscopy is unremarkable.

What is the most likely diagnosis?

A. Subarachnoid haemorrhage
B. First presentations of migraine
C. SUNCT (short lasting unilateral neuralgiform headache with conjunctival injection or tearing)
D. Medication overuse headache
E. Cluster headaches

E. Cluster headaches

The main differentials are between cluster headaches and SUNCT, both of which are headache syndromes resulting in severely painful headaches associated with autonomic symptoms. It would be unusual for autonomic symptoms such as conjunctival injection, lacrimation and rhinorrhoea with migraines. Subarachnoid haemorrhage headaches rarely re-occur without disastrous consequences, focal neurology and reduced GCS. Medication overuse headache are normally of onset after a longer period of analgesia use and are more chronic in character without autonomic symptoms.

In distinguishing cluster headaches and SUNCT, the former are more prevalent in younger males below the age of 40 while SUNCT is more common in older patients above the age of 40. Cluster headaches typically onset at night, last from 15 minutes to 3 hours, while SUNCT can occur at any time of day and typically lasts for seconds to minutes. Cluster headaches rarely onset more than 3 times per day while SUNCT has been described in up to 75 times per day. A transient Horners syndrome is typical during a cluster headache but may be lacking in SUNCT. Lastly, clusterheadaches are classically, but not always, triggered by alcohol . The combination of severe nocturnal onset unilateral headache following alcohol in a patient under 40 years old, lasting for 30 minutes, suggest cluster headaches over SUNCT

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A 21-year-old male presents with difficulties in his work life. He has been fired from four jobs in the past 12 months. He states that his bosses were 'stupid' and that they did not appreciate him or give him the respect that he was due. He states that he wants unlimited success and power and he often gets angry when he cannot get what he wants. He believes that his intelligence is far superior to the rest of the general population.

What is the most likely diagnosis?

A. Antisocial personality disorder
B. Schizotypal personality disorder
C. Narcissistic personality disorder
D. Histrionic personality disorder
E. Schizoid personality disorder

C. Narcissistic personality disorder

Narcissistic personality disorder is characterised by a grandiose sense of self importance

Narcissistic personality disorder is characterised by a grandiose sense of self importance. In addition, they also often have a deep need for excessive attention and admiration, troubled relationships, and a lack of empathy for others.

Schizotypal personality disorder is characterised by social isolation and odd beliefs. Schizoid personality disorder is similar in that a need for social isolation and apathy dominates, however strange beliefs are not a diagnostic criteria of this condition.

Histrionic personality disorder is characterised by excessive attention-seeking and emotional overreaction.

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An 82-year-old man attends the clinic with weakness affecting his hands. A systematic enquiry was otherwise unremarkable He has no significant past medical history and takes no regular medications. On examination, you note bilateral weakness of the finger and wrist flexors (MRC grade 3/5) and hip flexion (MRC grade 4/5). The neurological examination was otherwise unremarkable.

Blood results are as follows:

Hb	144 g/L	Male: (135-180) Female: (115 - 160)
Platelets	168 * 10 /L	(150 - 400)
WBC	7.2 * 10 /L	(4.0 - 11.0)
Na	136 mmol/L	(135 - 145)
K	4.2 mmol/L	(3.5 - 5.0)
Urea	8.4 mmol/L	(2.0 - 7.0)
Creatinine	96 µmol/L	(55 - 120)
CRP	3 mg/L	(< 5)
Creatine kinase	1042 IU/L	(40 - 320)

What is the most likely diagnosis?

A. C6/7 radiculopathy
B. Inclusion body myositis
C. Limb-girdle muscular dystrophy
D. Myotonic dystrophy
E. Polymyositis

B. Inclusion body myositis

Older man + quadriceps and finger/wrist flexors weakness + raised CK → inclusion body myositis

Inclusion body myositis is correct. The presence of weakness of the finger/wrist flexors and hip flexors, and a raised CK in an elderly man is highly suggestive of inclusion body myositis.

C6/7 radiculopathy is incorrect. Although bilateral radiculopathy would explain the weakness in hand/wrist flexors, it would not explain the weakness in the hips. Furthermore, the raised CK is suggestive of a myopathy.

Limb-girdle muscular dystrophy is incorrect. Although this remains within the differential diagnosis, the pattern of muscle weakness is more in keeping with inclusion body myositis. Limbgirdle muscular dystrophy would classically affect the shoulder and hips, and weakness in the hands and wrists would be unusual.

Myotonic dystrophy is incorrect. The pattern of involvement, in this case, is not typical of myotonic dystrophy. Myotonic dystrophy classically presents with facial weakness and distal muscle weakness. Furthermore, the absence of any other hallmark feature of this condition (e.g. frontal balding, and grip myotonia) makes it a less likely diagnosis.

Polymyositis is incorrect. The pattern of involvement, in this case, is not typical of polymyositis. The hallmark of polymyositis is a progressive disease with symmetric involvement of the proximal girdle muscles (shoulder and pelvis muscles) and neck flexors. Myalgia is also common.

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A 32 year-old accountant presents to the neurology clinic.

For the last year he has been troubled by severe headaches. These occur in the morning, sometimes waking him from sleep. There is a sudden onset of stabbing pain behind the left eye, at the onset of which he feels the need to pace around the room, pounding his head in an effort to ease the pain. During the attacks his left eye is watery and his nose his congested. On more than one occasion he has noticed that his right pupil appears enlarged during the attacks. He tells you very clearly that the pain is so severe that at times during the attacks he has considered jumping out of the top floor of the office building where he works. Each attack lasts one or two hours, and between the attacks he feels fine.

He shows you a detailed headache dairy in which he has documented every attack over the last year. For example, in January he had a period of nine days in which attacks occurred several times daily, but in February and March he was headache free. During April he developed severe headaches which occurred almost every day until June. He then had another period of relief until August, and so on.

He has no other past medical history and no allergies. Examination is unremarkable.

What is the best treatment for prophylaxis of this man's headaches?

A. 100% oxygen
B. Verapamil
C. Sumatriptan
D. Propranolol
E. Lithium

B. Verapamil

Verapamil is used for long-term prophylaxis of cluster headaches

This is a classic description of episodic cluster headache.

Interestingly, the patient mentioned that his right pupil was enlarged, but most likely he is describing ipsilateral Horner's syndrome, which may occur during cluster attacks.

Verapamil is the established first-line agent for the prophylaxis of episodic and chronic cluster headache.

100% oxygen is the first-line treatment for acute attacks of cluster headache.

Sumatriptan is commonly used for acute attacks of cluster headache and migraine, often subcutaneously.

Propranolol is a first-line drug for the prophylaxis of migraine. Lithium is a second-line agent for the prophylaxis of cluster headache.

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A 53-year-old lady was admitted to the medical admission unit having presented to the Emergency Department with a severe headache which had developed over the last five days. She described the headache as a continual dull pain across the front of her head and was significantly worsened when she sat up as well as when she coughed or strained. It was alleviated only when she lied down in a dark room; paracetamol 1g QDS and ibuprofen 400mg TDS did not provide any relief. She denied the presence of nausea or vomiting, her vision was not affected and other than feeling tired she did not complain of any other symptoms. Her past medical history was comprised of non-classical migraines and hypothyroidism for which she was prescribed levothyroxine 175mcg OD. Seven days ago she was admitted with a headache, fever and photophobia; review of her medical notes revealed the following results from the previous

admission:

Hb	142 g/l
Platelets	326 * 10 /l
WBC	11.2 * 10 /l

Glucose	5.4 mmol/l
ESR	32 mm/hr
CRP	46 mg/l
PTT	12.2 (NR 12-14s)
APTT	44s (NR 300-46s)
Fibrinogen	5.2 (NR 2-4 g/l)

Urine MCS: NAD Blood MCS: NAD

CT Head: normal intracranial appearances; no space occupying lesion or haemorrhage identified

Lumbar puncture:

Protein	0.3g (NR 0.2-0.4 g/L)
Glucose	3.6 mmol/l
WCC	8 (<5/mm3)
Opening pressure	15 (NR 10-20 cmH2O)
MCS	NAD

Examination revealed the presence of a female in a dark room lying flat. Her temperature was 36.6ºC, heart rate 77bpm, respiratory rate 16/min and blood pressure 136/78 mmHg. Examination her cardiovascular and respiratory systems were unremarkable. Examination of her neurological system revealed the presence of normal functioning cranial nerves 2-12 with unremarkable fundoscopy; there was no neck stiffness or photophobia objectively. There was no neck stiffness and Kernig's sign was negative. Examination of her peripheral neurological system was unremarkable.

What is the single next best management option?

A. Organise urgent cranial MR venogram scan
B. Organise urgent cranial MRI scan
C. Commence therapy with sumatriptan
D. Administer epidural blood patch
E. Send CSF for xanthochromia analysis

D. Administer epidural blood patch

This lady has developed post-lumbar puncture headache (PLPH), a common complication of a lumbar puncture and is thought to be caused by excess leakage of cerebrovascular fluid causing a relatively low intracranial pressure and is aggravated significantly when assuming a non-supine posture. There are of course many other diagnoses that need to be considered, including meningitis, migraine, cortical vein thrombosis, intracerebral haemorrhage and cluster headaches. In most instances however the diagnosis of PLPH can be made clinically. Patients frequently believe the cause of their headaches is due to migraine and therefore report that avoiding bright light alleviates their headache when in fact it is lying down which is alleviating the headache. Management is conservative, including administering analgesia and sufficient fluids. If this fails to resolve the headache the gold standard management is an epidural blood patch.

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A 45-year-old woman is admitted to the neurology inpatient unit with a 3-day history of fever and headache. To exclude meningoencephalitis, a lumbar puncture is performed. Following the procedure, she developed a severe headache that was initially attributed to a post-lumbar puncture headache; however, it persists despite lying flat, adequate hydration, and analgesia. Subsequent computed tomography (CT) of the brain reveals a subdural haematoma, although the CT brain scan at admission was normal.

What could have prevented this patient's condition?

A. Fall risk assessment
B. Increased volume of CSF removed
C. Intravenous mannitol
D. Use of cutting needle for LP
E. Use of pencil-point needle for LP

E. Use of pencil-point needle for LP

The use of pencil point (atraumatic) needles reduces the risk of post-lumbar puncture headache

The correct response is use of pencil-point needle for LP because the scenario depicts a patient who has developed a subdural haematoma after a lumbar puncture. Utilisation of an atraumatic pencil-point needle can mitigate this risk. There is an increased likelihood of subdural haematoma formation with larger gauge spinal needles. Additionally, an epidural blood patch can also reduce the incidence of post-lumbar puncture headaches. Notably, the headache associated with a subdural haematoma does not decrease with recumbency, intravenous hydration, or analgesics. Management involves an autologous blood patch, epidural saline or intravenous caffeine administration. Craniotomy and surgical decompression are seldom necessitated.

In this case, the subdural haematoma is attributable to the lumbar puncture rather than trauma. A fall risk assessment is imperative for preventing traumatic subdural haematomas and is crucial in managing patients with diminished consciousness levels; however, it is not pertinent to this situation.The volume of cerebrospinal fluid (CSF) extracted does not contribute to post-lumbar puncture headache or subdural haematoma formation. Consequently, increased volume of CSF removed would not have prevented this patient's condition.

Intravenous mannitol is an incorrect choice as it plays no role in the prophylaxis against postlumbar puncture subdural haematoma.

Since conventional spinal needles elevate the risk of post-lumbar puncture complications, opting for use of cutting needle for LP would not be effective in averting either post-lumbar puncture headaches or haematomas.

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A 19-year-old male presents to outpatient clinic with a 3-month history of worsening speech slurring. He finds it extremely embarrassing and is made fun of by his friends in college, who thinks he is turning up to school drunk. He is also finding that he is having difficulty with excessive drooling. Over the past 6 weeks, his has also noted increasing weakness in lifting his right arm. He has no other past medical history, is a non-smoker and drinks alcohol only socially.

On examination, his cranial nerves demonstrate weakness in raising his eyebrows, puffing his cheeks and in pursing his lips. He displays significant scapula winging bilaterally with mild wasting of his right deltoid muscle. The musculature of his left arm is normal. The power of his right shoulder abduction and adduction is 3/5, with 5/5 in all other movements. Reflexes are all present with a normal sensory examination.

His blood tests are as follows:

Hb	144 g/l
Platelets	388 * 10 /l
WBC	5.8 * 10 /l

Na	139 mmol/l
K	4.7 mmol/l
Urea	4.6 mmol/l
Creatinine	55 µmol/l
CRP	2mg/l
Creatine kinase	150 IU/l (50-335)
TSH	2.3 mu/l
HIV	negative

What is the most likely diagnosis?

A. Myoclonic epilepsy with ragged red fibres (MERRF)
B. Myotonic dystrophy type 1
C. Polymyositis
D. Limb girdle muscular dystrophy
E. Facioscapulohumeral dystrophy

E. Facioscapulohumeral dystrophy

Facioscapulohumeral muscular dystrophy: facial weakness (e.g. difficulty closing eyes, smiling, blowing), winged scapula and weakness of the shoulderm upper arm and hip girdle muscles

This is a difficult question regarding the cause of a myopathy, onset at late adolescence and progresses. Firstly, the normal CK, CRP and lack of systemic features point against a systemic inflammatory condition such as polymyositis. Although also involving proximal muscles and presenting typically without cutaneous manifestations, polymyositis is a multisystem disorder with cardiac, pulmonary features and oesophageal involvement.

Similarly, although myotonic dystrophy type 1 patients also present from age of 20 onwards, it is another multisystem disorder with cardiac structural and conduction abnormalities, respiratory muscle involvement, gastrointestinal symptoms, testicular failure and insulin resistance, amongst a large number of clinical features. Classically, patients with myotonic dystrophy display myotonia, a slowing of relaxation following normal muscular contraction.

An absence of myoclonus makes MERRF less likely. It is a mitochondrial disorder typically presenting with myopathy, peripheral neuropathy and frequently, ataxia, Wolff-Parkinson-White syndrome and bilateral deafness. The final assessment of the type of muscular dystrophy relies simply on the muscles involved: in this case, muscles involved are facial and upper limb girdle muscles. Be aware that muscular dystrophies such as facioscapulohumeral dystrophy can initially present asymmetrically before progressing to bilateral involvement.

Question 35 of 409

A 37-year-old Japanese female presents with her second episode of loss of colour vision and significant visual acuity impairment in both eyes. Three days later, she complains of vomiting, acute urinary retention, requiring urinary catheter insertion, and inability to move either leg. On examination, she was unable to correctly name any Ishihara plates. An MRI of her brain and spine demonstrates multiple hyperintense T2 white matter lesions in her spine suggestive of demyelination, one of which extends from C5 to T1.

Which investigation confirms the diagnosis?

A. Lumbar puncture for oligoclonal bands
B. Repeat MRI spine with diffusion weighting
C. Serum aquaporin 4 antibody
D. Serum anti-NMDA antibody
E. Repeat MRI spine with gadolinium contrast

C. Serum aquaporin 4 antibody

Neuromyelitis optica is associated with aquaporin 4 positive serum antibody

The patient has presented with optic neuritis, myelitis and vomiting, strongly suggestive of a diagnosis of neuromyelitis optica (NMO), also known as Devics disease.

In this case, CSF oligoclonal bands neither confirms nor excludes a diagnosis MRI spine with DWI would demonstrate acute ischaemic infarcts if a spinal stroke was suspected. Gadolinium enhanced imaging does not add to the diagnostic criteria for NMO. Anti-NMDA antibody is a serum autoantibody for a subtype of autoimmune encephalitis.

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A 72-year-old man is referred to the oncology team after presenting to the emergency department. The patient reports progressive lower thoracic back pain over the past three weeks. The pain had now reached an intensity that had prevented him from sleeping during the past two nights, despite over the counter analgesics. The patient reported that coughing caused a severe exacerbation of the pain, but denied radiation of the pain. The patient had previously suffered mild lower back pain after exercise, but could recall no episodes that compared in intensity to his present symptoms.

The patient's wife reported she felt the patient had been unsteady on his feet over the past few days, and reported that he had nearly fallen on several occasions. The patient denied any history of sphincter disturbance or sensory symptoms. On direct questioning, the patient revealed that he had been troubled by a severe cough over the past two months and had also lost 7 kilograms in weight. He denied any history of fevers or night sweats.

The patient had an unremarkable past medical history and took no regular medications. He was a retired builder, who had stopped smoking when he took retirement seven years previously.

Peripheral nerve examination of upper limbs: normal tone, normal power, normal coordination, normal sensation, reflexes intact and symmetrical.

Peripheral nerve examination of lower limbs: tone generally increased bilaterally with 5 beats of clonus in left lower limb; power generally slightly reduced (4+/5) bilaterally, although patient limited by back pain; normal coordination; light touch sensation reduced to mid-point between symphysis pubis and umbilicus; knee and ankle jerks brisk bilaterally, plantar response up-going in left lower limb.

Respiratory examination: borderline clubbing of fingers; reduced resonance and air entry in left upper zone.

Musculoskeletal examination: point tenderness in midline of back around level of T11 / T12 vertebrae.

What is the appropriate investigation of patient's back pain?

A. MRI whole-spine within 24 hours
B. MRI lumbar-thoracic spine within 24 hours
C. CT lumbar-thoracic spine within 24 hours
D. MRI whole-spine within 7 days
E. CT myelography within 7 days

A. MRI whole-spine within 24 hours

The presentation of this patient raises strong concerns about metastatic spinal cord compression as the cause for his symptoms. In the majority of cases, the condition is secondary to bony metastasis to the vertebral spine with collapse and extra-dural compression of the spinal cord.

In around 20 % of cases of metastatic spinal cord compression there is no pre-existing diagnosis of malignancy. While this is true in the case of this patient, there are several aspects of the above assessment that are highly suspicious of a primary lung cancer.

In 95 % of cases, back pain is the most common first symptom, and can be present for up to two months before other signs occur. The patient's history of progressive pain, exacerbated by coughing, and associated with midline tenderness is typical of pain secondary to a localised lesion; however, pain can also present in a radicular pattern due to nerve root compression.

The normal upper limb examination and presence of upper motor neurone signs in the lower limbs with a sensory level, would localise the lesion to the thoracic spinal cord, and correlates with the vertebral tenderness. Bladder and bowel symptoms are often a late feature of metastatic spinal cord compression, so the absence of autonomic dysfunction does not exclude the diagnosis.

United Kingdom NICE guidance is for MRI whole-spine to be performed within 24 hours in individuals with both pain and neurological symptoms suggestive of metastatic spinal cord compression. In the presence of pain without neurological symptoms, MRI whole-spine should be performed within 7 days. It is necessary to image the entire vertebral column, as multi-level involvement is present in 30-50 % of cases.

CT scanning is often used to aid surgical or radiotherapy treatment planning. CT myelography is occasionally used in individuals with contra-indications to MRI scanning.

Question 37 of 409

A 67-year-old male presents to your neurology clinic with his wife, reporting a 3-month history of worsening vision in his left eye. He is very reluctant to seek any medical help but says that a number of factors have finally convinced him to attend.

Over the past 2 months, he reports a constant and worsening headache, worse at night and with coughing. He has occasionally felt extremely nauseated and vomited on a number of occasions, at least twice waking him from sleep. His wife also notes significant personality change: her husband is a retired farmer who prides him on being a stoic man. She notes that he has become increasingly emotional and occasionally aggressive, which she puts down however to struggling with his symptoms. Prior to these symptoms starting 3 months ago, he has always been fit and well with no past medical or drug history.

On examination, you note a left relative afferent papillary defect with equally sized pupils. Visual acuity in the left eye is 6/60 and 6/9 on the right. Testing of colour vision with Ishihara plates demonstrates 0/17 on the left and 17/17 on the right. A central scotoma is found in the left eye. Fundoscopy of the left eye reveals a pale optic disc with poor vasculature while the right appears swollen. A full range of painless eye movements is demonstrated. The remaining examination of the cranial nerves, upper and lower limbs are unremarkable. Drops of blood are unremarkable, a MRI head is awaited.

What is the most likely diagnosis?

A. Foster-Kennedy syndrome
B. Idiopathic intracranial hypertension (IIH)
C. Multiple sclerosis (MS)
D. Frontotemporal dementia
E. Age related macular degeneration (ARMD)

A. Foster-Kennedy syndrome

This is a difficult question requiring recognition of a high-pressure headache, left optic atrophy and contralateral swollen disc secondary to papilloedema. The combination of an ipsilateral optic atrophy and contralateral papilloedema is an MRCP favourite:

Foster-Kennedy syndrome, typically caused by a frontal lobe mass, normally a meningioma, which compressing on the ipsilateral optic nerve and olfactory nerve, while increasing intracranial pressure for the contralateral optic nerve sheath.

Idiopathic intracranial hypertension may present asymmetrically but typically produces bilateral disc swelling. It is conceivable the patient has multiple sclerosis with optic atrophy as a sequelae of left optic neuritis, followed by a second episode of contralateral optic neuritis presenting as papillitis, inflammation of the optic nerve head. This is unlikely with only one episode of visual dysfunction described with an insidious progressive onset.

Frontotemporal dementia may explain the behavioural changes but does not account for visual symptoms.

Lastly, age-related macular degeneration can mimic papillitis on fundoscopy, with deposition of drusen making the optic disc 'look' swollen. However, the deposition of these retinal cell byproducts is generally more diffuse across the retina, with wet ARMD also displaying abnormal retinal vasculature. While retinal atrophy may be present, it does not typically produce optic atrophy.

Question 38 of 409

A 45 year old previously fit and well man presents to the emergency department with worsening leg weakness. He is in full time employment as a brick layer and is normally very active. Over the last 24 hours he has started dragging his feet and feels unsteady when walking, describing his gait 'like a drunk man'. On questioning he also describes increasing difficulty passing urine and has not had the sensation to empty his bladder for the past eight hours. He denies any preceding trauma, recent viral illness or similar previous symptoms in the past.

On examination he has normal muscle bulk and no fasciculations. There is symmetrical lower limb flaccid paralysis to the hips, with symmetrical hyporeflexia. He has a sensory level to T10 and is in urinary retention. Examination of the upper limbs and cranial nerves is entirely normal.

Which of the following would be the most useful initial investigation?

A. Aquaporin 4 antibodies
B. Lumbar puncture
C. MRI spinal cord
D. MRI brain
E. HIV serology

C. MRI spinal cord

This man presents with signs and symptoms of acute transverse myelitis. The underlying aetiological process can be diverse; it can be associated with underlying autoimmune systemic response, infectious cause or demyelinating process such as neuromyelitis optica.

The presence of a sensory level suggests a spinal cord lesion, as such a CT or MRI Brain would not be the first investigation. The most important initial approach in a patient presenting with acute disturbance of motor, sensory or autonomic function (and a sensory level) is to rule out a compressive cord lesion; an MRI spine is therefore an essential first step. Although HIV serology would be important in this patient, as aforementioned, the most important step is to exclude a compressive lesion.

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You are called to see a 67-year-old lady by psychiatry who is asking for your opinion on management. Over the past 3 weeks, her husband has become increasingly concerned by her behaviour. She has become suspicious of him and accused him of stealing 'my true husband'. Initially, the lady would avoid her husband and refuse to eat food that he had prepared. However, today she threatened him with a knife and the police had to be called.

The psychiatry doctor is concerned as he notes that she was admitted to hospital 5 weeks ago and was treated for a suspected urinary tract infection with intravenous antibiotics. Subsequent testing showed the pathogen to be an extended-spectrum beta-lactamase producing bacteria. She currently has a temperature of 38.7 degrees heart rate 105bpm regular RR 18 Sats 99% on room air. Her husband notes that she has been spending more time in the toilet over the past 3 weeks but is unsure if this is due to her paranoia.

Her husband describes an episode 30 years ago where she required antidepressants, antipsychotics and ECT after a close family bereavement. She is otherwise well and has no history of cognitive problems. Her husband states that there is a strong history of mental health problems in her family but he is unable to be more specific.

When you speak to her she appears to be confused and scores 19/30 on the Mini Mental State Examination. She can point to, name and recognise her husband and can also pick him out from pictures. However, she tells you that the man standing next to her is not her husband but a lookalike who has replaced him. Despite all your best efforts to show evidence to the contrary, she cannot be persuaded to change her opinion.

Which of the following below would best describe her presentation?

A. Delirium causing Cotard syndrome
B. Delirium causing Fregoli syndrome
C. Delirium causing Capgras syndrome
D. Late onset schozphrenia causing Capgras syndrome
E. Atypical severe depression

C. Delirium causing Capgras syndrome

Firstly, this woman has features that suggest delirium having a factor to play in her presentation: fever, acute cognitive impairment, infective symptoms, recent treatment for a highly resistant infection. Late-onset schizophrenia is very rare and when it does occur has no association with family history of mental health problems or personal history of depression. In addition, the duration of symptoms is probably too rapid for late onset schizophrenia.

Secondly, the woman is describing a delusion where she believes her husband to be replaced by an imposter. This is known as Capgras syndrome and can be seen in organic states such as delirium as well as in schizophrenia. It is rare but is most commonly seen in older women. The delusion most commonly relates to a life partner and can sometimes lead to serious violence being perpetrated against the supposed imposter.

Cotard syndrome is a nihilistic delusion seen in severely depressed people where they believe that they, or a part of their body is dead.

In Fregoli syndrome, the patient believes that an individual (who is almost always a persecutory figure and someone close to them) has taken on many different guises. This syndrome is named after an artist called Leopoldo Fregoli renowned for his ability to change costumes very quickly. A person with Fregoli syndrome will identify several different strangers as being the persecutor in disguise.

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A 28-year-old male presents to the acute medical unit with confusion. His girlfriend states that over the past couple of days he has become progressively more confused. His past medical history includes epilepsy. His drug history includes sodium valproate. On examination, you note myoclonus and his Glasgow coma score is reduced at 13 (M6, V4, E3).

Blood results are as follows:

Hb	142 g/l	Na	138 mmol/l	Bilirubin	14 µmol/l
Platelets	388	K	4.4 mmol/l	ALP	82 u/l
WBC	10.8	Urea	5.8	ALT	24 u/l
Neuts	8.4	Creatinine	72	γGT	36 u/l
Lymphs	1.2	Albumin	42 g/l	Plasma ammonia	65 µmol/L (normal range 11-32 µmol/L)

What treatment is indicated?

A. L-carnitine
B. Lactulose
C. Rifaximin
D. Sodium phenylbutyrate
E. Sodium benzoate

A. L-carnitine

L-carnitine can be used in the treatment of hepatotoxicity or hyperammonemic encephalopathy in the context of an acute or chronic overdose of sodium valproate

Valproate-induced hyperammonaemic encephalopathy (VHE) is an unusual complication characterised by a decreasing level of consciousness, focal neurological deficits, cognitive slowing, vomiting, drowsiness, and lethargy. Asymptomatic hyperammonaemia occurs in 15-50% of patients on valproate.

Treatment with L-carnitine may be beneficial in reducing ammonia levels. Lactulose and rifaximin are used in the prevention of hepatic encephalopathy.

Intravenous sodium phenylbutyrate and sodium benzoate are used to treat hyperammonaemia in patients with urea cycle enzyme deficiencies.

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A 52-year-old male presents to the emergency department with a 5-week history of constipation not relieved by over the counter laxatives. His past medical history was significant for hypertension, COPD and schizophrenia. His current medications include ramipril, clozapine and tiotropium. He denied weight loss, poor appetite or other alarming symptoms. His family history was significant for bowel cancer with his father and uncle having died of colon cancer aged 78 and 82 respectively. His sister had a hysterectomy at the age of 72 but he was not sure why. On examination, his blood pressure was 124/78mmHg lying and 115/80mmHg standing. Pulse was 63/min. Clinical examination did not reveal any significant findings.

Hb	137 g/l
MCV	82 fl
Platelets	420 * 10 /l
WBC	7 * 10 /l
Creatinine	89 umol/L
Urea	4.7 umol/L
Na+	143 mmol/L
K+	3.9 mmol/L
Corrected Calcium	2.3mmol/L
FOB	negative
Abdominal X-ray	faecal loading

What is the most likely cause of constipation in this case?

A. Clozapine
B. Colon cancer
C. Illicit drugs
D. Dehydration
E. Malingering

A. Clozapine

One of the most common side effects of clozapine is constipation/intestinal obstruction

This is an interesting case and the differential is between colon cancer and clozapine-induced constipation. Clozapine is reserved for drug-resistant schizophrenia and rightfully so as it comes with serious side effects one of which is constipation.

Constipation occurs in 14% of people on clozapine and it is believed to be an anticholinergic effect. There are papers in literature which report more serious implications like bowel infarction and death from longstanding clozapine-induced constipation.

Cancer is unlikely here as this patient has no other alarming symptoms or signs (no weight loss, normal haemoglobin, negative FOB) to suggest this and also the cancer cases in his family history are unlikely due to a hereditary condition as it has appeared in elderly relatives with no evidence of anticipation. In practice though cancer should be excluded.

There is no indication of illicit drug use and dehydration is unlikely to cause such long history of constipation without physical findings. Here we are not told of any finding suggesting dehydration on clinical examination. Malingering usually indicates personal gains of which there are not mentioned.

Clozapine is a common cause of constipation and should not be ignored. The above case is a good example.

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A 74-year-old man is brought to the emergency by ambulance with limb weakness and speech disturbance. He awoke at 8 am with the symptoms and was last seen by his wife at 10 pm the previous night, at which point he was well. His past medical history includes hypertension and hypercholesterolemia. He takes ramipril and atorvastatin. He does not smoke or drink alcohol. He is functionally independent at baseline and is a non-executive director of a major multinational organization.

His observations are heart rate 88 beats per minute, blood pressure 128/67 mmHg, respiratory rate 17/minute, oxygen saturation 96% on room air and temperature 37ºC.

Neurological examination reveals an expressive dysphasia and 3+/5 power in the right upper and lower limbs with correspondingly reduced sensation.

Blood glucose testing is normal. An ECG demonstrates atrial fibrillation.

Urgent CT head and CT angiography are organized which demonstrate a proximal occlusion of the anterior circulation and no haemorrhage.

The patient proceeds to a diffusion-weighted MRI, which demonstrates a limited core infarct volume.

What is the best choice of management?

A. Apixaban
B. Clopidogrel
C. Intravenous thrombolysis
D. Intravenous thrombolysis + thrombectomy
E. Thrombectomy

E. Thrombectomy

For thrombectomy in acute ischaemic stroke, an extended target time of 6-24 hours may be considered if there is the potential to salvage brain tissue, as shown by imaging such as CT perfusion or diffusion-weighted MRI sequences showing limited infarct core volume.

Thrombectomy is the correct answer. This treatment can be offered to those patients who were last known to be well 6-24 hours previously (including wake-up strokes) and who have confirmed occlusion of the proximal anterior circulation but limited core infarct volume on diffusionweighted MRI. This patient fulfils all of those criteria.

Apixaban is incorrect. While this patient has new atrial fibrillation, anticoagulation is typically delayed for two weeks after an ischaemic stroke to prevent haemorrhagic complications.

Clopidogrel is incorrect. This is used for secondary prevention of ischaemic stroke (not caused by atrial fibrillation) and typically follows treatment in the initial period with high dose aspirin.

Intravenous thrombolysis is incorrect. This patient presents outside the thrombolysis window (4.5 hours) and so this treatment is unsuitable.

Intravenous thrombolysis + thrombectomy is incorrect. This patient presents outside the thrombolysis window and so this treatment would not be offered in combination with thrombectomy.

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An 85-year-old woman was referred by her General Practitioner to the stroke team on an urgent outpatient basis after her rest home staff reported an unusual episode the previous week. The patient normally suffered from mild dementia but was independent with activities of daily life with minimal assistance from care staff. One week previously, she had been found unusually drowsy in an armchair at her home. Staff recalled that she had been inconsistent with following commands and in particular was unable to raise her left arm in the air and was unable to stand. The drowsiness and arm weakness had resolved within 30 minutes. However, the patient had subsequently been more confused than normal with reduced mobility. On direct questioning, rest home staff report that a few days before the above episode, the patient had lost her balance and had sat down heavily on the ground. The care worker with her at the time was certain that the patient had not hit her head during this incident.

Past medical history included mild dementia, myocardial infarction and atrial fibrillation. Regular medications included aspirin 75 mg daily, ramipril 2.5 mg daily, bisoprolol 2.5 mg daily, simvastatin 20 mg daily and warfarin (target INR 2-3). Review of recent blood test monitoring showed that the patients INR had been well controlled over recent months with no results outside of the therapeutic range. The patient did not drink and was a long-term ex-smoker. As mentioned above, the patient normally needed minimal assistance with self-care and was fully continent.

Examination in clinic demonstrated a significant cognitive impairment (abbreviated mental test score 4/10) but without evidence of dysphasia. Pupils were equal and reactive to light with no papilloedema on fundoscopy. There was a full range of conjugate eye movements. No facial weakness or sensory loss was demonstrated. Tongue and palate function was unremarkable with no weakness of trapezius muscle. Examination of the peripheral nerves was unremarkable except for possible slight pronator drift in the left arm and an up going left plantar response. Cardiovascular examination was unremarkable except for an irregular pulse. The respiratory and abdominal examination was unremarkable.

Results of investigations performed at the time of clinic assessment are given below. Electrocardiogram: atrial fibrillation at rate 80 bpm, normal axis, inferior T wave inversion Chest x-ray: clear lung fields

Carotid doppler: no significant stenosis in right internal carotid artery; 50 % stenosis in the left internal carotid

Transthoracic echocardiogram: mild left ventricular systolic impairment, normal valvular function, no mural thrombus

What is most likely finding on CT brain scan performed after clinic review?

A. Sub-arachnoid haemorrhage
B. Cerebral infarction
C. Intracerebral haemorrhage
D. Normal pressure hydrocephalus
E. Sub-dural haematoma

E. Sub-dural haematoma

The diagnosis of sub-dural haematoma can be challenging, especially in those individuals with underlying dementia. In particular, a high index of suspicion is required in elderly patients treated with anticoagulant drugs. Sub-dural haemorrhage commonly occurs after minor injury, with up to 50 % of cases preceded by a fall without head injury. Transient focal neurological deficit can occur in up to 20 % of cases, leading to the potential for diagnostic confusion with transient ischaemic attack.

In this case the patient's history of a fall, anticoagulation and worsening confusion all point towards sub-dural haematoma as the most likely underlying pathology. The lack of risk factors for cerebral infarction other than appropriately treated atrial fibrillation make this less likely. The history is less likely to be consistent with the other possible answers.

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An urgent medical consult was requested regarding a 52-year-old female who had been admitted to the neurology ward a few hours previously and who had developed hypotension. She had presented to the Emergency Department with new weakness in her legs that had occurred over a course of three days. She also complained of continual lower back pain and pain in the top of her thighs, and as her illness progressed she was no longer able to weight bear. She had a past medical history of hypothyroidism, for which she was prescribed levothyroxine 125mcg OD, and other than an episode of viral gastroenteritis diagnosed by her GP two weeks prior to her current admission she was fit and well. Upon transfer to the neurology ward blood investigations, a chest x-ray, CT head scan and lumbar puncture were performed, the results of which are as follows:

Hb	132 g/l
Platelets	248 * 10 /l
WBC	8.2 * 10 /l

Na	138 mmol/l
K	5.2 mmol/l
Urea	6.4 mmol/l
Creatinine	77 µmol/l

CRP	15 mg/l
Glucose	7.2 mmol/l
TSH 0.26	(NR 04-3.6 mu/l)
Free T4 10.6	(NR 10 - 20 pmol/L)

Autoantibody screen: awaiting results

Urinalysis: negative Urine MCS: awaiting results Blood MCS: awaiting results

Chest x-ray: normal appearances of heart and lung fields

CT head: normal intracranial appearances

Lumbar puncture:

Protein	0.8g (NR 0.2-0.4 g/L)
Glucose	6.3 (blood glucose 7.2)
WCC 8	(<5/mm³)
Opening pressure	15 (NR 10-20 cmH2O)
Microscopy	nil seen, awaiting culture and sensitivity result

Whilst awaiting definitive measurement her blood pressure was noted to fluctuate between 55/30 mmHg and 162/84 mmHg within a matter of minutes; at present her blood pressure was 62/42 mmHg. Upon examination of her cardiovascular system, she was clinically well perfused with a normal and palpable radial pulse, a JVP of 3cm and a peripheral and central capillary refill time of less than two seconds. He heart rate was 82 bpm. Examination of her respiratory system was unremarkable with no respiratory distress, a respiratory rate of 16/min and oxygen saturations of 99% on air. Examination of her gastrointestinal system was unremarkable. Examination of her peripheral nervous system revealed a power deficit of grade 2/5 in all muscle groups of the lower limbs, power 5/5 in the upper limbs and normal sensation and coordination throughout. Knee and ankle reflexes were absent bilaterally. Cranial nerve examination was unremarkable.

What is single next best step with regards to management of her blood pressure?

A. Commence dobutamine infusion
B. Commence noradrenaline infusion
C. Commence intravenous fluid resuscitation with 500ml of colloid stat
D. Commence intravenous immunoglobulin
E. Arrange urgent plasmapheresis

D. Commence intravenous immunoglobulin

IVIG is generally used first-line for Guillain-Barre syndrome

This lady has developed Guillain Barre Syndrome (GBS) with evidence of a temporary autonomic neuropathy, as manifested by her highly labile blood pressure. The treatment of this is therefore to treat the underlying disorder, which in this instance is with intravenous immunoglobulin. In the presence of GBS complicated by autonomic neuropathy blood pressure is not a reliable indicator and any thus any decisions regarding fluid status must be in concordance with clinical examination findings. In this question, she is clearly euvolaemic clinically and so there is no indication for fluid or inotropic support.

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A 60-year-old man presents to the neurology clinic with progressive weakness in the upper and lower limbs bilaterally. Symptoms initially came on gradually over 3 years and he is now bedbound. His past medical history is significant for hypertension, which is well controlled on amlodipine 10 mg once daily. On examination, there is no sensory abnormality and deep tendon reflexes are intact.

Calcium	2.5 mmol/L	(2.1-2.6)
Phosphate	1.2 mmol/L	(0.8-1.4)
Magnesium	0.9 mmol/L	(0.7-1.0)
Thyroid stimulating hormone (TSH)	4.0 mU/L	(0.5-5.5)
Free thyroxine (T4)	13 pmol/L	(9.0 - 18)
Amylase	80 U/L	(70 - 300)
Uric acid	0.53 mmol/L	(0.18 - 0.48)
Creatine kinase	1020 U/L	(35 - 250)
Electromyography (EMG)	Suggestive of inflammatory myopathy	

Power is significantly reduced to 1/5 in the quadriceps and wrist flexors. Relevant investigations are shown below:

What is the most likely specific finding on muscle biopsy?

A. Absent dystrophin protein
B. CD8+ T-cell infiltrates
C. Cytoplasmic inclusions
D. Muscle necrosis
E. Ragged red fibers

C. Cytoplasmic inclusions

Older man + quadriceps and finger/wrist flexors weakness + raised CK → inclusion body myositis

The scenario describes a patient with inclusion body myositis. Weak flexors and extensors and involvement of both proximal and distal muscles, as described in this scenario, are characteristic of inclusion body myositis. It typically occurs in older males and CK is raised, as with all inflammatory myopathies. Normal sensations and intact deep tendon reflexes also point towards inflammatory myopathy. Muscle biopsy findings of cytoplasmic inclusions are the distinguishing feature of inclusion body myositis. The cytoplasmic inclusions are ovoid, electron-dense bodies that are surrounded by clear haloes.

The age and onset of this patient's disease do not favour a diagnosis of Duchenne muscle dystrophy, which would arise in childhood. A biopsy would instead show absent dystrophin proteins.

Polymyositis is the most common AIDS-related myopathy, characterised by the presence of CD8+ T-cell infiltrates in the muscles. The patient in this scenario denies any significant medical problems and has no history of repeated infections, weight loss, fever or any HIV-related features.

Muscle necrosis, muscle fibre regeneration, and inflammatory infiltrates are the biopsy findings of all inflammatory myopathies. These are not specific to inclusion body myositis.

Ragged red fibres on muscle biopsy are a feature of mitochondrial myopathies. These rare diseases are unlikely in this patient, as they would present at a much younger age.

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A 55-year-old male is found confused on street by a policeman. Hospital records document that he is a known to be a type 2 diabetic with previously DNAs to clinic and known poor compliance with medications. He also had two previous admissions to hospital, 2 and 10 years ago, for alcohol withdrawal. On examination, the patient is not orientated in time or place, scoring 0 out of 10 on abbreviated mental. He is able to follow your commands in lifting his upper and lower limbs during his neurological exams. All reflexes were present. He fails to follow your finger with his eyes on cranial nerve examination. His gait is grossly ataxic, as you noted when he tried to walk to the toilet against medical and nursing advice. His blood sugar is awaited.

What is the diagnosis?

A. Wernicke's encephalopathy
B. Delirium
C. Miller-Fisher syndrome
D. Hypoglycaemia secondary to anti-glycaemic overdose
E. Acute ischaemic stroke

A. Wernicke's encephalopathy

The patient presents with a triad of ophthalmoplegia, ataxia and encephalopathy, suggestive of Wernicke's encephalopathy. The pathophysiology relies on an underlying thiamine deficiency, which is a key co-factor in energy metabolism and is often deficient in alcoholics. Miller-Fisher syndrome is a key differential in this case: this is a variant of Guillain-Barre syndrome presenting with ophthalmoplegia, ataxia and areflexia. Delirium and hypoglycaemia should not present with ophthalmoplegia.

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You have been referred a 45-year-old man by the Accident and Emergency doctors with a severe headache. The headache woke him up at 3am, and he describes it as the worst headache he's ever had, (although he admits he's not a regular headache sufferer). He has had seven episodes like this over the past two weeks that have followed a very similar pattern, with the other two headaches lasting around 60 minutes before going. The pain is mainly around the left eye and temple and is sharp in nature. You have to ask him to sit down to examine him because he is up and pacing around his room, clearly very agitated. On examining him you note that his left eye is watering and swollen, and there's some redness and mild bruising just above the eye. When you ask him about this bruising he says that the pain was so bad he bashed his head against the fridge door to try and help take it away.

On examination he has normal power, sensation and reflexes in all four limbs.

On examining his cranial nerves you notice that he has a mild left sided ptosis and miosis, and there is conjunctival injection and lacrimation on that side too. His neck movements are slow but complete, and he has no obvious stiffness or photophobia

What is the most likely cause of his headache?

A. Cluster headache
B. Subarachnoid haemorrhage
C. Paroxysmal hemicrania
D. Migraine
E. Tension headache

A. Cluster headache

What is described above is the classical cluster headache. NICE guidelines characterise cluster headaches as one-sided headache in or around the eye or temporal region associated with signs of autonomic dysfunction on the same side.

Attacks of pain:

- Usually last for 15-180 minutes
- Almost always described as the most severe pain known
- Tend to recur at the same time each day
- Often wake the person shortly after falling asleep

Signs of symptoms of autonomic dysfunction include:

- Rhinorrhoea or nasal congestion
- Red eye and/or lacrimation
- Facial or forehead sweating or flushing
- Constriction of the pupil and/or ptosis
- Eyelid oedema
- A sense of aural fullness

Tension headaches tend to be bilateral, pressing, with a pain intensity of mild to moderate and without other associated signs or symptoms.

Migraines can be both unilateral or bilateral and tend to be throbbing or banging in nature. While they can be severe in nature, they tend not to be described as the worst pain the patient has ever had. Unlike the agitation that comes with cluster headaches, patients with migraines tend to avoid activities of daily living shutting themselves in quiet dark rooms to provide relief. In addition the headaches tend to be longer, most commonly being between 4 hours and 3 days in adults.

Paroxysmal hemicrania attacks then to be much shorter (2-30 minutes), happen with much higher frequency (>5 times/day), and are more common in women. In an exam situation they will also tend to mention that the patient has found complete relief with indometacin (or this will be the subject of the question).

Subarachnoid haemorrhage headaches do not have a preceding history of previous attacks.

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A 54-year-old gentleman is referred to Neurology clinic with a 2 month history of nocturnal headache and difficulty walking.

On examination he has an ataxic gait and impaired co-ordination affecting the left arm and leg. Blood results show the following

Hb	190 x10 /L	130-168 x10 /L
WCC	5.4 x10 /L	4.2-10.6 x10 /L
Platelets	180 x10 /L	130-370 x10 /L
CRP	4 mg/L	0-5 mg/L
Creatinine	90 µmol/L	60-125 µmol/L
eGFR	>90 mL/min/1.73 m	>90 mL/min/1.73 m
Na	140 mmol/L	133-146 mmol/L
K	4.1 mmol/L	3.5-5.3 mmol/L
ALT	31 IU/L	0-45 U/L
ALP	54 IU/L	30-130 IU/L
Bilirubin	10 µmol/L	0-21 µmol/L

A CT (computed tomography) brain scan is done which shows shows as a well-defined, low attenuation region in the posterior fossa with an enhancing nodule on the wall

What is the most likely underlying lesion?

A. Meningioma
B. Cerebellar metastasis
C. Astrocytoma
D. Haemangioblastoma
E. Medulloblastoma

D. Haemangioblastoma

Cerebellar haemangioblastomas release erythropoietin so can cause a rise in haemoglobin

Cerebellar haemangioblastomas release erythropoietin as a paraneoplastic syndrome causing an elevated haemoglobin. None of the other tumours mentioned are associated with this phenomenon.

Haemangioblastoma is an uncommon tumour, but it is the most common primary intra-axial tumours in the posterior fossa in adults.

Astrocytomas and medulloblastomas are encountered in the posterior fossa of younger adults but are rare in older adults, accounting for <1% all tumours

The outcome for haemangioblastomas is very good, if surgical extraction of the tumor can be achieved

Haemangioblastomas are commonly seen in von- hippel Lindau (VHL) syndrome. These individuals have a worse prognosis than those who have sporadic tumors since those with VHL syndrome usually have more than one lesion

Question 49 of 409

A 35-year-old gentleman presents with weakness raising up his arms and some drooping of the eyelids and his shoulder blades are pronounced. He says his symptoms are mild and he probably would not have noticed it except his father had similar symptoms that have steadily got worse. This gentleman has no other past medical history of note and is taking no regular medications. On examination there is mild evidence of scapula winging, proximal weakness of both upper limbs, ptosis and he has difficulty whistling. There is normal power of his lower limbs and reflexes and sensation is intact throughout.

Creatine kinase 220 units/L

A muscle biopsy is sent but is pending, what is the most likely diagnosis?

A. Facioscapulohumeral dystrophy
B. Myotonic dystrophy
C. Emery-Dreifuss muscular dystrophy
D. Becker muscular dystrophy
E. Oculopharyngeal muscular dystrophy

A. Facioscapulohumeral dystrophy

Facioscapulohumeral muscular dystrophy: facial weakness (e.g. difficulty closing eyes, smiling, blowing), winged scapula and weakness of the shoulderm upper arm and hip girdle muscles

Facioscapulohumeral dystrophy is usually an autosomal dominant condition and although it affects men and women equally, men may be more symptomatic. Shoulder, upper arm and facial muscles are the most often implicated and lower limb involvement is less common. Winging of the scapulae are one characteristic finding which may necessitate surgical fixation. It is a progressive condition however prognosis is usually good with minor disability and lifespan unaffected.

Myotonic dystrophy is the most common muscular dystrophy of which symptoms include delayed relaxation of muscles wasting and weakness as well as cardiac conduction deficits, cataracts and characteristic facies. Becker muscular dystrophy usually presents in childhood with predominantly lower limb and girdle weakness. Patients with Emery-Dreifuss muscular dystrophy also usually present in childhood, characteristically with contractures and oculopharyngeal muscular dystrophypresents usually over the age of 40 with eyelid, facial and throat muscle weakness initially followed by pelvic and shoulder weakness.

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A 45-year-old man complains of lower limb numbness associated with weakness. He also noted some recent problems with urinary incontinence and has recently attended the opticians due to some blurring of vision

On examination he has bilateral lower limb weakness (grade 3+/5). His tone was spastic on both sides with exaggerated reflexes. The planter reflexes were up-going bilaterally. The sensory level was at the tenth thoracic vertebra (T10). Eye examination revealed a normal visual acuity with a normal fundal examination.

He has a history of asthma and is currently well controlled with just a salbutamol inhaler.

Magnetic resonance imaging (MRI) of the brain is normal. MRI of the spine shows a hyper intense lesion spanning from (T7-T12). The following investigations had been ordered:

Na+	135 mmol/l
K+	4 mmol/l
Creatinine	80 µmol/l
Urea	4.5 mmol/l
CRP	5 mg/l
ESR	10 mm/hr
Urine analysis	Normal
ANA	Negative

Which one of the following investigations would be most useful to reach a diagnosis?

A. Auditory evoked response potentials
B. CSF analysis for oligoclonal bands
C. Anti-aquaporin 4 antibodies
D. Serum B12
E. Visual evoked response potentials

C. Anti-aquaporin 4 antibodies

Neuromyelitis optica is associated with aquaporin 4 positive serum antibody

Neuromyelitis optica (Devic disease) should be suspected when the following is noted:

- quadriparesis or paraparesis within days to weeks
- vision changes
- normal brain MRI
- presence of anti-NMO antibodies (anti-aquaporin 4 antibodies)
- responsive to immunosuppressant

Question 51 of 409

A 62-year-old man is being reviewed in the neurology clinic for 6 months of progressive difficulty walking and memory issues. He has difficulty initiating movements and has become increasingly forgetful. He also suffers from increased urinary frequency, with a few instances of urinary incontinence. The patient has a history of subarachnoid haemorrhage 8 months prior, for which a craniotomy and clipping were performed.

A CT scan is requested and performed.

What abnormality is most likely to be relevant to his presentation?

A. Frontal & temporal lobe atrophy
B. Midbrain atrophy & enlargement of third ventricle
C. Normal
D. Ventriculomegaly with enlarged sulci and shrunken gyri
E. Ventriculomegaly with normal-sized sulci and gyri

E. Ventriculomegaly with normal-sized sulci and gyri

Urinary incontinence + gait abnormality + dementia = normal pressure hydrocephalus

Dementia, urinary incontinence, and gait abnormality, as described in this scenario, are the classic triad of normal pressure hydrocephalus (NPH). The patient's symptoms started 2 months after a subarachnoid haemorrhage, most likely due to reduced cerebrospinal fluid (CSF) absorption by the arachnoid villi. Neuroimaging in NPH shows ventriculomegaly with normal-sized sulci and gyri. There is an enlargement of the frontal, temporal, and occipital horns of the lateral ventricles. This enlargement is disproportionate to the sulci, which are mostly normal in size. The definitive treatment for NPH is ventriculoperitoneal (VP) shunting. The dementia, gait disturbance, and incontinence associated with NPH are reversible and patient improves significantly after a successful VP shunt.

Frontal and temporal lobe atrophy may be seen in a patient with frontotemporal dementia (FTD). Behavioural changes are the most common presenting feature of FTD, which is absent in this scenario and therefore FTD is a less likely diagnosis here. A history of urinary incontinence along with dementia and gait abnormality is more in favour of normal pressure hydrocephalus(NPH) than FTD.

Midbrain atrophy and enlargement of the third ventricle is a characteristic imaging feature of progressive supranuclear palsy (PSP). Gait disorder in PSP is also associated with a typical supranuclear gaze which is absent in this scenario.

NPH is diagnosed by ventricular enlargement on neuroimaging and so a normal study is unlikely to be seen in such a scenario.

Alzheimer's disease may show ventriculomegaly with enlarged sulci and shrunken gyri on imaging. The acute onset of symptoms and urinary incontinence in this scenario makes Alzheimer's disease much less likely.

Question 52 of 409

A 62-year-old man is admitted to the emergency department at 09:15 with right-sided arm and leg weakness and dysphasia. The symptoms were present when he woke up that morning around 08:30. He was asymptomatic when he went to bed last night around midnight.

He lives alone and is fully independent. He drives to the shops each weekend. He is an ex-smoker. His past medical history consists of hypertension and carpal tunnel syndrome.

On arrival, he has a National Institutes of Health Stroke Scale (NIHSS) of 9. His Glasgow coma scale (GCS) is 15. His cardiac monitor shows he is in atrial fibrillation.

He is transferred to radiology where he has a CT angiogram with CT perfusion. The report reads evidence of ischaemic stroke to the left proximal anterior circulation. On perfusion imaging, there is a small central area with markedly decreased cerebral blood flow suggestive of a limited ischaemic core. In proportion, there is a large surrounding area with only moderately reduced cerebral blood flow and near-normal cerebral blood volume suggestive of penumbra. There is mild cerebral oedema.

What is the appropriate management?

A. Delayed thrombolysis
B. Start the patient on a direct-acting oral anticoagulants
C. Start the patient on aspirin and apply anti-embolism stockings
D. Start the patient on aspirin and high-dose statin
E. Thrombectomy

E. Thrombectomy

For thrombectomy in acute ischaemic stroke, an extended target time of 6-24 hours may be considered if there is the potential to salvage brain ptissue, as shown by imaging such as CT perfusion or diffusion-weighted MRI sequences showing limited infarct core volume.

As the patient was well going he went to sleep, the longest time from onset to presentation is 9 and a half hours. He has a good baseline function (functional status would be less than 3 on the modified Rankin scale) and his NIHSS is greater than 5 making him a potential candidate for thrombectomy. This is usually used within 6 hours of onset but if there is potential to salvage brain tissue, as shown by imaging such as CT perfusion or diffusion-weighted MRI sequences showing limited infarct core volume, then it can be considered up to 24 hours from onset (including wakeup strokes).

Thrombolysis with alteplase should only be given if it is administered within 4.5 hours of the onset of stroke symptoms. In this case, the stroke onset is likely outside this window and therefore thrombolysis should not be considered.

NICE has written guidance for 'Interventions for people with acute coronary syndromes or acute stroke or for acutely ill patients' where it is advised 'do not offer anti-embolism stockings for VTE prophylaxis to people who are admitted for acute stroke.'

Atrial fibrillation and stroke is a common combination. NICE recommends 'that people with disabling ischaemic stroke who are in atrial fibrillation are treated with aspirin 300 mg for the first 2 weeks before anticoagulation treatment is considered'.

Immediate initiation of statin therapy isn't recommended in acute stroke. There is an association with an increased risk of haemorrhagic transformation. Treatment should be delayed by 48 hours after stroke symptom onset (as per BNF and NICE).

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A 66-year-old male is brought to the emergency department by ambulance complaining of an abrupt onset of bilateral weakness progressing over the last 4 hours. He has a past medical hypertension and hypercholesterolaemia. His medications include amlodipine, ramipril and atorvastatin. He does not smoke cigarettes.

On examination, there is bilateral flaccid weakness of the lower limbs, with power 0/5 in all muscle groups. The patient is areflexic. There is loss of pain and temperature sensation from the level of the umbilicus to the feet. Proprioception and vibration sense is preserved.

Over the course of the next few weeks, the weakness becomes spastic in nature. The lower limbs develop hyperreflexia and the plantar reflexes are now noted to be upgoing.

What is the likely diagnosis?

A. Anterior spinal artery occlusion
B. Basilar artery occlusion
C. Cauda equina syndrome
D. Posterior spinal artery occlusion
E. Vitamin B 12 deficiency

A. Anterior spinal artery occlusion

Bilateral spastic paresis and loss of pain and temperature sensation - anterior spinal artery occlusion

Anterior spinal artery occlusion is the correct answer. The patient has risk factors for atherosclerosis (hypertension, hypercholesterolaemia) and this is a common cause of anterior spinal artery occlusion. An infarct of this artery causes bilateral weakness below the level of the lesion (in this case, the umbilicus suggests T10 as the level) associated with loss of pain and temperature sensation.

This anterior cord syndrome is caused by injury to the spinothalamic and corticospinal tracts. Initially there were will be a flaccid paralysis but this will become more reflective of an upper motor neuron lesion with development of spastic paraparesis over the following days to weeks. Basilar artery occlusion is incorrect. This causes 'locked-in syndrome' which causes complete paralysis of voluntary muscles, except for the muscles that control the eyes.

Posterior spinal artery occlusion is incorrect. This causes a dorsal cord syndrome characterized by loss of proprioception and vibration.

Vitamin B 12 deficiency is incorrect. This causes subacute combined degeneration of the spinal cord if severe. However, the onset would be more insidious and there would be mixed upper motor and lower motor neuron signs in the lower limbs that would occur concurrently e.g. absent ankle reflexes but upgoing plantar reflexes.

Cauda equina syndrome is incorrect. This causes lower motor neuron weakness. It would not progress to a spastic paraparesis.

Question 54 of 409

A 78-year-old male with a background of atrial fibrillation, ischaemic heart disease, hypertension is brought to hospital by his wife who is concerned about a recent deterioration in his health. For the last few weeks his wife has noticed that the patient has been more unsteady on his feet which has resulted in falls around the house. His wife also informs you that the patient has been more forgetful and less interested in his hobbies than usual. When you ask the patient about his symptoms, his main concern is that he has recently developed problems with urinary incontinence which he finds embarrassing.

As part of the investigation into this patient's condition, a CT scan of the patient's head is requested.

What is the most likely finding?

A. Diffuse cerebral atrophy with enlargement of cortical sulci and ventricles
B. A hyper-dense bi-convex extra-axial collection beneath the squamous part of the temporal bone
C. Small areas of low density in the distribution of the middle cerebral artery, suggestive of old lacunar infarcts
D. A concave-shaped extra-axial collection with increased attenuation
E. Enlarged third and lateral ventricles, disproportionate to the enlargement of the cortical sulci

E. Enlarged third and lateral ventricles, disproportionate to the enlargement of the cortical sulci

Normal pressure hydrocephalus neuroimaging findings: ventriculomegaly in the absence of, or out of proportion to, sulcal enlargement

The patient's history of atrial fibrillation places him at increased risk of stroke and so it may be tempting to attribute his deterioration to cerebrovascular disease. Another pitfall with this question is the report of falls at home, which may lead to a suspicion that the patient has developed a subdural haematoma (SDH). However, it is important to appreciate that his symptom of ataxia pre-dates his tendency to fall. The critical part of this question is that this patient's presenting complaints of ataxia, dementia and urinary incontinence are the classical triad of clinical findings associated with normal pressure hydrocephalus. As such, a CT scan of his head would be expected to show ventricles which are dilated out of proportion to the enlargement of his sulci.

Question 55 of 409

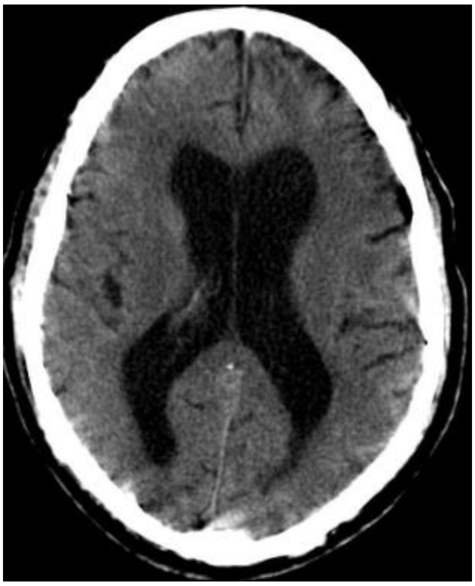
A 62-year-old man is reviewed in the neurology clinic due to a gait disorder for the last 2 months. His wife tells that he has become forgetful and has difficulty continuing his job. The patient has a history of hospital admission 4 months back due to bacterial meningitis requiring intravenous antibiotics. Past medical history is significant for hypertension well-controlled on amlodipine 10 mg once daily.

A lumbar puncture is performed and an interpretation of the CSF analysis is shown below:

Appearance	Clear, colourless	(Clear, colourless)
Opening pressure	6 mmHg	(8-16)
Cells	4 cells /mm	(0-4)
Glucose	3.9 mmol/L	(2.5-4.4)
Protein	50 mg/100 mL	(20-60)

Blood glucose	5.6 mmol/L

A CT head is performed, as shown below:



What is the most appropriate treatment option for this patient?

A. Intravenous corticosteroids
B. Intravenous mannitol
C. Oral memantine
D. Therapeutic lumbar puncture
E. Ventriculoperitoneal (VP) shunting

E. Ventriculoperitoneal (VP) shunting

The scenario describes a patient with normal pressure hydrocephalus, for which the definitive treatment is ventriculoperitoneal shunting . The patient has gait apraxia and dementia that started 2 months after meningitis. The CT scan shows enlarged frontal, temporal, and occipital horns of the lateral ventricles disproportionate to the sulci, which appear normal in size. These CT findings when combined with the patient's history suggest the diagnosis of normal pressure hydrocephalus (NPH) in this scenario. NPH can occur following meningitis or subarachnoid haemorrhage due to reduced CSF absorption by arachnoid villi. The classical triad of NPH includes gait apraxia, dementia, and urinary incontinence. LP reveals a normal or low opening pressure which is also hinted at in this question. The definitive treatment for NPH is VP shunting. Symptoms of NPH including dementia are reversed with successful VP shunting in 60-90 % of the patients. 10% develop complications like postural headache, subdural effusion, shunt infection, and bacterial meningitis.

The history of gait problems and dementia in this scenario may suggest Alzheimer's disease for which memantine is the most appropriate treatment option. However, the acute onset of symptoms and normal sulci on CT scan in this scenario rule out Alzheimer's disease. Neuroimaging in Alzheimer's reveals dilated ventricles with enlarged sulci and decreased volume gyri.

Intravenous mannitol is unlikely to benefit this patient with NPH. It is an osmotic diuretic and helps in decreasing intracranial pressure.

There is no role of intravenous corticosteroids in treating NPH. Corticosteroids are used to reduce hearing loss and neurological sequelae in meningitis.

Therapeutic lumbar puncture (LP) treats conditions with increased intracranial pressure without a space-occupying lesion. It is an important treatment option for idiopathic intracranial hypertension. This patient with NPH requires continuous drainage of CSF which is not possible via LP. A lumbar peritoneal (LP) shunt may be used as an alternative to VP shunt to treat NPH.

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A 76-year-old gentleman with diabetes mellitus was referred to a specialist memory clinic by his family doctor. His wife was concerned as he had been more forgetful recently and had developed urinary incontinence. His family doctor had treated him with a course of trimethoprim for a urinary tract infection but his symptoms had persisted.

On examination this gentleman smelt strongly of urine. He had a broad-based, unsteady gait. Examination of his cardiovascular and respiratory systems was unremarkable but on abdominal examination there was suprapubic tenderness. He was orientated to place but scored only 22/30 on a Mini Mental State Examination (MMSE).

Urinalysis:

- Blood negative
- Protein Trace
- Nitrites -
- White cells +
- Microscopy 50 white cells seen, bacteria +
- Cultures Mixed growth, please send repeat sample

Magnetic Resonance Imaging (MRI) of the brain: 'Disproportionately enlarged ventricular system compared with the degree of sulcal atrophy.'

Lumbar Puncture (LP):

Opening pressure	18 cmH20 (12 20)
Appearance	Clear and colourless
Red cells	4
White cells	1 (0 5)
Gram stain	No organisms seen
Culture	No organisms seen
Protein	30 mg/100ml (15 60)
Glucose	98mg/100ml (50 80)
Virology	No viruses detected on PCR
Cytology	No abnormal cells seen
Timed 10 metre walk pre-LP	15 seconds
Timed 10 metre walk post-40 mL CSF drainage	12 seconds

Given the underlying diagnosis what is the definitive treatment of choice?

A. Repeated therapeutic lumbar punctures
B. Acetazolamide
C. Referral for a ventriculo-peritoneal shunt
D. Vitamin B12 and folate replacement
E. Referral for lumbar drain insertion

C. Referral for a ventriculo-peritoneal shunt

This gentleman presents with all three components of the classic triad of Normal Pressure Hydrocephalus (NPH): urinary incontinence, ataxia and memory impairment. He may also have a co-existent urinary tract infection but this alone cannot explain his presentation. Further evidence to support a diagnosis of NPH includes the MRI scan, which shows ventricular enlargement, and the improvement in his 10 metre walk time following therapeutic CSF drainage.

This gentleman needs a referral to the neurosurgical team for consideration of a definitive surgical procedure. There are alternative surgical options and the specifics of each particular case will dictate which approach is used. The most commonly performed procedure is ventriculo-peritoneal shunting as this has a lower failure rate than alternative procedures.

Repeated therapeutic lumbar punctures (LP) can be performed but this approach has fallen out of favour as it is unpopular with patients and exposes them repeatedly to an invasive procedure with associated risks.

Acetazolamide is a diuretic used to treat Idiopathic Intracranial Hypertension and does not have a role in NPH.

Vitamin B12 and folate replacement is not indicated in this case although vitamin B12 deficiency can cause memory impairment and walking problems (subacute combined degeneration of the cord).

A lumbar drains diverts CSF from the lumbar subarchnoid space to the outside world. It can be useful in determining whether or not a patient may respond well to a shunting procedure but due to the high risk of infection it is not in itself a definitive treatment for NPH.

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Vincent, 28, has treatment resistant schizophrenia, with his usual symptoms being auditory hallucinations and persecutory delusions. He was recently prescribed clozapine, fluoxetine and lactulose. He has been complaining of constipation recently, but now presents to the emergency department with acute abdominal pain and vomiting. On examination abdomen is distended.

What is the most likely cause?

A. Intestinal obstruction
B. Appendicitis
C. Constipation
D. A bezoar
E. Ingestion of foreign object

A. Intestinal obstruction

One of the most common side effects of clozapine is constipation/intestinal obstruction

The most likely cause of this patients presentation is intestinal obstruction. Intestinal obstruction is one of the more under-recognised complications of clozapine therapy, yet one of the more serious. Gastric hypomotility is common in patients who are treated with clozapine, and its presentation can range from a simple constipation to more severe conditions such as intestinal obstruction, bowel ischaemia and necrosis.

The important pieces of information to consider when answering this question are the recent prescription of clozapine, the presence of constipation previous to current presentation, and his physical presentation of acute abdominal pain and vomiting with distension. When answering a question such as this, recent prescriptions of medications prior to the deterioration of their physical condition should raise the thought that maybe one of the medications is causing the symptoms. In this scenario, the patient has recently been prescribed clozapine, fluoxetine and lactulose. Therefore, considering each of these medications and their side effects is a must.

The patients presentation, and recent past medical history, provides a good indication of what could be the underlying issue. The patient has presented with acute abdominal pain and vomiting. When you consider this with the recently experienced constipation, it becomes clear that this patient is likely suffering from an obstruction, as opposed to the other options on offer.Constipation would be an inappropriate answer to this question. Although he has had constipation recently, and abdominal pain could be a presentation of constipation, vomiting is not generally observed in constipation. This would means that this diagnosis is less likely.

Bezoars are indigestible masses that become trapped in the gastrointestinal tract. They can occur when individuals consume a variety of items, including hair, soil, chewing gum etc. These items form a mass, which ultimately becomes lodged in the gastrointestinal tract, often requiring surgical intervention to relieve the obstruction. The symptoms that the patient in this scenario has experienced could be explained by a bezoar, however, we do not have any evidence that he consumes any items that may lead to a bezoar. Furthermore, the recent commencement of clozapine means that we cannot select a bezoar as the most likely cause of this patients symptoms above intestinal obstruction. Similarly, we do not have any evidence that the patient ingests foreign objects, meaning that this cannot be classed as a likely cause in this question.

Appendicitis could explain the acute presentation of this patient. However, the fact that the patient experienced constipation prior to him developing acute abdominal pain and vomiting means that appendicitis is less likely.

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An 84-year-old male is reviewed in clinic after a 7-month history of progressive confusion, unsteadiness on his feet and new urinary incontinence. He had previously minimal past medical history and continued to volunteer at his local charity shop, taking ramipril alone for hypertension. On examination, his abbreviated mental test score was 2/10, his gait was wide based and ataxic. A mini-mental state examination scores 17/30. His urine dip demonstrated no positive findings and serum results were unremarkable. Although there has been no history of head trauma, a CT head was performed, demonstrated no acute haemorrhages or infarcts. A subsequent MRI demonstrated large ventricles with periventricular white matter changes. Lumbar puncture demonstrated acellular cerebrospinal fluid with no organism growth. Opening pressure was 16 cm H20. You arrange a CSF infusion test, demonstrating raised CSF outflow resistance.

Which treatment is most appropriate?

A. Therapeutic repeat lumbar punctures
B. Ventriculoperitoneal shunt
C. Donepezil
D. Acetazolamide
E. Aspirin 300mg

B. Ventriculoperitoneal shunt

The history and examination are consistent with normal pressure hydrocephalus, with the triad of new onset dementia, urinary incontinence and gait disturbance. The key points in the investigations indicate the suitability of the patient for a ventriculoperitoneal shunt: the raised CSF resistance on CSF infusion test indicates impaired CSF absorption and correlates towards surgical improvement. In addition, prominent gait disturbance also points towards an increased likelihood of surgical success.

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A 78-year-old male presents with a one-year history of progressive unsteadiness on walking. He had previously been extremely fit and healthy, walking around 2 miles to the shops every day and only retired as a publicist 3 years ago. He underwent radiotherapy for localised squamous cell carcinoma of his vocal cords 18 months ago but otherwise had no other past medical history. He admits to having drunk 'more than he should have' while working in the city but says he has since cut down to moderate levels. He stopped smoking 5 years ago, with a 30 pack year history. Over the past 3 months, he has become incontinent of urine and has to rely on pads, which he is greatly embarrassed by.

On examination, he has a shuffling gait in his lower limbs with good arm swing. He is markedly slowed and takes 120 seconds to walk 20 metres. He turns around 180 degrees in 6 steps with no resting tremor, rigidity or bradykinesia. Examination of his tone, power, sensation, coordination and reflexes are all unremarkable. His voice is quiet and whispering. His cranial nerves are normal with a full range of eye movements. An abbreviated mental test scores 9/10, a Montreal cognitive assessment (MOCA) scored 29/30. His initial blood tests are as follows:

Hb	94 g/l
MCV	103.3 fl
Platelets	232 * 10 /l
WBC	6.3 * 10 /l
Na	139 mmol/l
K	4.9 mmol/l
Urea	7.5 mmol/l
Creatinine	98 µmol/l
CRP	2 mg/l
Vitamin B12	327ng/l
Folate	5.2 nmol/l

An MRI head is performed, demonstrating diffuse mild microangiopathic changes with prominently dilated lateral and third ventricles. No intracranial masses are noted. You perform a lumbar puncture, with the patient lying in the left lateral position using a 22G spinal needle and obtain cerebrospinal fluid with the first pass. His opening pressure is 16.6 cmH2O.

What is the most appropriate course of action?

A. Remove 30mls cerebrospinal fluid and refer to neurosurgeons
B. Prescribe urgent intravenous pabrinex
C. Urgent urological referral for urodynamic studies +/- flexible cystoscopy
D. Prescribe Sinemet 250 with anti-emetic and re-measure walking time
E. Urgent CT angiography and prescribe aspirin 300mg

A. Remove 30mls cerebrospinal fluid and refer to neurosurgeons

There are two of three features of the classic triad of gait disturbance, urinary incontinence and cognitive impairment, suggestive of normal pressure hydrocephalus (NPH). The normal opening pressure and dilated prominent ventricles lend further support to this diagnosis. Mild microangiopathic disease is not uncommon in this age group and potentially could cause gait disturbances; there is no indication of an acute ischaemic stroke secondary to embolic events however, urgent vascular imaging and aspirin loading would thus be inappropriate. Despite the previous alcohol history, the patient's vitamin B12 and folate are within normal range with no neuroimaging features of degeneration, intravenous Pabrinex is unlikely to resolve these symptoms. Although there may be a primary urological cause for the patient's urological symptoms, his gait features are not easily explained. The patient does not display any 'hard signs' of Parkinsonism to warrant a Sinemet trial.

The definitive treatment for normal pressure hydrocephalus is neurosurgical implantation of a ventricular shunt, most commonly into the peritoneum. Of the three main features of NPH, gait apraxia is the most commonly improved feature with shunting, while dementia and cognitive impairment rarely improve after surgery. Despite the normal pressure, removal of a relatively significant amount of CSF and repeat of the patients walking time is a useful clinical predictor of whether the patient will improve after surgery.

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A 35-year-old woman is referred to a headache clinic with frequent migraines over the last 9 months. They occur unpredictably, around 3 times a month. Her past medical history includes eczema, brittle asthma and depression. She is not sexually active.

She is keen on something to reduce the frequency of her migraines. In the past, she has tried topiramate but stopped as she felt it caused her to get eye discomfort.

What is the most appropriate management option?

A. Candesartan
B. Pizotifen
C. Propranolol
D. Re-trial topiramate
E. Zolmitriptan

A. Candesartan

Candesartan may be considered for migraine prophylaxis if other first-line options either don't work or are contradicted

Either topiramate or propranolol is usually used first-line for prophylaxis of migraine. When standard treatments are not appropriate then other treatment options may be considered by specialists, but these are outside the remit of NICE guidelines. Candesartan is recommended by the British Association for the Study of Headache guidelines.

Pizotifen is no longer recommended as adverse effects such as weight gain and drowsiness are common.

In this case, topiramate has caused side effects on previous use. Eye discomfort is a recognised uncommon side effect and can rarely cause eye inflammation. Hence, this should be avoided.

Propranolol is best avoided given the risk of bronchospasm, listed as an uncommon side effect in the BNF. This is because this patient is asthmatic and particularly given that it is brittle asthma.Zolmitriptan can be used as a type of 'mini-prophylaxis' for women with predictable menstrual migraine. However, this patient suffers from unpredictable migraines so this is not likely to be effective.

Question 61 of 409

A 38-year-old man with severe ulcerative colitis presents to his GP with a red right eye that has developed over the last 24 hours. He is finding it uncomfortable to move his eye in any direction but feels his vision is unchanged. On examination his left eye is normal, but his right eye is erythematous, watery, and there is asymmetric miosis. During the examination, he asks you to turn down the lights as he finds them uncomfortable.

Which of the following is the most appropriate treatment for this man's red-eye?

A. Supportive management
B. Topical atropine
C. Topical corticosteroids
D. Topical cyclopentolate and topical corticosteroids
E. Topical pilocarpine

D. Topical cyclopentolate and topical corticosteroids

Anterior uveitis is most likely to be treated with a steroid + cycloplegic (mydriatic) drops

The painful eye movements, lacrimation and irregular small pupil are strongly indicative of anterior uveitis. Uveitis may be idiopathic but is often associated with HLA-B27-related diseases, such as inflammatory bowel disease (IBD). Uveitis occurs in 0.5%-3% of patients with IBD and is often bilateral, but not in this case. In terms of extraintestinal manifestations of IBD, uveitis is less common than episcleritis. However, episcleritis is not typically painful and does not present with photophobia. If episcleritis were the diagnosis, then supportive management would be appropriate.

Anterior uveitis describes inflammation of the anterior portion of the uvea - the iris and ciliary body. Uveitis does not affect visual acuity unless it involves the posterior segment - the posterior vitreous, retina, choroid, retinal vasculature and optic nerve. Uveitis can lead to blindness and should be referred to an experienced ophthalmologist. Treatment is with topical corticosteroids and cycloplegic (mydriatic) drops. Topical corticosteroids are appropriate in anterior uveitis but note that intermediate and posterior uveitis usually require locally injected or systemic corticosteroids.Cyclopentolate is a muscarinic antagonist, and cycloplegic. It relieves the pain associated with uveitis as it dilates the pupil and prevents iridospasm. Atropine is another cycloplegic medication that can be used in anterior uveitis. Used alone it is less appropriate than the combination of cyclopentolate drops and steroids, which is the single best answer here.

Topical pilocarpine is a muscarinic receptor agonist and would cause miosis, worsening the situation here. It may be used in acute-angle glaucoma, another cause of acute red eye which would instead present with increased pain in the dark.

Question 62 of 409

A 70-year-old male presents with a 3-month history of left foot drop. He complains of having to lift his thighs higher than normal to accommodate this pathology. On examination, he has a high stepping gait. Power is normal in all movements except left ankle dorsiflexion (2/5) and eversion (2/5). Ankle inversion is intact (5/5), ankle jerks are present and plantars are downgoing. He reports reduced sensation on the dorsum of his foot.

What is the most likely diagnosis?

A. L5 radiculopathy
B. Common peroneal palsy
C. Functional neurology
D. Sciatic nerve compression
E. Lumbar plexopathy

B. Common peroneal palsy

'Foot drop' - ?common peroneal nerve lesion

The differential diagnosis of foot drop is a MRCP favourite. In this case, a number of features favour a common peroneal nerve palsy instead of a L5 radiculopathy: common peroneal nerve palsy patients normally have an intact ankle inversion and flexion of the big toe. Sensory loss is usually around the lateral aspect of the lower leg and the dorsum of the foot while L5 appears as a thin strip down the middle of the anterior lower limb, usually not affect the lateral lower leg. Ankle jerks are present in both, as the tibial nerve is branched from S1 and the sciatic nerve. There is little to suggest the involvement of multiple lumbar levels in this scenario. The most common causes of common peroneal nerve palsy are trauma or compression at the fibula head, classically by tight plaster casts.

Question 63 of 409

A 46-year-old woman presents to her GP with a neck lump. After examination and initial investigations, she is urgently referred to the local endocrine multidisciplinary team, which perform further scans and diagnose cancer of the thyroid.

Ultimately, she undergoes a thyroidectomy. The procedure itself is uneventful.

Post-thyroidectomy, she notices that her voice has become very hoarse, which was not a symptom prior to the surgery.

What is most appropriate next?

A. Acoustic analysis
B. Chest X-ray
C. Laryngeal electromyography
D. Laryngoscopy
E. Reassure and monitor

D. Laryngoscopy

Hoarseness following total thyroidectomy should be investigated with laryngoscope.

Hoarseness following thyroidectomy indicates damage to the recurrent laryngeal nerve. This is a relatively common complication of thyroidectomy surgery. The correct answer is laryngoscopy this should be performed if hoarseness develops, to assess for vocal cord paralysis and evaluate motility.

Acoustic analysis - that is, assessment of the change in voice itself - is insufficient. This may help to give an idea of the extent to which the hoarseness has affected speech, but more objective assessment is required via laryngoscopy.

Chest X-ray is inappropriate. This would not allow for assessment of the vocal cords. Laryngoscopy is required.

Laryngeal electromyography can be used to assess vocal cord paralysis and estimate prognosis.However, first-line, laryngoscopy is generally performed before any further investigation.

Reassurance and monitoring would be inappropriate. Further assessment is required here, given the new onset of hoarseness following thyroidectomy.

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A 40-year-old lady with myasthenia gravis is admitted under the medical team with pneumonia. She is also experiencing a flare of her myasthenia symptoms. She is treated with antibiotics, pyridostigmine and methylprednisolone begins to improve clinically. She is also given IV magnesium replacement to correct hypomagnesemia.

The next day her symptoms suddenly worsen and she develops profound weakness on exertion and new dysphagia requiring insertion of a nasogastric (NG) tube.

Which of the following drugs administered by the medical team is the most likely cause?

A. IV magnesium
B. Co-amoxiclav
C. Low molecular weight heparin
D. Cyclizine
E. Naproxen

A. IV magnesium

Don't given IV magnesium in myasthenia as can precipitate a crisis Important for me Less important The following drugs may exacerbate myasthenia:

- Magnesium
- Penicillamine
- Quinidine
- Procainamide
- Beta-blockers
- Lithium
- Phenytoin
- Antibiotics: gentamicin, macrolides, quinolones, tetracyclines

Question 65 of 409

A 56-year-old man presents to the emergency department complaining of weakness and shortness of breath. His symptoms started initially with a tingling sensation and weakness in his feet and hands about 1 week ago. This has got progressively worse and now he is struggling to lift his arms and cannot walk. Over the last 24 hours, he has become short of breath.

He has a past medical history of asthma and anxiety. He felt that he had a mild asthma flare alongside a cold around 3 weeks ago but recovered well without antibiotics or steroids. Usually, his asthma is well-controlled.

An arterial blood gas reveals type 2 respiratory failure with a pH of 7.33. He is commenced on non-invasive ventilation.

What is the most appropriate management?

A. Intravenous botulinum antitoxin
B. Intravenous immunoglobulin
C. Intravenous methylprednisolone
D. Intubation and ventilation
E. Plasma exchange

B. Intravenous immunoglobulin

IVIG is generally used first-line for Guillain-Barre syndrome

The ascending neurological impairment coupled with a recent upper respiratory tract infection is highly suggestive of Guillain-Barre syndrome. In this case, this has progressed to the extent of affecting the respiratory muscles. Intravenous immunoglobulin or plasma exchange can be used in treatment. Immunoglobulin is as effective as plasma exchange, is better tolerated, and is easier to administer. Therefore, immunoglobulin is generally used first-line for Guillain-Barre syndrome where patients require immunomodulatory therapy.

Steroids and immunosuppressants have not been shown to be beneficial in Guillain-Barre syndrome. Therefore, intravenous methylprednisolone is incorrect. Immunoglobulin is better tolerated and easier to administer than plasma exchange. Therefore plasma exchange would not be the first-line management choice.

Intravenous botulinum antitoxin is the treatment for botulism. This typically presents with a descending weakness, starting in the cranial nerves. Given this patient has ascending weakness this diagnosis is less likely and so the treatment is not correct.

For patients with severe respiratory failure, intubation and ventilation may be required. Given that this patient is only mildly acidotic it can be inferred that type 2 respiratory failure is not too severe, especially given it is acute so there will not be significant metabolic compensation. Intubation and ventilation carry risks and are not warranted at present. If there was poor response to non-invasive ventilation then this would need to be re-considered.

Question 66 of 409

A 54-year-old man presents to the emergency department with reduced mobility. He describes an absence of feeling in his lower limbs bilaterally that makes him feel very unsteady on his feet. His past medical history includes type 2 diabetes, hypercholesterolaemia and hypertension, for which he takes metformin, sitagliptin, atorvastatin and ramipril.

On examination, he has 5/5 power in his upper limbs. There is increased tone and rigidity in his lower limbs, although he is able to weight bear. His proprioception is intact but there is an absence of pain and temperature sensation.

What is the most likely diagnosis?

A. Anterior spinal artery occlusion
B. Brown-Sequard syndrome
C. Central cord syndrome
D. Primary lateral sclerosis
E. Subacute combined degeneration of the spinal cord

A. Anterior spinal artery occlusion

Bilateral spastic paresis and loss of pain and temperature sensation - anterior spinal artery occlusion

This patient has anterior spinal artery occlusion, affecting the lateral corticospinal and spinothalamic tracts. Symptoms of anterior spinal artery occlusion include bilateral spastic paresis and loss of pain and temperature sensation, as described in this scenario. This patient has significant risk factors for arterial occlusion including diabetes, hypertension and hypercholesterolaemia.

Brown-Sequard syndrome refers to hemisection of the spinal cord, usually secondary to traumatic injury. Symptoms include ipsilateral spastic paresis below the level of the lesion along with ipsilateral loss of proprioception and vibration sensation and contralateral loss of pain and temperature sensation. The absence of these symptoms and history of trauma make BrownSequard syndrome less likely.Central cord syndrome is a very common type of incomplete spinal cord injury, usually secondary to trauma (e.g. following a hyperextension injury in a patient with cervical spondylosis). Rather than a complete loss of movement or sensation, there tends to be a partial pattern of motor and sensory loss with greater motor impairment seen in the upper limbs and variable sensory loss below the level of the injury.

Primary lateral sclerosis, or motor neuron disease, is a neurodegenerative condition affecting the upper and lower motor neurons, causing a combination of upper motor neuron signs (increased tone) and lower motor neuron signs (muscle fasciculations and weakness). The combination of symptoms in primary lateral sclerosis is purely motor, unlike this patient's presentation that also has a sensory deficit, making primary lateral sclerosis unlikely.

Subacute combined degeneration of the spinal cord is commonly related to vitamin B12 deficiency, causing degeneration of the lateral corticospinal tracts and the dorsal columns. Symptoms include bilateral spastic paresis similarly to this patient. However, sensory symptoms in subacute combined degeneration of the spinal cord are secondary to dorsal column loss and include the absence of proprioception and vibration sensation rather than pain and temperature sensation.

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A 42 year old caucasian man presents to his GP because of concerns about weight gain. He has put on 10kg in weight in the past 6 months but has been eating and exercising the same amounts for the past few years. The GP decides to do some further investigations and finds the following abnormalities. He tells you that he was on a medication for schizophrenia but this was changed one year ago due to abnormal movements. In the past year he has also been given medications to help with symptoms of nausea and has recently started medication for gynaecomastia.

ECG: sinus rhythm 84/min QTC 462ms

Hb	13.8 g/dl
Platelets	154 * 10 /l
WBC	3.8 * 10 /l
Neuts	1.5 * 10 /l
Lymphs	1.0 * 10 /l
Eosinos	1.2 * 10 /l
Fasting glucose	11.9 mmol/l
Prolactin	264 mu/l

Which drug is most likely to have caused these abnormalities?

A. Haloperidol
B. Clozapine
C. Bromocriptine
D. Domperidone
E. Ondansetron

B. Clozapine

One of the most common side effects of clozapine is constipation/intestinal obstruction

Clozapine is an atypical antipsychotic that is used in treatment resistant schizophrenia and also if there has been tardive dyskinesia on a previous antipsychotic which is described in this question. Clozapine can cause neutropaenia, eosinophilia and prolongation of the QTc as well as increased weight gain and reduced insulin tolerance which are all seen in this individual

Haloperidol can also cause QTc prolongation. It is less likely to cause weight gain and reduced glucose tolerance than clozapine and it is not associated with neutropaenia or eosinophilia. Haloperidol more commonly causes tardive dyskinesia than Clozapine and it is possible that he was on this before switching due to hyperprolactinaemia and tardive dyskinesia

Bromocriptine is a treatment designed to reduce prolactin levels and does not cause reduced glucose tolerance or neutropaenia.

Domperidone can cause prolonged QTc but would not explain any of the other abnormalities.

Ondansetron can cause prolongation of the QTc but does not cause any of the other abnormalities described here

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A 68-year-old man presents with back pain. He was recently diagnosed with peripheral neuropathy however no cause was identified. He has no other past medical history and takes no regular medicines. On examination he has tenderness of his lumbar spine. You also note hepatosplenomegaly and hyperpigmentation of his skin.

Blood results are as follows:

Hb	105 g/l	Na	136 mmol/l
Platelets	110 * 10 /l	K	5.1 mmol/l
WBC	10.2 * 10 /l	Urea	14.6 mmol/l
Neuts	8.1 * 10 /l	Creatinine	162 µmol/l
Lymphs	1.1 * 10 /l	CRP	24 mg/l
Eosin	0.2 * 10 /l	Calcium (corrected)	2.85 mmol/l

Serum electrophoresis and immunofixation was also performed:

Monoclonal band	45 g/l
Immunofixation	IgG light chain spike

A whole body MRI scan is performed:

Whole body MRI	Osteoblastic lesion present in L4 vertebrae

What is the most likely diagnosis?

A. Osteosarcoma
B. Plasmacytoma
C. POEMS syndrome
D. Waldenstrom macroglobulinemia
E. Monoclonal gammopathy of unexplained significance

C. POEMS syndrome

POEMS syndrome is a rare paraneoplastic syndrome caused by a clone of abnormal plasma cells. It is associated with polyneuropathy, organomegaly, endocrinopathy, M-protein band from a plasmocytoma, and skin pigmentation

The patient has anaemia, renal impairment, and hypercalcaemia. This triad of features should raise suspicion of multiple myeloma, especially in elderly patients who present with back pain. The monoclonal band of plasma cells confirms a plasma cell dyscrasia. The differential for plasma cell dyscrasias include myeloma, monoclonal gammopathy of unexplained significance, plasmacytoma, POEMS syndrome and Waldenstrom macroglobulinemia.

The features of organomegaly, hyperpigmentation of the skin, and a single osteoblastic lesion in the spine should raise concern of POEMS syndrome. POEMS syndrome is a rare paraneoplastic syndrome caused by a clone of abnormal plasma cells. It is associated with polyneuropathy, organomegaly, endocrinopathy, M-protein band from a plasmacytoma, and skin pigmentation. It is therefore the most likely diagnosis in this patient.

Waldenstrom macroglobulinemia is a type of non-Hodgkin lymphoma (NHL) associated with very high levels immunoglobulin M (IgM). IgM is a very large molecule and when present in excess can result in hyperviscosity syndrome. This can also result in organomegaly, peripheral neuropathy and a variety of other features. In this case, the presence of an IgG monoclonal band suggest an alternative diagnosis.

The presence of end-organ damage rules out the diagnosis of monoclonal gammopathy of unexplained significance.

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A 62-year-old man presents with an episode of collapse. He states that the episode of the collapse was preceded by the worst headache of his life. He denies any recent head injuries. On examination, you note nuchal rigidity and photophobia. Observations are within normal limits.

A CT brain is arranged: CT brain (non-contrast) Hyperdense material filling the subarachnoid space

What is urgently required?

A. CT angiogram
B. Digital subtraction angiogram
C. Endovascular coiling and neurosurgical clipping
D. Lumbar puncture
E. MRI angiogram

A. CT angiogram

In non-traumatic subarachnoid haemorrhages, a CT angiogram should be performed to identify intracranial aneurysms

CT angiogram is correct. A non-contrast CT has confirmed a SAH. The patient does not have a history of trauma and as such an aneurysmal bleed is at the top of the differential diagnosis. Therefore an urgent CT angiogram is required to identify the causal pathology, define the anatomy of the aneurysm, and plan specific treatment.

Digital subtraction angiogram is incorrect. Digital subtraction catheter angiography remains the gold standard for diagnosis and characterisation of vascular abnormalities, and in many centres, even if the causative lesion is identified on MRA or CTA and it is thought to require surgical management, a catheter study is carried out. However, in the first instance, a CT angiogram is required.

Endovascular coiling and neurosurgical clipping is incorrect. Although this may well be required for definitive management of an aneurysmal bleed, in the first instance the lesion needs to beidentified and characterised by a CT angiogram.

Lumbar puncture is incorrect. A lumbar puncture is only required if the initial CT brain is negative. In this case, the CT demonstrates clear evidence of a SAH.

MRI angiogram is incorrect. Although MRI is helpful to assess underlying brain parenchyma, MRI angiograms (MRA) are not as good as CT angiograms. Unlike CT angiography, MRA is not able to see and capture images of calcium deposits within the blood vessels. MRA evaluation of small vessels, in particular, may be difficult.

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An 82-year-old woman is brought to see you by her daughter. They are concerned as she has had a tremor for several years that has been worsening and is feeling slightly more forgetful and slowed down in the past 2 months.

On examination, there is a relatively large amplitude tremor on finger-nose testing which is worse on the left side and improves when moved passively. Gait is normal and there is no lower limb involvement. A 10-point abbreviated mental test score is 10/10.

Given the likely diagnosis, what would be the treatment of choice?

A. Dopamine agonists
B. Primidone
C. Propranolol
D. Quinine sulphate
E. Rivastigmine

C. Propranolol

Propranolol is the treatment of choice for essential tremor

The most likely diagnosis here is essential tremor given the worsening of the tremor on intentional movement and improvement when the muscles are relaxed and moved passively by the examiner. In essential tremors, the upper limbs and head/neck are most commonly affected. If there is lower limb involvement alternative pathology should be suspected.

Propranolol is the correct answer. This is the first-line treatment for essential tremors.

Primidone is incorrect. This would usually form second-line management, or treatment if propranolol was contraindicated.

Dopamine agonists is incorrect. These would form part of the management of Parkinson's disease. In this case, we have a tremor that is worsened by movement rather than a resting tremor, gait is normal, and the lower limbs are not affected. Parkinson's disease is therefore less likely.

Rivastigmine is incorrect. This is a cholinesterase inhibitor used in the treatment of Alzheimer's dementia, dementia associated with Parkinson's disease, and Lewy body dementia. In this case, the patient did notice some deterioration in her cognitive function, but the initial mental screening was normal. It may be reasonable to investigate this further with more in-depth mental testing, blood testing, and consideration of a CT head. An MMSE, for example, helps us grade dementia as being mild, or more severe. According to the ICD-10 classification, a decline in memory should have occurred over at least 6 months before a diagnosis of dementia is determined. Given that the symptoms of cognitive decline have been recent - over the past 2 months - it would be acceptable to take a 'watch and wait' approach after risk assessing the symptom severity.

Quinine sulphate is incorrect. This medication is used to treat malaria but also can be used in the management of leg cramps. Lower limb symptoms are not present here, and in fact, the most recent drug and safety guidance is that it should not be prescribed routinely for leg cramps unless very severe or frequent, where organic causes have been ruled out, and where nonpharmacological measures have been ruled out (e.g. exercises, passive stretching) as there is a risk of severe thrombocytopenia and impact on QT interval.

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A 50-year-old woman attends an annual diabetic retinopathy screening appointment. She has a past history of type 1 diabetes mellitus. She reports a gradual decrease in vision in her left eye over the past year.

Examination reveals retinal neovascularisation and formation of fibrous tissue anterior to the retinal disc. The macula appears normal. Her recent HbA1c level is 48 mmol/mol. She is advised that she will need pan-retinal laser photocoagulation (PRP).

In addition to PRP, what is the most appropriate treatment option for this patient?

A. Intravenous aflibercept
B. Intravitreal ranibizumab
C. Optimise glycaemic control
D. Screening for cataract
E. Vitreoretinal surgery

B. Intravitreal ranibizumab

Intravitreal VEGF inhibitors may be used in addition to panretinal laser photocoagulation to treat proliferative diabetic retinopathy

Intravitreal VEGF inhibitors should be used in combination with PRP for the treatment of patients with proliferative diabetic retinopathy. The scenario describes a patient with proliferative diabetic retinopathy (neovascularisation and fibrous tissue on the retina) for which she is already advised PRP. In addition to PRP, intravitreal ranibizumab slows the progression of diabetic retinopathy and improves visual acuity. The best effective treatment of proliferative diabetic retinopathy is PRP plus intravitreal VEGF inhibitors.

Intravitreal, not intravenous, aflibercept is used in treating proliferative diabetic retinopathy. Aflibercept is an anti-vascular endothelial growth factor (anti-VEGF) and its intravenous preparation has a role in chemotherapies.

The most important step in managing diabetic retinopathy is to optimise glycaemic control. However, the patient in this scenario has current HbA1c within the limits which implies goodcontrol already.

Screening for cataracts is an incorrect response because, although important, in a diabetic patient, cataract screening is irrelevant in this scenario.

The fundoscopy in this scenario does not show any vitreous haemorrhage - therefore, vitreoretinal surgery like vitrectomy is not a suitable treatment option here. Vitrectomy is for severe proliferative diabetic retinopathy with vitreous haemorrhage.

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A 40-year-old man presents to the clinic with a three-day history of left-sided headache. He reports tearing in his left eye and pain on eye movement. Examination reveals periorbital swelling and reduced sensation in the distribution of the ophthalmic nerve on the left side. His past medical history is unremarkable, except for a recent visit to an ENT clinic for sinusitis. An MRI of the brain is pending.

What is the most appropriate treatment option for this patient?

A. Heparin, Indomethacin, and prednisolone
B. Heparin, indomethacin, and metronidazole
C. Heparin, prednisolone, and metronidazole
D. Heparin, vancomycin, ceftriaxone, and metronidazole
E. Prednisolone, ceftriaxone, and vancomycin

D. Heparin, vancomycin, ceftriaxone, and metronidazole

Unilateral headache, periorbital inflammation, ophthalmoplegia → ? cavernous sinus thrombosis

The correct response is heparin, vancomycin, ceftriaxone, and metronidazole because the clinical presentation suggests a diagnosis of cavernous sinus thrombosis (CST) in this patient. Features such as unilateral headache, painful ophthalmoplegia, and periorbital swelling are indicative of CST. Additionally, the involvement of the ophthalmic division of the trigeminal nerve corroborates this diagnosis. The patient's history of sinusitis is relevant due to its association with septic CST, which necessitates treatment with intravenous antibiotics and anticoagulation. The appropriate antibiotic regimen includes vancomycin to target methicillin-resistant Staphylococcus aureus (MRSA), supplemented by ceftriaxone or cefepime. Metronidazole should be added when a sinus or dental infection is suspected, as indicated in this scenario. Antibiotic therapy should be continued for at least 3 weeks due to limited penetration into the thrombus. Early initiation of anticoagulation with a continuous infusion of heparin has been shown to improve outcomes in patients with septic CST.

Heparin, indometacin, and metronidazole constitutes an incorrect answer because it omits ceftriaxone, which is the first-line antibiotic for managing septic CST. Additionally, vancomycin isessential for MRSA coverage. Indomethacin is not indicated in treating CST; it is utilised for paroxysmal hemicranias characterised by unilateral headache, ptosis, and ophthalmoplegia but lacks efficacy in CST management.

The choice of heparin, indomethacin, and prednisolone is also incorrect. Prednisolone does not have a therapeutic role in CST; instead, steroids are primarily used in managing Tolosa-Hunt syndrome—an important differential diagnosis for CST—which involves idiopathic granulomatous inflammation of the cavernous sinus. In contrast to Tolosa-Hunt syndrome which has no preceding infection history typically seen with CST such as sinusitis described here antibiotics combined with heparin remain the cornerstone treatments for septic CST.

Selecting heparin, prednisolone, and metronidazole fails to provide an adequate treatment strategy because it lacks an appropriate first-line antibiotic therapy combination necessary for managing CST effectively; moreover vancomycin must be included for MRSA coverage Steroids do not play a part in treating CST

Lastly prednisolone ceftriaxone and vancomycin represents an inaccurate response due to the absence of heparin which is integral to the recommended treatment protocol

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A 47-year-old man is brought in by ambulance to the emergency department. He was found agitated and wandering the street by a member of the public. Collateral history from his relative reveals that he has a past medical history of hepatic steatosis, hypertension and asthma. His regular medications include amlodipine and Symbicort. He drinks up to 1 litre of vodka daily and has smoked 20 cigarettes per day for 30 years. There is no history of recreational drug use. He has not been seen by his family for 2-3 days before these events.

His observations are heart rate 111 beats per minute, blood pressure 170/94 mmHg, respiratory rate 23 /min, oxygen saturations 97% on air and temperature 37.2ºC. He is tremulous, diaphoretic and agitated. Cardiovascular, respiratory and abdominal examinations are otherwise unremarkable.

Shortly after he is examined, he has a seizure lasting 30 seconds that is self-terminating.

Blood tests

Hb	164 g/L	Male: (135-180) Female: (115 - 160)
Platelets	189 * 10 /L	(150 - 400)
WBC	6.0 * 10 /L	(4.0 - 11.0)
Na	129 mmol/L	(135 - 145)
K	4 mmol/L	(3.5 - 5.0)
Urea	8.9 mmol/L	(2.0 - 7.0)
Creatinine	77 µmol/L	(55 - 120)
CRP	3 mg/L	(< 5)
Glucose	4.1 mmol/L	(4 - 11)

A CT head is organized, which is reported as being normal.

What is the most appropriate medication choice to prevent further seizures?

A. Oral chlordiazepoxide
B. Intravenous glucose
C. Intravenous hypertonic saline
D. Intravenous levetiracetam
E. Intravenous pabrinex

A. Oral chlordiazepoxide

Chlordiazepoxide, lorazepam or diazepam are used in the treatment of delirium tremens/alcohol withdrawal

Chlordiazepoxide is the correct answer. Minor alcohol withdrawal symptoms such as tremor, anxiety and headache start approximately 6-12 hours after alcohol is stopped. This can progress to alcoholic hallucinosis after 12-24 hours and subsequently withdrawal seizures and delirium tremens from 48 hours onwards. The combination of tachypnoea, tachycardia, agitation and seizures suggest delirium tremens and alcohol withdrawal seizures as the diagnosis. The first-line treatment for delirium tremens and to prevent further seizures is chlordiazepoxide. The Clinical Institute Withdrawal Assessment for Alcohol (CIWA) score is used for the management of alcohol withdrawal in hospital. It is a ten item scoring system where higher total scores correspond to a greater severity of alcohol withdrawal. Depending on the score, specific management decisions are suggested, such as the administration of benzodiazepines.

Intravenous glucose is incorrect. The patient's glucose is within the normal range, albeit at the lower end, making this an unlikely cause of seizures.

Intravenous hypertonic saline is incorrect. While his sodium is mildly low at 129, this value is not typically low enough to cause seizures.

Intravenous levetiracetam is incorrect. While this is an effective anti-seizure medication, it is not the most appropriate choice given the underlying diagnosis.

Intravenous pabrinex is incorrect. This will not prevent or terminate seizures but is used to prevent the development of Wernicke's encephalopathy.

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A 31-year-old patient is referred to the neurology outpatient department with recurrent migraines. These occur 4-5 times a month. She has a past medical history of asthma. She has previously trilled amitriptyline, riboflavin, and acupuncture but unfortunately, these have been ineffective.

At the time of review, the examination is unremarkable.

What is the best option for preventing the recurrence of her headaches?

A. Candesartan
B. Ibuprofen
C. Medical cannabis
D. Propranolol
E. Sumatriptan

A. Candesartan

Candesartan may be considered for migraine prophylaxis if other first-line options either don't work or are contradicted

Candesartan is correct. This patient presents with recurrent migraines and requires prophylactic treatment in an effort to decrease their frequency. Candesartan is an option for this indication if other options are unsuitable, ineffective, or not tolerated.

Ibuprofen is incorrect. This is a treatment for acute migraine rather than prevention. Furthermore, her history of asthma makes this option unsuitable.

Medical cannabis is incorrect. There is emerging evidence that cannabidiol (CBD) oil may relieve the symptoms of migraine. However, it is not currently approved for migraine prophylaxis in the UK and so this is incorrect.

Propranolol is incorrect. This is a good option for the prevention of migraine but it is contraindicated in patients with asthma.

Sumatriptan is incorrect. This is a treatment for the acute relief of migraine. It is not indicated in the prophylaxis of migraine.

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A 20-year-old man presents to the neurology clinic following his second seizure. His partner describes episodes during which the patient is unresponsive and appears to be 'staring into space'. The patient has no recollection of these events but does recall that after his reported absences, he often feels nauseous and disorientated for several hours.

Blood tests to identify electrolyte abnormalities are all within normal limits. An MRI head is reportedly within normal limits. An EEG (electroencephalogram) shows interictal epileptiform discharges.

Given the likely diagnosis, which of the following drug therapies would be the most appropriate first-line agent?

A. Sodium valproate
B. Levetiracetam
C. Carbamazepine
D. Phenytoin
E. Lamotrigine

A. Sodium valproate

Absence seizures: sodium valproate and ethosuximide are first-line

This patient has a history and EEG consistent with a diagnosis of absence seizures. Sodium valproate or ethosuximide are considered first-line in the treatment of absence seizures.

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A 55-year-old woman attends neurology clinic for follow-up. At her first appointment 6 months previously, a clinical diagnosis of trigeminal neuralgia had been made and treatment with carbamazepine initiated. During clinic review the patient complained of ongoing symptoms with only limited benefit from drug treatment.

On her previous assessment, the patient had reported pain episodes associated with the cheek and jaw of the right side of her face. Subsequently, in addition to similar ongoing episodes, the patient had suffered from two episodes of pain affecting the forehead and peri-orbital region on the left side of her face. On one occasion, she had suffered from both right and left sided pain at the same time. She reported that the carbamazepine treatment had initially reduced the intensity of the pain episodes by perhaps 50 %, but that the severity of recent attacks had again worsened to their original level. When her drug history was checked, it was apparent that the carbamazepine dose had been titrated up appropriately by the patients GP.

The patient also stated that following her previous clinic appointment she had been discussing the situation with her sister, who had reminded the patient that their mother had suffered several episodes of visual loss during her life but had not undergone any medical investigation.

Cranial nerve examination demonstrated normal eye movements and pupillary responses. There was no evidence of facial nerve weakness, however a subjective numbness of sensation was noted in the left cheek region (this had not been noted on previous examination in clinic). There were no lesions around the head and neck or inside the mouth. Peripheral neurological examination was remarkable for borderline dysdiadochokinesia in the right upper limb and a positive Babinski response in the right lower limb.

What is the most appropriate investigation for this patient?

A. Visual evoked potentials
B. Three-plane fine-slice MRI brain with contrast
C. CSF oligoclonal bands
D. CT brain with contrast
E. Standard protocol MRI brain with contrast

E. Standard protocol MRI brain with contrast

The patients previous clinical diagnosis of trigeminal neuralgia clearly needs to be reviewed in light of several red-flag features. In particular, the limited response to carbamazepine, the occurrence of simultaneous bilateral pain and the history of pain associated with the ophthalmic division are inconsistent with a typical presentation of trigeminal neuralgia. Moreover, the patient has developed some soft neurological signs that are suggestive of CNS lesions in various areas of the brain.

Multiple sclerosis is the most likely alternative diagnosis, although other causes of trigeminal pain such as space occupying lesions, malignant invasion of the trigeminal nerve or thalamic infarction are also possibilities. The best investigation to assess for all these differentials is standard protocol MRI brain with contrast. Demyelination or trigeminal nerve infiltration would not be reliably noted on CT brain with contrast making this less likely to progress the diagnosis.

Visual evoked potentials and CSF oligoclonal bands are other investigations that may support a diagnosis of multiple sclerosis but would not confirm or refute this diagnosis in isolation.

Typical cases of trigeminal neuralgia are considered to result from contact between blood vessels and the trigeminal nerve. Such vascular contacts can be observed on specialised MRI protocols requiring fine-slicing in three-planes. However, given the additional expense and the fact that such contacts can be a normal variant, this investigation is not normally performed. A standard protocol MRI brain is sufficient to assess for the important differentials of trigeminal neuralgia discussed above.

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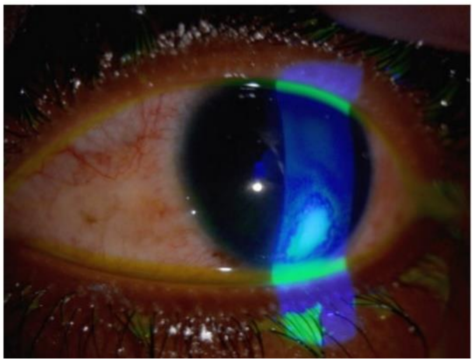
A 23-year-old man presents to the eye casualty department with 3 days of pain in the right eye, accompanied by photophobia and watering. He has also noticed frequent bouts of yellow discharge. The patient has no significant past medical history but is a contact lens wearer. He admits to having fallen asleep and leaving his contact lenses in, several nights ago.

The eye is urgently examined using a slit lamp and appears as shown below:



Given the likely diagnosis, what is the most appropriate management?

A. Intramuscular ceftriaxone
B. Oral moxifloxacin
C. Topical dexamethasone
D. Topical ganciclovir
E. Topical gentamicin

E. Topical gentamicin

This patient's presentation, coupled with his history of contact lens overuse, is suggestive of keratitis. This is confirmed by the examination - fluorescein staining has been used and, under slit lamp investigation, a corneal ulcer is clearly demonstrated on the right eye. Treatment depends on the underlying cause. With the history of discharge, a bacterial cause can be presumed and so treatment involves the use of antibiotics - topical gentamicin is an appropriate choice and is often combined with another antibiotic such as topical vancomycin.

Intramuscular ceftriaxone is generally not recommended except for suspected gonococcal eye infection. In the absence of any systemic symptoms being mentioned here, this is unlikely.

Oral moxifloxacin is a suitable choice when oral antibiotics are indicated for keratitis. This is not usually necessary except for in cases of corneal perforation, which is not seen here.

Topical dexamethasone is used in a variety of eye diseases to reduce inflammation. However, it is not usually used in bacterial keratitis or corneal ulcers, as the evidence demonstrates no significant benefit.

Topical ganciclovir is used in the management of dendritic or herpetic keratitis, which is viral in nature. This is differentiated upon careful slit lamp examination - fluorescein staining instead would show a distinct branching pattern.

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A 27 year-old woman attends the neurology clinic complaining of headache and visual disturbance. She has recently immigrated from Ghana. Her symptoms began approximately one month ago, shortly after the birth of her first child. She experiences dull frontal headache which is worst in the mornings and on coughing or straining, as well as transient episodes of 'darkening' of her vision. She saw a doctor in Ghana and was diagnosed with idiopathic intracranial hypertension. She is taking acetazolamide 250mg BD and no other medication.

On examination the visual fields are markedly constricted and the right blind spot is enlarged. Fundoscopy shows bilateral papilloedema worse on the right. The remainder of the neurological examination is unremarkable. BMI is 18 kg/m².

Plain computed tomography of the brain is normal.

Incidentally as she is leaving the clinic she mentions that she has also been experiencing pins and needles in the hands and feet.

What is the best course of action?

A. Increase dose of acetazolamide
B. Request nerve conduction studies
C. Organise for therapeutic lumbar puncture
D. Refer to neurosurgeons for consideration of ventriculo-peritoneal shunting
E. Request CT venography

E. Request CT venography

The history of headache suggestive of raised intracranial pressure associated with transient visual obscurations, as well as the examination findings, are all compatible with idiopathic intracranial hypertension (IIH). However, the onset of symptoms in the puerperium, in a slim patient with no other risk factors for IIH, raises the suspicion that this may actually be a cerebral venous sinus thrombosis. All patients with IIH should have imaging of the venous system with CT or MR to exclude a thrombus which can be more appropriately treated with anticoagulation.

Therapeutic lumbar puncture can ease the headache of IIH but is a short-term measure.

Ventriculo-peritoneal shunting can be used where medical management has failed.

There is no role for nerve conduction studies in this patient. Paraesthesia are a common side effect of acetazolamide, and this patient is unlikely to tolerate an increase in the dose.

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A 56-year-old consultant surgeon has recently been diagnosed with idiopathic Parkinson's disease, and attends clinic for review.

You read the previous clinic letter and see that he presented with tremor affecting mainly the right hand, which forced him to stop operating. On examination he also had some bradykinesia and rigidity of the right hand. He was started on co-beneldopa 100/25, initially one tablet three times daily, with instructions to up-titrate as tolerated.

When you see him today, he is concerned that there has been no improvement in the tremor. This is causing him considerable distress as he hopes to return to clinical work. His motor control is otherwise good, and throughout the day and night he has no difficulty with 'off' periods or freezing. He currently takes co-beneldopa 100/25, two tablets three times daily, and reports no complications.

On examination there is a resting tremor of the right hand. There is no discernable rigidity or bradykinesia.

What is the best treatment option?

A. Increase dose of co-beneldopa
B. Increase frequency of co-beneldopa
C. Add procyclidine
D. Add entacapone
E. Add selegiline

C. Add procyclidine

Tremor-predominant Parkinson's disease can be highly disabling and tremor may persist despite standard treatment with dopaminergic agents, which primarily improve bradykinesia. First-line treatment is such cases is the addition of an anticholinergic drug such as procyclidine, orphenadrine, or trihexyphenidyl.

The main side-effect which limits the use of anticholinergics in older patients is cognitive dysfunction, and dementia is a relative contra-indication. Other side effects include blurred vision, urinary retention, nausea, constipation, and dry mouth.

Second-line treatments for persistent tremor include amantidine, clozapine, and propranolol. Deep brain stimulation may be used in refractory cases.Increasing the dose of L-dopa is required when 'off' symptoms (bradykinesia, rigidity, freezing) do not respond to the starting dose, however this patient is taking a reasonable dose of L-dopa (600mg) and is not troubled by 'off' symptoms.

Increasing the frequency of L-dopa, which has a short half-life, is required when patients become 'off' in between doses. As the disease progresses the on period after each dose becomes shorter.

Entacapone and selegiline are used as adjuncts to prolong the 'on' period after a dose of L-dopa

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You see a 46 year-old man who has been referred by his GP to the neurology clinic.

He gives a one year history of facial pain. The pain particularly comes on when he is shaving or brushing his teeth, and he describes it as 'stabbing' through the teeth of his upper jaw and over the left side of his face. He has seen a succession of dentists and had several teeth removed, with no relief. The pain has been getting progressively worse, and whereas before it occurred in discrete attacks, it now occurs almost all the time. He has read several online sources and has become convinced that he has a brain tumour, which has led to him becoming depressed and withdrawn.

His past medical history includes essential hypertension, for which he takes perindopril. He also suffers from sinusitis, and has had a sinus washout on more than one occasion. Two years ago whilst on a business trip abroad he had a problem with the vision in his right eye, which spontaneously resolved over a few weeks, and for which he sought no treatment.

General examination is unremarkable. Cranial nerve examination is largely normal but you notice that there is a patch of numbness over the left cheek. Power is 5/5 across all muscle groups in the limbs, reflexes are normal, and plantars are downgoing. Sensation is the limbs is normal.

What is the most appropriate course of action?

A. Reassure him he has trigeminal neuralgia, for which unfortunately there is no treatment
B. Reassure him he has trigeminal neuralgia, and start carbamazepine 300mg daily
C. Refer to the neurosurgeons for microvascular decompression
D. Perform MR head with gadolinium contrast
E. Perform CT head with contrast

D. Perform MR head with gadolinium contrast

This man needs investigation before any further management is considered. He has pain which is typical for trigeminal neuralgia. However, objective sensory loss does not occur in idiopathic trigeminal neuralgia and is highly suggestive of an underlying cause. This may occur due to compression of the trigeminal nerve by a space-occupying lesion such a tumour, or by a demyelinating plaque of multiple sclerosis affecting the trigeminal root at the pons. The episode of transient unilateral visual disturbance that this man suffered two years ago raises the possibility of optic neuritis, and thus the history is suspicious for MS. Malignancy is also a distinct possibility. Gadolinium-enhanced MR of the head is the best investigation to detect both.

Carbamazepine is an effective treatment for trigeminal neuralgia and produces at least some response in 70% of patients. Neurosurgical options such as microvascular decompression are considered only once medical options have been exhausted.

Question 81 of 409

A 68-year-old man who has a history of Type 2 diabetes for the past 12 years comes to the clinic for review. His diabetes is currently managed with BD mixed insulin and over the past year he has suffered increasing pins and needles and pain in both feet. Apart from Type 2 diabetes, a previous myocardial infarction and intermittent urinary retention over the past year are noted. His GP has diagnosed him with benign prostatic hypertrophy. Examination reveals a blood pressure of 148/82 mmHg, his pulse is 80 beats per minute and regular. There is peripheral neuropathy affecting both feet, which are numb to just below the ankle. Routine bloods including renal function are unremarkable.

Which of the following is the most appropriate intervention?

A. Amitriptyline
B. Carbamazepine
C. Duloxetine
D. Pregabalin
E. Tramadol

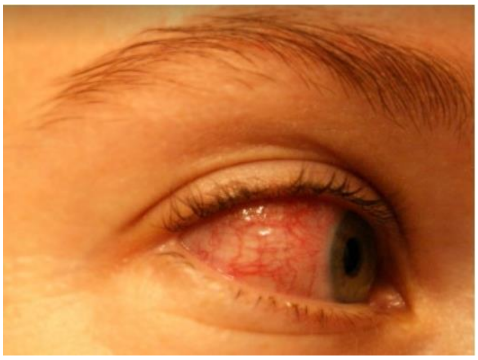
D. Pregabalin

The correct answer is pregabalin. Pregabalin is a GABA analogue and is recommended as an option by NICE for the treatment of neuropathic pain. it has the advantage versus gabapentin of less frequent daily dosing which drives improved compliance. It is also not associated with urinary retention which drives its selection versus other options listed.

Both amitriptyline and duloxetine are alternative first-line options for the treatment of diabetic neuropathic pain, although they are avoided here because both can be associated with urinary retention. Carbamazepine is not recommended for first-line treatment of diabetic neuropathy, and nor is tramadol because it is only partially effective and has addictive potential.

Question 82 of 409

A 42-year-old woman with rheumatoid arthritis presents to the clinic with complaints of pain and redness in the right eye. Her symptoms of rheumatoid arthritis are well-controlled and she is currently taking methotrexate and hydroxychloroquine. On examination, her right eye is watery and red. An image of her right eye is attached.



What is the most appropriate next step?

- A. Reassurance only
- B. Same-day assessment by an ophthalmologist
- C. Stop hydroxychloroquine
- D. Topical chloramphenicol
- E. Topical glucocorticoids

B. Same-day assessment by an ophthalmologist

The patient in this scenario has developed scleritis and therefore requires same-day assessment by an ophthalmologist . Painful red eye in a patient with rheumatoid arthritis and the image shown in this scenario is typical of scleritis. The image shows a thickening of the sclera (pinkishcoloured) with dilated episcleral vessels. Scleritis is a full-thickening inflammation of the sclera and is characterised by pain and redness of the eye with a gradual decrease in vision. Rheumatoid arthritis is the most common disease associated with scleritis. Patients with scleritis should be offered a same-day assessment by an ophthalmologist because vision-threatening complications can occur if scleritis is left untreated.

Episcleritis is the most important differential here that can be managed by reassurance only , in contrast to scleritis. Episcleritis is characterised by a gritty sensation in the eye and redness of the sclera with normal vision. Episcleritis is a painless condition and a history of painful red eye in this scenario rules out episcleritis.

There is no indication to stop Hydroxychloroquine as this drug has no role in causing scleritis. Hydroxychloroquine is known for its ophthalmic toxicities but it rarely causes scleritis. Ocular sideeffects of hydroxychloroquine include retinopathy, colour blindness, lens opacity, and keratopathy.

Topical chloramphenicol has no role in treating scleritis. Chloramphenicol ointment is the firstline agent to treat conjunctivitis which can be considered here given the acute onset of red eye. There is no history of itching or discharge in this scenario which makes conjunctivitis a less-likely diagnosis.

Oral glucocorticoids, and not topical glucocorticoids are used in treating scleritis. Oral glucocorticoids are reserved for only severe presentations of scleritis. Other immunosuppressive drugs are used to treat resistant cases of scleritis.

Question 83 of 409

A 52-year-old man is admitted to the stroke unit with a right total anterior circulation syndrome (TACS) infarct. He arrived at hospital 2.5 hours after the onset of his symptoms and was treated with intravenous alteplase at 3 hours post-onset.

He is known have an atrial septal defect which was discovered after a murmur was heard at a routine insurance medical several years ago. He works in the oil business and has recently returned from a business trip to Saudi Arabia.

On examination the following day there subtle signs of improvement with increased movement in his left hand. However, the rest of his arm remains flaccid and he has persisting dense hemiplegia affecting his right leg. He has a notable homonymous hemianopia on examination. A routine CT Brain 24-hours post-thrombolysis revealed established ischaemic changes in the MCA territory with new petechial haemorrhage along the border of the infarct.

Later that evening, his conscious level falls. His Glasgow Coma Scale changes from E4 M6 V2, to E2, M4 V2. His blood pressure is 187/112 mmHg.

Urgent bloods reveal: Haemoglobin is 120 g/l, Prothrombin time is 27 seconds, Activated partial thromboplastin time (APTT) 49 seconds

What intervention is likely to have the most benefit?

- A. Correction of coagulopathy
- B. Decompressive craniotomy
- C. Control of blood pressure
- D. Protamine sulphate
- E. Tranexamic acid

B. Decompressive craniotomy

This patient is at high risk of malignant middle cerebral artery (MCA) syndrome. Malignant MCA syndrome typically occurs in younger patients who have suffered an extensive ischaemic stroke (usually in the MCA territory). After infarction the brain swells. As younger brains are likely to have less atrophy, there is less room for expansion. Subsequently, intracranial hypertension develops.This typically presents 48 hours after onset of stroke with reduced conscious level.

Thrombolysis should reduce the risk of infarction and subsequent brain oedema, however, recanalisation is not achieved in every patient and thus thrombolysed patients can still develop malignant MCA syndrome. Furthermore, this patient was thrombolysed 3 hours after onset and so is likely to have established tissue infarction regardless. Treatment with urgent decompressive craniotomy can be life-saving. The procedure is particularly indicated in situations like this, where the patient has a non-dominant stroke and is, therefore, likely to recover language functions and have a more favourable outcome if they survive the acute event.

Acute haemorrhagic transformation is the main differential in this case. While the change in his conscious level should prompt repeat imaging (which would discriminate bleeding from oedema) the risk of large haemorrhagic transformation >24 hours post-thrombolysis is lower than the risk of malignant MCA syndrome. This is especially true when the previous scan has shown only grade I

Haemorrhagic infarction (HI-1) as classified using the ECAS II grading system. Urgent discussion with neurosurgery is recommended.

Question 84 of 409

An 86-year-old man is brought in by ambulance following suspicion of a stroke. His wife noticed sudden right-sided weakness. He describes sudden onset of weakness with no preceding symptoms. He has a past medical history of hypertension, diabetes and glaucoma. On examination, he has right sided pyramidal weakness of the arm and leg with associated sensory loss as well as reduced tone. His left side is normal. Cranial nerve examination shows homonymous hemianopia but is otherwise normal.

What is the most likely diagnosis?

A. Total anterior circulation stroke
B. Partial anterior circulation stroke
C. Lacunar stroke
D. Posterior circulation stroke
E. Brainstem stroke

B. Partial anterior circulation stroke

The correct answer is partial anterior circulation stroke. He has had sudden onset of unilateral weakness and sensory loss with homonymous hemianopia but no other evidence of higher cognitive dysfunction. The most likely diagnosis, as per the Oxford classification, is partial anterior circulation stroke. If he would have had evidence of higher further higher cognitive dysfunction, such as dysphasia, then a total anterior circulation stroke would be a better description. A lacunar stroke would cause isolated sensory or motor symptoms, not both. A posterior circulation stroke is more likely to affect cerebellar function whilst a brainstem stroke would affect cranial nerves profoundly.

Question 85 of 409

A 24-year-old female is reporting difficulty in walking in an inpatient rehabilitation unit during recovery from spinal surgery. Six weeks previously, she underwent emergency spinal haematoma evacuation surgery under the neurosurgeons from T2 to T4 after sustaining a fall playing netball. She was previously fit and well, with no past medical history, giving birth to a healthy baby boy 18 months ago.

On examination, the patient has normal tone in her upper and lower limbs. Examination of her power while lying on her bed demonstrates 5/5 on the MRC power scale with normal sensation to cotton wool, pin prick and proprioception. Reflexes were present (2+) in her biceps, triceps, supinator, patella and ankles, both plantars were downgoing. On walking, the patient's gait is markedly abnormal, with both feet sliding along the floor for 80 metres of walking without lifting of her feet between steps. Cognition is intact.

What is the optimal management?

A. Repeat MRI head
B. Repeat MRI whole spine
C. Nerve conduction studies and electromyography
D. Lumbar puncture
E. Education and reassurance

E. Education and reassurance

This patient's gait is inconsistent with any pattern of organic pathology: it is inconsistent to be unable to lift your feet but have normal ankle dorsiflexion and plantarflexion. The patient has a combination of recent significant organic pathology and inorganic pathology, which is frequently the case in functional neurological disorders. Repeat MRI imaging, electrophysiology or lumbar puncture are unlikely to help and only medicalise the disorder. It is crucial to reinforce this message by avoiding unnecessary investigations or treatments. Management of functional neurology is based on education and reassurance, telling the patient that it is a 'conceptualisation' problem and not an issue with the brain or nerves. Psychiatric input may also have a role in this situation if the patient continues to be refractory to first line management: cognitive behavioural therapy may be helpful.

Question 86 of 409

A 36-year-old male presents to your outpatient clinic with a progressive history over the past 5 years of increasing, progressive 'clumsiness'. His work colleagues had a long running joke with him that he is poorly coordinated for about the past five years but in recent weeks, he has noticed that he is unable to write legibly or even hold a key still using either hand to open a door. He denies any recent weight loss of night sweats, is otherwise healthy with no other past medical history. He is a lifelong non-smoker with a minimal alcohol history and lives with his wife and 2 children.

On examination, his cranial nerves were unremarkable except for mild multidirectional nystagmus at primary gaze. Fundoscopy was normal. Limb examination revealed a significant impairment of finger-nose and heel-shin testing. His gait, tone, power, sensation and reflexes were normal with downgoing plantars. A brief mini-mental state examination scored 30/30. An MRI head is awaited. His blood tests are as below:

Hb	158 g/l
Platelets	323 * 10 /l
WBC	6.5 * 10 /l
Na	141 mmol/l
K	4.9 mmol/l
Urea	6.6 mmol/l
Creatinine	85 µmol/l
CRP	2 mg/l
Creatine kinase	223 IU/l (50-335)
TSH	3.3 mu/l
Free T4	17 nmol/l
HIV	negative
Anti-neuronal antibodies	negative

Which investigation is most likely to yield the diagnosis?

A. Neurogenetics testing
B. CT chest, abdomen, pelvis
C. Vitamin B12 and folate levels
D. Lumbar puncture for cerebrospinal fluid including 14-3-3 and S100
E. Anti-GQ1b antibodies

A. Neurogenetics testing

There appears to be a chronic onset syndrome of pure ataxia, without systemic features or cognitive impairment, nor history of alcohol excess. The likely diagnosis is one of spinocerebellar ataxias (SCA), in particular one of the classified 'type III' syndrome such as SCA6, an adult onset cerebellar ataxia diagnosed by neurogenetic testing.

It is important to rule out key differential diagnoses however: patients with acute onset cerebellar syndrome should be considered for Miller-Fisher syndrome, a post-viral variant of Guillain Barre syndrome resulting in a triad of ophthalmoplegia, ataxia and areflexia, commonly after gastrointestinal infections, diagnosed with positive anti GQ1b antibodies. The time course for this illness over 5 years makes Miller-Fisher unlikely.

A subacute course of weeks and small number of months should alert you to possible prion disease such as Creutzfeldt-Jakob disease (CJD) and a paraneoplastic syndrome. The former typically presents with rapid cognitive decline and myoclonus, classically with MRI features (pulvinar sign, cortical ribboning), CSF features (14-3-3, S100, real-time QUIC) and EEG features (periodic spiking). The latter can present years before a solid tumour is identified on structural imaging with positive anti-neuronal antibodies, resulting in progressive symptoms as the cerebellum degenerate over months. Due to the low sensitivity of anti-neuronal antibodies, a negative result does not necessarily rule out paraneoplastic syndrome but such a long time course of 5 years would be unusual. The likelihood of identifying a significant mass on cross sectional CT imaging is low.

Lastly, the lack of a alcohol or gastrointestinal history makes cerebellar degeneration due to vitamin deficiency very unlikely. As a result, neurogenetics testing for SCA is most likely to yield the underlying diagnosis.

Question 87 of 409

A 32-year-old female presents with a 6-month history of progressive lower limb weakness and suprapubic tenderness. On examination, cranial nerves and upper limbs were unremarkable. You note 1/5 power in both lower limbs, hyperreflexic patella and ankle reflexes, bilateral clonus and upgoing plantar reflexes. The abdominal examination also revealed a suprapubic mass. After a urethral catheter was inserted, you note a residual volume of 1.8l. She is known to be HIV positive, diagnosed 8 years ago. Blood tests from an outpatient appointment revealed:

CMV IgG	positive
EBV IgG	positive
HTLV antibody	positive
Hepatitis B	negative
Hepatitis C	negative

An MRI spine is awaited.

What is the most likely diagnosis?

A. Tropical spastic paraparesis
B. Spinal toxoplasmosis
C. CMV transverse myelitis
D. EBV transverse myelitis
E. Tabes dorsalis

A. Tropical spastic paraparesis

This is a HIV and HTLV-1 positive patient presenting with paraparesis and urinary retention, a classic presentation of tropical spastic paraparesis. Toxoplasmosis in HIV patients typically presents in the brain, lungs or as a chorioretinitis. Transient viral inflammatory transverse myelitis by CMV and EBV typically resolves within 3 months, the presence of IgG in both suggest previous exposure and is of limited significance. Tabes dorsalis is a possibility but peripheral reflexes are typically lost with an extensor plantar reflex. Diagnosis of HTLV-1 tropical spastic paraparesis relies on a combination of clinical features and HTLV-1 load in serum and CSF. Treatments are limited, with steroids, presumably as an anti-inflammatory, producing only mild effects in a small population of patients.

Question 88 of 409

A patient with refractory focal epilepsy on two agents is commenced on levetiracetam in addition.

Which adverse effect should patients that have been commenced on this drug most importantly be warned about?

A. Oral hairy leukoplakia
B. Weight gain
C. Irritability and aggression
D. QT-interval prolongation
E. Nystagmus

C. Irritability and aggression

It is important to recognise and know something about levetiracetam as it is increasingly becoming a favourite for neurologists to use these days, not least because of its favourable adverse effects profile (wherein it hardly has any compared to many others), but also its ease of use given that you do not need to monitor levels, and its oral and IV bioavailability are the same (useful in nil by mouth patients). It is also recently more affordable having now lost its patent. The one thing to watch out (and ensure you warn patients about) with levetiracetam is behavioural abnormalities and psychotic symptoms. These range from irritability to frank aggression. If there is a background of psychotic symptoms you may wish to avoid levetiracetam. It is something not to miss in the drug history of an epileptic patient who presents to the acute take with a change in behaviour.

Question 89 of 409

A 48-year-old lady develops a sudden-onset left-sided headache in the face and neck areas 36 hours ago whilst at rest. It is of 10/10 severity and reached this maximum intensity within seconds. It has not subsided since and is throbbing in nature. She also developed a transient period of loss of vision in the left eye lasting 2 hours before resolving. She also says that 'food tastes funny' since these problems developed. On examination, she has small, sluggishly light-responsive left pupil compared to the right and partial left ptosis. The face is otherwise unremarkable to examine as is the remaining neurological examination. Routine blood investigations are unremarkable. Plain computerised tomography (CT) of the head is unremarkable. Lumbar puncture is negative for xanthochromia. CT

angiogram of the head and neck vessels demonstrates a pseudo-lumen of the carotid artery.

Which of the following treatments would you initiate?

A. High flow 100% oxygen
B. Prednisolone
C. Indomethacin
D. Aspirin
E. Acetazolamide

D. Aspirin

She has an internal carotid artery dissection on the left side culminating in a partial left-sided Horner's syndrome. The term 'partial' Horner's is used because anhidrosis is absent. The sympathetic fibres innervating the facial sweat glands are anatomically located on the external rather than the internal carotid artery; thus, anhidrosis is not a finding in the setting of internal carotid dissection.

In terms of management, there is little role for surgery in spontaneous carotid artery dissections. All dissections put people at risk of thromboembolic complications because of turbulent flow at the sight, therefore we normally initiate antiplatelet or anticoagulation therapy. There is little evidence for which is better over the other in this area although pragmatically speaking antiplatelets are safer. Candidates for angioplasty and stent placement include patients with persistent ischaemic symptoms despite adequate anticoagulation, patients with a contraindication to anticoagulant/antiplatelet therapy, and patients with significantly compromised cerebral blood flow.

In this particular case, the loss of vision points towards possible amaurosis fugax, backing up the need for anti-thromboembolic agents.

Question 90 of 409

A 28-year-old gentleman student from Germany presents to you with right foot drop ongoing for two weeks with some numbness and tingling of the foot. These symptoms developed after he knelt down to pick something up from the floor. Three years ago he woke up from sleep with clawing of his fourth and fifth digit after having been asleep in a prone position and this lasted a week. Eight years ago he also had a left wrist and finger drop lasting three weeks after he sat on the couch with his left arm draped over the back of the couch for ten minutes. He denies falling asleep or remaining on the couch for a prolonged period. He has no other past medical history of note and has never sought medical advice for his problems. On examination, there is right foot drop (2/5 power) and similar weakness of dorsiflexion and eversion of the right foot. There is also sensory loss over the lower lateral part of the right leg and dorsum of the right foot in all modalities. Reflexes are intact. Neurological examination and general examination are otherwise unremarkable.

Which of the following tests would confirm the suspected diagnosis?

A. Nerve conduction studies
B. Electromyography
C. Nerve biopsy
D. PMP22 gene testing
E. HBA1C

D. PMP22 gene testing

The diagnosis is Hereditary Neuropathy with Liability to Pressure Palsy (HNPP). This is a neurological syndrome in which trivial trauma to a peripheral nerve e.g. sleeping on a limb, results in a mononeuropathy which may take weeks to resolve. It usually presents in second or third decade of life. The condition is most common in families with Dutch or German ancestry. It is caused by a deletion in the peripheral myelin protein 22 gene on chromosome 17. It is an autosomal dominant condition.

The patient's presenting mononeuropathy is a common peroneal nerve palsy. Three years ago he had an ulnar nerve palsy and eight years ago he had a radial nerve palsy (also called Saturday night palsy).

Nerve conduction studies in HNPP are characteristic of a demyelinating neuropathy: conduction is slow and action potentials are small. Nerve biopsy may show a predominance of smaller fibres and localised thickening of the myelin sheath. These investigations are helpful in supporting the diagnosis although gene testing, if positive, is confirmatory.

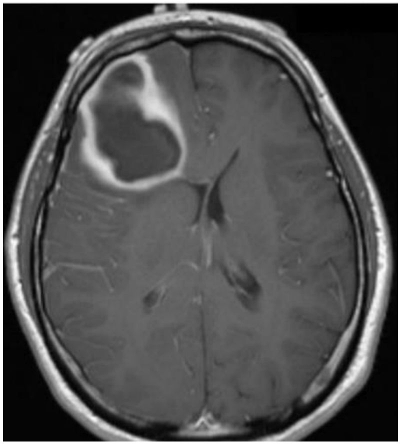
Management is conservative with e.g. wrist splints, ankle-foot orthoses, and protective padding.

Question 91 of 409

A 51-year-old man presents to the emergency department following a witnessed tonic-clonic seizure of 2 minutes duration. There is no history of epilepsy and he has never had a seizure before. Over the last week, he has been complaining of intermittent headaches and fevers. His past medical history includes hypertension for which he takes amlodipine, ramipril, and indapamide once daily.

On examination, he is drowsy with a GCS of 13 (E3V4M6). His chest sounds are clear and his heart sounds are normal. He has a soft and non-tender abdomen. Temperature is recorded as 38.1ºC.

A contrast-enhanced MRI scan is performed, as shown below:



Given the likely diagnosis, which of the following is the most probable causative pathogen?

A. Cytomegalovirus

B. Cryptococcus neoformans
C. Mycobacterium tuberculosis
D. Staphylococcus aureus
E. Toxoplasma gondii

D. Staphylococcus aureus

This patient's MRI brain shows a radiolucent space-occupying lesion with outermost ring enhancement in the frontal lobe. Causes of intracerebral lesions with ring enhancement include toxoplasmosis and intracerebral abscesses. Bacterial intracranial abscesses commonly arise from infections in adjacent structures including otitis media, sinusitis or mastoiditis. Symptoms of intracerebral abscesses depend on the structures involved but commonly include headache, fever, signs of increased intracranial pressure (e.g. papilloedema and seizures) and focal neurological signs. The clinical history of fever, headache and seizures combined with the imaging point toward the diagnosis of an intracerebral brain abscess. The most common pathogens responsible for intracerebral abscesses include Staphylococcus aureus and Streptococcus pneumoniae.

Cytomegalovirus (CMV) is a viral infection that commonly causes illness in patients with HIV, particularly in those with a CD4 count < 50 cells/mL. The most common form of CMV is CMV retinitis. However, CMV encephalitis is also common. On imaging, cerebral oedema may be seen. Meningoencephalitis and ventriculitis are also features of infection with CMV. These are not seen here, making CMV encephalitis unlikely.

Cryptococcus neoformans causes an opportunistic fungal infection (most frequently seen in patients with a history of human immunodeficiency virus) that causes headache, fevers, confusion and signs of raised intracranial pressure. Imaging studies show leptomeningeal enhancement, cryptococcomas, hydrocephalus or dilated perivascular spaces. These features are not seen in the imaging above, and the absence of a history of immunosuppression makes cryptococcal meningitis unlikely.

Mycobacterium tuberculosis can cause tuberculous meningitis with similar symptoms to those of bacterial meningitis (e.g. fever, headache, meningism). Tuberculous meningitis is more common in patients with HIV and can be seen on imaging as leptomeningitis from the presence of thick exudative material in the subarachnoid space.

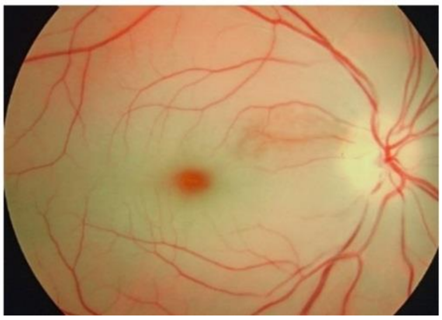
Toxoplasmosis is an opportunistic infection caused by the parasite Toxoplasma gondii. It is the most common cause of neurological brain abscesses in patients with HIV with symptoms including headache, fever and neurological changes including seizures, hemiplegia, cerebellar signs, or an acute confusional state. Toxoplasmosis also presents with ring enhancement on contrast imaging. However, toxoplasmosis often presents with multiple ring-enhancing lesions. Furthermore, the absence of a history of immunosuppression makes an intracerebral abscess more likely.

Question 92 of 409

A 65-year-old man presents to the emergency department with sudden onset blindness in his left eye. He reports only being able to see out of his right eye since waking this morning. There is no history of eye pain or photophobia. His past medical history includes hypertension and hypothyroidism. He takes amlodipine, levothyroxine and regular co-codamol for recurrent headaches over the past 4-6 weeks.

On examination, he is alert and oriented with a GCS of 15/15. There is normal power and sensation in his upper and lower limbs bilaterally with normal tone and coordination. The visual acuity in his right eye is 6/9 and his left eye is only able to make out gross hand movements. The remainder of his cranial nerves are intact.

Fundoscopy of his eye is completed, as shown below:



Laboratory investigations are requested: ESR 55 mm/hr < (age / 2)

What is the recommended management for this patient?

A. Hyperbaric oxygen therapy
B. Intravenous methylprednisolone
C. Intravitreal anti-vascular endothelial growth factor (anti-VEGF)
D. Oral aspirin and referral for intra-arterial thrombolytic therapy
E. Topical corticosteroids

B. Intravenous methylprednisolone

This patient has a fundoscopic image representing central retinal artery occlusion with the classic appearance of a 'cherry red spot' on a pale retina. Central retinal artery occlusion presents as an acute onset painless loss of vision that can either be secondary to thromboembolic or arteriopathy (e.g. temporal arteritis). The history of recurrent headaches and raised ESR makes temporal arteritis the more likely underlying diagnosis.

It is important to note that temporal arteritis can cause visual loss via two mechanisms. Most commonly, patients lose their vision secondary to arteritic anterior ischaemic optic neuropathy, which causes occlusion of blood supply to the optic nerve. However, in 10% of patients with visual loss secondary to temporal arteritis, the visual disturbance is caused by central retinal artery occlusion, as in this case.

Intravenous methylprednisolone is correct. Visual loss secondary to temporal arteritis is an ophthalmological and rheumatological emergency and treatment should be initiated promptly including intravenous methylprednisolone to avoid permanent vision loss. This can later be deescalated to oral prednisolone.

Hyperbaric oxygen therapy is incorrect. This has been used as a treatment method to maintain adequate oxygenation of the retina whilst waiting for reperfusion. However, this treatment is not routinely used and would not reverse the underlying inflammatory process in temporal arteritis.

Intravitreal anti-vascular endothelial growth factor (anti-VEGF) is incorrect. This is used in the management of proliferative retinopathy (e.g. proliferative diabetic retinopathy). Proliferative retinopathy presents with multiple tortuous vessels and pre-retinal haemorrhages which are not seen here.

Oral aspirin and referral for intra-arterial thrombolytic therapy is incorrect. This would be an appropriate treatment for a thromboembolic cause of central retinal artery occlusion. However, this patient has a history of chronic headaches in association with a raised ESR which is suggestive of temporal arteritis. The pathology of artery occlusion in temporal arteritis is arteriopathy and inflammatory change rather than emboli and, therefore, methylprednisolone would be more appropriate.

Topical corticosteroids is incorrect. This is used to treat inflammatory ocular diseases such as anterior uveitis. This usually presents with eye pain and reduced visual acuity. Topical corticosteroids are not potent enough to treat arteriopathy. Rather, intravenous methylprednisolone is needed in the initial stages.

Question 93 of 409

A 34 year old man attended the Emergency Department after experiencing a severe sudden onset headache at home. The pain was felt across the whole of the head and reached a 10/10 severity within a few seconds of onset. Since the headache began the patient had experienced a loud pulsatile pounding noise in his right ear. There was no reported loss of consciousness or other neurological symptoms. In the preceding days the patient had been fit and well. On close questioning he recalled that that morning he had been struck hard in the face by a soccer ball while watching his son's team.

The patient had no significant past medical history and took no medications. There was no family history of neurological disease.

On examination, the patient's right pupil was constricted compared to his left, with both pupils reactive to light. There was a partial ptosis of the right eye. Cranial nerve examination was otherwise unremarkable except for possible right hypoglossal palsy. Examination of the remainder of the peripheral nervous system was remarkable.

Details of initial investigations are given below.

CT brain (non-contrast): no acute intracranial pathology identified; normal ventricular system; no boney injury.

Lumbar puncture:

CSF red cells	2 / mm³
CSF white cells	4 / mm³
CSF gram stain	unremarkable
CSF glucose	60 % serum level
CSF protein	0.65 g / L
CSF	negative for haemoglobin break down products

What is the correct diagnosis?

A. Vertebral artery dissection
B. Internal carotid artery dissection
C. Posterior reversible encephalopathy syndrome
D. Reversible cerebrovascular vasoconstriction syndrome
E. Posterior communicating artery aneurysm

B. Internal carotid artery dissection

Internal carotid artery dissection can present with thunderclap headache, commonly following minor trauma, as in this case. Possible associated symptoms and signs include homolateral Horner's syndrome, tinnitus or tongue palsy. CT brain and lumbar puncture are usually normal (or near normal in the case of LP) with diagnosis confirmed by angiography.

The remaining options are all possible causes of thunderclap headache. Vertebral artery dissection would typically cause a posterior circulation stroke pattern of signs and symptoms. Reversible cerebrovascular vasoconstriction syndrome is associated with hypertension and seizures and diagnosed by arterial beeding on angiography. Posterior reversible encephalopathy syndrome is also associated with hypertension and specific changes on MRI brain secondary to vasogenic brain oedema. Posterior communicating artery aneurysm causes compression of the third nerve with unilateral mydriasis without other signs of third nerve palsy.

Question 94 of 409

An 18-year-old female attends the clinic complaining of speech difficulties and slowing of movements. On examination you note bilateral rigidity, akinesia with loss of arm swing, and bradykinesia.

What drug may have caused this disorder?

A. Sodium valproate
B. Lithium
C. Metoclopramide
D. Fluoxetine
E. Domperidone

C. Metoclopramide

Metoclopramide can cause extrapyramidal effects, especially in children and young adults (15-19 years old)

The clinical features are suggestive of Parkinsonism. Drug induced Parkinsonism often presents with bilateral feature (in contrast to idiopathic Parkinson's disease which often presents asymmetrically). It can be caused by several drugs including anti-psychotics, anti depressants, antiemetics and calcium channel blockers.

Metoclopramide has a high risk of extrapyramidal effects, especially in children and young adults (15-19 years old), making this the best answer.

Several weak studies have shown an association of sodium valproate, lithium, domperidone and SSRIs to drug induced Parkinsonism, however the evidence is much weaker and they are less common causes compared to metoclopramide.

In many cases, the first approach to treatment will be to try stopping the offending drug for a sufficient length of time, reducing it, or changing it to another drug that may be less likely to cause drug-induced parkinsonism. If it is not possible to stop taking the offending drug, then anticholinergic drugs may be used, for example procyclidine.

Question 95 of 409

A 74-year-old man is admitted to the acute medical unit with loss of consciousness. His wife states that she found him slumped in the arm chair in an unresponsive state approximately one hour ago. He had been feeling well before hand.

On examination you note bilateral 1mm pupils. His Glasgow coma score (GCS) is 4 (M2, E1, V1).

His past medical history includes hypertension, hypercholesterolaemia and osteoarthritis. His drug history includes codeine, paracetamol, ramipril and atorvastatin. You administer 400 micrograms of naloxone. There is no change in GCS or pupil size.

What is the most likely diagnosis?

A. Pontine haemorrhage
B. Medulla haemorrhage
C. Midbrain haemorrhage
D. Subarachnoid haemorrhage
E. Opioid overdose

A. Pontine haemorrhage

Pontine haemorrhage commonly presents with reduced GCS, paralysis and bilateral pin point pupils

The patient has severely reduced GCS and is in a coma. The differential of coma is wide and includes vascular (e.g. intracerebral haemorrhage), inflammatory (e.g. cerebral vasculitis), neoplastic (e.g. paraneoplastic encephalitis, or cerebral space occupying lesion), drugs (e.g. sedatives), infective (e.g. meningitis, encephalitis), and metabolic (e.g. hyponatraemia, hypoglycaemia, hypercapnia) causes.

Pontine haemorrhage commonly presents with reduced GCS, paralysis and bilateral pin point pupils. Lesions of the pons can compress the first order sympathetic tracts resulting in bilateral Horner's syndrome.The differential clearly includes opioid overdose, however there is nothing to suggest that an overdose has occurred, and the fact that his GCS and pupil size do not respond to naloxone further suggests that this is not the correct diagnosis.

Midbrain lesions typically cause fixed, midpoint pupils.

Lesions of the lateral medulla can result in an ipsilateral Horner's syndrome and a pin point pupil. A bilateral medulla lesion is a possible differential, however the sudden onset of coma and pin point pupils is more characteristic of pontine haemorrhage.

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A 28-year-old female is admitted after a paracetamol overdose. She took twenty 500mg tablets. She states that she wants to die and that life is not worth living any more. She had left a note for her parents. On mental state examination she has poverty of thought, a low affect and evidence of delusions of nihilism. She has a past medical history of severe depression. She is deemed to be medically fit for discharge however due to ongoing suicidal ideation she is transferred to a psychiatric unit for further assessment. During the admission she develops catatonia and stops eating and drinking.

Your team decides that electroconvulsive therapy is indicated.

Which one of the following options is an absolute contraindication for electroconvulsive therapy?

A. Retinal detachment
B. Cardiac conduction disease
C. Malignant hypertension
D. Raised intracranial pressure
E. Myocardial infarction within the last 3 months

D. Raised intracranial pressure

The only absolute contraindication for electroconvulsive therapy is raised intracranial pressure

With the exception of raised intracranial pressure, there are no absolute contraindications to ECT, although there are a number of clinical situations in which extra caution is required.

All the other options listed are relative contraindications.

ECT leads to increases in cerebral blood flow and increased intracranial pressure (ICP). This is the reason that raised ICP is an absolute contraindication.

Initially, the seizure activity causes a large parasympathetic discharge with bradycardia, atrial or ventricular premature beats, and occasional asystole. Therefore, cardiac conductive disease is a relative contraindication.

The initial parasympathetic stimulus is followed by sympathetic discharge causing tachycardia, hypertension, ST-segment depression and T-wave inversion not associated with myocardialenzyme changes. It can rarely cause ventricular tachycardia. Therefore, uncontrolled hypertension, and recent MI are relative contraindications.

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A 28-year-old female presents with a frontal headache with associated fever and photophobia. She states that she has been suffering from an ear infection over the past week.

Lumbar puncture results are as follows:

Cell count 228 (95% neutrophils) Protein 1.2 g/l (normal 0.2-0.4) Glucose 2.2 mmol/l

A plasma glucose is as follows: Plasma glucose 6.8 mmol/l

What is the most likely organism?

A. ECHO virus
B. Streptococcus pneumoniae
C. Neisseria meningitidis
D. Listeria monocytogenes
E. Mycobacterium tuberculosis

B. Streptococcus pneumoniae

Meningitis which occurs as a complication of an ear infection is nearly always caused by Streptococcus pneumoniae

The patient has clinical features of meningitis. The CSF results confirm a neutrophilic pleocytosis with a high protein and low glucose. This is indicative of bacterial meningitis. Streptococcus pneumoniae and Neisseria meningitidis are high up the differential. Meningitis occurring as a complication of an ear infection is almost always caused by Streptococcus pneumoniae, making this the best answer.

ECHO virus is a cause of viral meningitis which presents with a lymphocytic pleocytosis and normalglucose, and normal / mildly raised protein. Listeria monocytogenes meningitis tends to only affect elderly and immunosuppressed individuals.

Mycobacterium tuberculosis meningitis causes a lymphocytic pleocytosis and tends to present subacutely with a several week prodrome phase.

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A 24-year-old woman presents with right eye vision loss and painful eye movements. She had a previous episode affecting her left eye last year. Following an MRI head and spine with contrast and a lumbar puncture examination, she is diagnosed with multiple sclerosis. She is started on high-dose steroids which induces remission.

You start the patient on maintenance ocrelizumab.

What is the mechanism of action of this drug?

A. Anti-TNF
B. Dihydrofolate reductase inhibitor
C. Humanized anti-CD20 monoclonal antibody
D. Recombinant monoclonal antibody that antagonises alpha-4 beta-1-integrin
E. Sphingosine 1-phosphate (S1P) receptor modulator

C Humanized anti-CD20 monoclonal antibody

Ocrelizumab is considered a high-efficacy drug that is often used first-line to reduce the risk of relapse in MS

Humanized anti-CD20 monoclonal antibody is correct. Ocrelizumab selectively targets the CD20-positive B-cells by binding to the surface proteins of the cells and protecting the nerve cells from mediated damage caused by CD20-positive B lymphoma cell lines.

Anti-TNF is incorrect. Drugs in this class include adalimumab, infliximab, and etanercept. Dihydrofolate reductase inhibitor is incorrect. This is the mechanism of action of methotrexate.

Recombinant monoclonal antibody that antagonises alpha-4 beta-1-integrin is incorrect. This is the mechanism of action of natalizumab.

Sphingosine 1-phosphate (S1P) receptor modulator is incorrect. This is the mechanism of action of fingolimod.

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A 56-year-old man comes to the office due to blurred vision and drooping of his left eyelid for over two weeks. He was diagnosed with hypertension and hyperlipidaemia 2 years ago, for which he takes lisinopril and atorvastatin. The patient has smoked a pack of cigarettes daily for the past 30 years.

Vital signs are within normal limits. Physical examination shows normal pupils and eye movements. After closing the eyes for 5 minutes, the drooping resolves. However, after a few minutes, the drooping recurs, worsening progressively. His cardiovascular, respiratory and neurological examinations are normal.

Which of the following is the best next step in the management of this patient?

A. Botulinum antitoxin
B. Intravenous immunoglobulin
C. Prednisone
D. Pyridostigmine
E. Smoking cessation

D. Pyridostigmine

Pyridostigmine is the 1st line drug in the management of ocular myasthenia gravis

This patient with ptosis and eyelid fatigue has ocular myasthenia gravis. Ocular myasthenia gravis presents with ptosis and oculomotor paresis. The ptosis is often unilateral and may alternate from one side to another. Eyelid fatigability is another classic sign of ocular myasthenia gravis. In myasthenia gravis, autoantibodies against acetylcholine receptors prevent acetylcholine from properly binding to these receptors. Furthermore, acetylcholine is quickly broken down into choline and acetic acid by the cholinesterase enzyme at the neuromuscular junction. Pyridostigmine is a cholinesterase inhibitor. It slows down the breakdown of acetylcholine and is the first-line drug in the management of symptomatic myasthenia, like ocular myasthenia gravis.

Patients with botulism present with acute symmetric descending flaccid paralysis which progresses over time. This patient's presentation is not progressing, and is therefore, unlikely to be botulism.IV immunoglobulins and plasmapheresis are used in the management of Guillain-Barre syndrome. They don't have a role in treating ocular myasthenia gravis.

The use of prednisone and other corticosteroids is controversial in ocular myasthenia gravis. Corticosteroids are associated with severe adverse effects and difficulty weaning off. Moreover, several weeks to months of corticosteroid therapy are required to maintain beneficial effects in patients with ocular myasthenia gravis. Anticholinesterase agents, like pyridostigmine, should be used before resorting to corticosteroids.

While this patient should consider smoking cessation, quitting smoking is unlikely to improve symptoms of ocular myasthenia gravis.

Question 100 of 409

A 23-year-old woman presents to the emergency department having been found by her father in a reduced state of consciousness one hour ago. She has a past medical history of epilepsy for which she is taking levetiracetam and anxiety and depression for which she has been prescribed short courses of diazepam in the past. Several empty packets of diazepam were found by her bedside.

On initial assessment, her oxygen saturations are 91% on 15L via a non-rebreather. Her heart rate is 61/min and her blood pressure is 91/64mmHg. She is responsive to pain only.

What is the most appropriate next step in management?

A. Activated charcoal
B. Anaesthetic support
C. Haemofiltration
D. Intravenous flumazenil
E. Naloxone

B. Anaesthetic support

Benzodiazepine overdose is best managed supportively and with airway protection and ventilation if needed. Flumazenil should be avoided unless for reversal of anaesthesia Important for me Less important

The primary concern here is related to this woman's airway. Despite 15L of oxygen via a nonrebreather, her oxygen saturations are only holding at 91%. Airway obstruction is likely a result of her reduced conscious level. As per the ALS algorithm, airway manoeuvres and anaesthetic support for potential intubation and ventilation should be sought.

Activated charcoal may be helpful in acute ingested poisonings to limit subsequent serum levels however this does not address the most urgent concern here which is airway obstruction and is therefore not the most appropriate first step.

Haemofiltration can be used to clear various drugs however again this will take some time to have an effect and does not address the most pressing concern, airway obstruction. Therefore this is not the correct answer.

Intravenous flumazenil is the primary reversal agent for benzodiazepine overdose however has to be used with extreme caution as its use can cause intractable seizures and arrhythmias. As thiswoman is young and particularly as she has a history of epilepsy, airway support and ventilation if required would be a more favoured approach in this case.

Naloxone is an agent used to reverse opioid overdose however as there is no indication of opioid use here, this is unlikely to reverse this woman's condition.

Question 101 of 409

A 27-year-old woman comes for review. She is having problems with increasingly frequent migraine attacks. These occur throughout her menstrual cycle and have no relation to her periods. She has tried a combination of paracetamol and ibuprofen to try and control the attacks but this seems to have had a limited effect. Her current medication includes paracetamol and ibuprofen as required and Cerazette (a progestogen-only pill).

What is the most appropriate medication to try and reduce the frequency of her migraine attacks?

A. Propranolol
B. Zolmitriptan
C. Topiramate
D. Amitriptyline
E. Switch Cerazette to a combined oral contraceptive pill

A. Propranolol

Propranolol is preferable to topiramate in women of childbearing age (i.e. the majority of women with migraine)

Zolmitriptan is useful to abort attacks but is not generally used for prophylaxis, except in the case of menstrual-related migraine (see below for more details). From the history it is clear that the headaches are not confined to the days around menstruation.

NICE recommend either propranolol or topiramate for migraine prophylaxis. Propranolol should be used in preference to topiramate in women of child bearing age as it may be teratogenic and it can reduce the effectiveness of hormonal contraceptives

The combined oral contraceptive pill is contraindicated given her history of migraine.

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A 34-year-old woman attends with weakness in her left leg. The symptoms started over a few hours and have persisted for 2 days.

On examination of the left leg, she has a brisk ankle jerk and an upgoing plantar, dorsiflexion is reduced in power, and tone is normal. Examination of the right leg is unremarkable.

Further questioning reveals an episode of numbness and tingling in her right hand 3 months ago.

Given the likely diagnosis, what treatment is most likely to reduce relapses for this patient?

A. Dexamethasone
B. Fingolimod
C. Glatiramer
D. Interferon beta
E. Natalizumab

E. Natalizumab

Monoclonal antibodies such as natalizumab have the strongest evidence base for reducing relapse in multiple sclerosis

The most likely diagnosis here is multiple sclerosis given the upper motor neuron signs separated by time. MR imaging of the brain and spinal cord would be appropriate to confirm the diagnosis. Be aware that, during the acute phase, tone may be normal.

Natalizumab is the correct answer. Natalizumab is a high-efficacy disease-modifying therapy and has the strongest evidence base for reduced relapse rates.

Dexamethasone can be used in acute relapsing disease, but it does not confer any long-term benefit in reducing further relapses.

Fingolimod is a SIP1 modulator. It can be used as a disease-modifying treatment in multiple sclerosis but has less supporting evidence for reducing relapses compared to natalizumab.

Interferon beta is another disease-modifying drug used in the treatment of multiple sclerosis. It is what is termed a 'low-risk' therapy as it has been around for quite a long while and has a goodsafety profile. Some clinicians favour starting with a 'low-risk' therapy and escalating to 'high efficacy' treatments if relapses aren't controlled. The evidence, however, does state that natalizumab has better evidence for controlling relapse rates overall.

Glatiramer is also termed a 'low-risk' therapy due to its good safety profile. Again, the evidence for a reduction in relapse rate is not as strong as that for natalizumab.

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A 28-year-old man presents to his general practitioner with peripheral vision loss. He explains that his vision has gradually 'narrowed' over the last 2 months. He also describes difficulty seeing in the dark, which has preceded the current symptoms by several years, although he has not sought medical attention for this.

On examination, fundoscopy reveals black bone spicule-shaped pigmentation in the peripheral retina. Blood tests are also performed:

Na	138 mmol/L	(135 - 145)
K	3.3 mmol/L	(3.5 - 5.0)
Bicarbonate	29 mmol/L	(22 - 29)
Urea	9.1 mmol/L	(2.0 - 7.0)
Creatinine	320 µmol/L	(55 - 120)

What is the most likely cause of this patient's abnormal blood tests?

A. Alport syndrome
B. Autosomal dominant polycystic kidney disease (ADPKD)
C. Autosomal recessive polycystic kidney disease (ARPKD)
D. Bartter syndrome
E. Renal artery stenosis

A. Alport syndrome

Retinitis pigmentosa classically presents with black bone-spicule pigmentation on fundoscopy

Tunnel vision, night blindness, and fundoscopy findings in this patient are characteristic of retinitis pigmentosa. The presence of renal disease with retinitis pigmentosa hints at Alport syndrome , which is due to an abnormality in the gene that codes for type 4 collagen. Sensorineural hearing loss, although not featured in this scenario, is another prominent feature of Alport syndrome.

The common disease associations of autosomal dominant polycystic kidney disease (ADPKD) are cerebral aneurysms, hepatic cysts, colonic diverticula, mitral regurgitation and mitral prolapse.Retinitis pigmentosa is not associated with ADPKD.

Patients with autosomal recessive polycystic kidney disease (ARPKD) rarely survive beyond 5 years of age. ARPKD is not associated with retinitis pigmentosa.

Although mild hypokalemia and alkalosis are present in this scenario, this is less likely to be due to Bartter syndrome , which is extremely rare. Furthermore, there is no association between Bartter syndrome and retinitis pigmentosa.

Hypokalemic metabolic alkalosis may be seen with renal artery stenosis (RAS) due to the stimulation of the renin-angiotensin-aldosterone system. The normal blood pressure and presence of retinitis pigmentosa make Alport syndrome more likely.

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A 42-year-old woman is referred into the TIA clinic due to recurrent left arm weakness. She has been having recurrent episodes of left sided weakness with associated unilateral throbbing headache, nausea and photophobia. During each episode she find that her left arm becomes weak. She has a past medical history of headaches, DVT during pregnancy and hypothyroidism. Currently there are no neurological features.

What is the most likely diagnosis?

A. TIA
B. Subdural haematoma
C. Sinus venous thrombosis
D. Brain tumour
E. Migraines

E. Migraines

Recurrence of the same neurological signs or symptoms is unlikely to be due to TIA

This patient has recurrent weakness as a feature of neurology with headache and photophobia. The most likely diagnosis is migraines which can cause weakness. The recurrence of symptoms makes TIA less likely. This is because it is unlikely for clots to occur in the same vessel and cause the same symptoms. Subdural haematoma is unlikely to cause photophobia, and also unlikely to cause intermittent weakness. Brain tumours are associated with progressive and unrelenting headaches as well as mass effect symptoms.

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A 76-year-old residential home resident was seen in the neurology clinic. She had a history of Alzheimer's disease, and her behaviour had become progressively more challenging over the past 4 months, for which she had been started on regular haloperidol.

The residential home staff had reported that over the previous four weeks she had developed involuntary movements of her mouth, with frequent tongue protrusion and lip-smacking. There were no neurological deficits and no history of blackouts. Two weeks ago she was reviewed by the general practitioner who stopped her haloperidol. However, her involuntary facial movements became more prominent following this.

On examination, she appeared well, other than continuous involuntary lip-smacking, tongue protrusion, and frequent eye-blinking. She was alert, and there were no focal neurological deficits.

What is the most likely cause for her symptoms?

A. Temporal lobe epilepsy
B. Progression of Alzheimer's disease
C. Chronic tardive dyskinesia
D. Frontotemporal dementia
E. Huntington's disease

C. Chronic tardive dyskinesia

Antipsychotics may cause tardive dyskinesia

First-generation antipsychotic agents (e.g. haloperidol, chlorpromazine) may result in extrapyramidal side effects due to relatively non-selective dopamine blockade. This may manifest as chronic tardive dyskinesia, as in this case. Clinical features of chronic tardive dyskinesia include involuntary repetitive movements, such as lip-smacking, tongue protrusion, and distal chorea. It is treated by withdrawal of the offending agent, though it may paradoxically worsen in the first few weeks of stopping the drug. The typical features and temporal relationship make chronic tardive dyskinesia secondary to haloperidol the most likely diagnosis in this case. Other extrapyramidal side effects include acute dystonic reactions, akathisia, and Parkinsonism.Lip-smacking may occur in temporal lobe epilepsy, but this would be associated with impaired consciousness and would only last a few minutes. Advanced Alzheimer's disease may be associated with involuntary movements (e.g. myoclonus), but is not typically associated with dyskinesias. Parkinsonism may occur in frontotemporal dementia, but dyskinesia is not associated. A new diagnosis of Huntington's disease would be very unlikely in this age group.

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A 42 year old computer programmer is brought to the Emergency Department by his wife with chest pains. He is haemodynamically stable with a blood pressure of 105/69mmHg and a heart rate of 92bpm but he is peripherally shut down with cool, dusky extremities and weak peripheral pulses. He complains of crushing central and left sided chest pain with some evidence of sweating and pallor. He also complains of severe global headache. His chest is clear and his heart sounds are normal. Chest x-ray is unremarkable.

An ECG shows pronounced inferior and lateral ST segment depression. A baseline troponin at 4 hours since onset of chest pain is raised. His wife tells you he has been taking clarithromycin from the GP for the past 6 days for a chest infection and she has been concerned he has been acting strangely since starting this with confusion, excitability, delusions and visual hallucinations. He has also had stomach cramps and diarrhoea for the past 2 days. His medical history is significant for migraines for which he takes PRN zolmitriptan and prophylactic ergotamine.

Which one of the following explains this patients symptoms most completely?

A. Acute non-ST segment myocardial infarction
B. Acute meningoencephalitis
C. Clarithromycin induced haemolytic uraemic syndrome (HUS)
D. Ergotism
E. Serotonin syndrome

D. Ergotism

Concomitant use of ergotamine and macrolides may cause ergot poisoning (ergotism) with confusion, headache, seizures, psychosis and global vasoconstriction leading to critical limb ischaemia, cardiac ischaemia and bowel hypoperfusion

In this case, this patient is quite clearly having an acute coronary event but non ST elevation myocardial infarction does not explain all his symptoms. Meningoencephalitis is a possibility but without further evidence of infection is difficult to prove and does not explain the cardiac ischaemia or bowel and limb symptoms. Serotonin syndrome may well be seen with drugs which modulate serotonergic neurotransmission such as zolmitriptan and cause confusion and agitation but the hallmark of this syndrome is hyperpyrexia which is absent here; cool peripheries would notbe expected. Clarithromycin induced HUS could explain the majority of symptoms described here, however it is exceedingly rare and given that this patient has taken both clarithromycin and ergotamine concomitantly, ergotism is a far more likely diagnosis.

Management of ergotism is largely supportive and discontinuation of offending drugs is important. Seizures and psychosis may be controlled with benzodiazepines. Hypotension and haemodynamic instability should be managed with filling; avoid vasopressors such as adrenaline, noradrenaline or vasopressin; dobutamine or milrinone may be used cautiously if needed. Vasodilators such as GTN, iloprost or nitroprusside may be needed for critical ischaemia. Coronary ischaemia should be assessed as in any patient. Rebound migraine may be expected on cessation of ergot derivatives.

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A 22 year old woman is brought to the Emergency Department by ambulance. She is accompanied by her boyfriend who tells you he thinks she has taken a deliberate overdose of up to 80 fluoxetine tablets (20mg) within the past eight hours. On assessment her airway is patent but threatened with a respiratory rate of 26 and peripheral oxygen saturations of 97% on air. Her chest is clear. The heart rate is 118bpm and the blood pressure is 98/39mmHg. Capillary blood glucose is 5.0mmol/L. The ECG shows a sinus tachycardia with QRS duration 114msec and corrected QT interval 575msec. She is globally hypertonic, shivering, nauseated, vomiting and sweaty with a tympanic temperature of 37.7°C and dilated pupils and prominent clonus. She suddenly has a prolonged tonic-clonic seizure and receives 20mg intravenous diazepam with no response after 15 minutes.

Which of the following is the safest subsequent intervention in this patients management?

A. Further 10mg intravenous diazepam
B. 10mg rectal diazepam
C. Phenytoin infusion 20mg/kg
D. Levetiracetam 1000mg intravenous infusion
E. Intubate and ventilate

E. Intubate and ventilate

Care should be taken in status epilepticus due to drug toxicities refractory to benzodiazepines; phenytoin is cardiotoxic and phenobarbital is a potent respiratory depressant. Intubation is often safest

Seizures in drug overdoses are relatively common occurrences and may be related to the drug itself, its metabolites or as a consequence of electrolyte derangements. Generally, brief and isolated convulsions do not require specific treatment and may be managed conservatively. Recurrent or prolonged seizures predispose to injury, hypoxia and renal failure; first line treatment is with benzodiazepines such as diazepam, lorazepam or midazolam. In pre-hospital environments, or in those patients in whom intravascular access is yet to be established, rectal diazepam is a reasonable route of administration since it is rapidly absorbed across the bowel wall and enters the pudendal vasculature and is rapidly distributed to the central nervous system without passing through the liver first. If intravenous access is present however, as in the above patient, this routeshould be used. If a choice of intravenous benzodiazepines is available, lorazepam should be used preferentially since diazepam emulsion has a high risk of thrombophlebitis. In the above vignette, 20mg intravenous diazepam has been administered with no clinical effect; this is the maximal dose permitted in 4 hours and further doses are unlikely to be helpful. Second line agents which are recommended in persistent seizure activity are phenytoin or phenobarbital. Care must be taken when using phenytoin as an anti-seizure drug in overdoses since it exerts its anti-epileptic effects by inhibiting sodium channels; this is an effect shared by many cardiotoxic drugs taken in overdose. Co-administration of phenytoin and many drugs may lead to significant widening of the QRS duration, prolongation of the QT interval and ultimately to torsades de pointes, ventricular tachycardias and cardiac arrest. Phenytoin should not be used when there is any ECG evidence of prolongation of QRS or QT phases.

Levetiracetam is a relatively new antiepileptic drug which works by modulating NMDA glutamate neurotransmission. It can be given intravenously when oral routes are unavailable but it is licensed only for use in established epilepsy and not as a treatment for status epilepticus or drug induced seizures and should not be used here.

Phenobarbital is a drug which is rarely used in status epilepticus due to the risk of significant respiratory depression. It is however considered a second line agent to treat seizures as an alternative to phenytoin and could potentially be used here, although given the rest of the clinical information and probable evolving serotonin syndrome, intubation, ventilation and paralysis is probably safest.

In poisonings with cardiotoxic drugs, use of antiarrhythmic drugs to control ECG anomalies is not recommended; intravenous sodium bicarbonate may be given in patients with prolongation of QRS duration and magnesium sulphate can be given in QT prolongation or torsades de pointes, although the QT duration may not reduce but the incidence of escape rhythms is lessened.

Question 108 of 409

A 58-year-old man presents to the emergency department with eight hours of a painful red eye. He has a past medical history of inflammatory bowel disease, type 2 diabetes and hypertension. His regular medications include metformin, amlodipine and mesalazine. He does not smoke or drink alcohol. He wears contact lenses.

On examination, his right eye is red. Visual acuity is decreased in the right eye (6/18) in comparison to the left eye (6/6). The pupil in the right eye is fixed and mid-dilated.

Fluorescein staining of the right eye is normal.

Which of the following options is used in the management of the likely diagnosis?

A. IV acetazolamide
B. Oral steroids
C. Saline irrigation
D. Topical antibiotics
E. Topical steroids

A. IV acetazolamide

Alongside eye drops, IV acetazolamide is used in the initial emergency medical management of acute angle-closure glaucoma

IV acetazolamide is the correct answer. The patient presents with a painful red eye with reduced visual acuity. The characteristic finding of acute glaucoma is a fixed mid-dilated pupil. Diabetes and hypertension are risk factors for the development of this condition. Alongside eye drops, IV acetazolamide is used in the emergency management of this condition.

Saline irrigation is incorrect. This has a place in the management of a chemical injury to the eyes but there is no history of such an event.

Topical and oral steroids are both incorrect. Both these options have a place in the management of anterior uveitis. However, the pupil is typically miotic in this condition. Additionally, the onset is more insidious and frequently bilateral when affecting those patients with inflammatory bowel disease.Topical antibiotics is not correct. This may be indicated in the management of bacterial conjunctivitis or a corneal abrasion. The lack of discharge and the existence of visual obscuration make conjunctivitis an unlikely cause. Normal fluorescein staining makes a corneal abrasion unlikely despite the history of contact lens use.

Question 109 of 409

A 72-year-old man is brought to the emergency department by ambulance after complaining of sudden onset weakness. He has a past medical history of hypertension and atrial fibrillation. His medications include amlodipine, bisoprolol and apixaban.

On examination, power is 4/5 in the right upper limb and right lower limb. Cranial nerve examination reveals his left eye to have deviated inferiorly and laterally. There is left-sided ptosis. The pupillary responses are normal.

A CT head is unremarkable.

Based on the likely diagnosis, which cerebral artery is most likely affected?

A. Left anterior cerebral artery
B. Basilar artery

C. Left middle cerebral artery
D. Left posterior cerebral artery
E. Left retinal artery

D. Left posterior cerebral artery

Weber's syndrome is a form of midbrain stroke characterised by the an ipsilateral CN III palsy and contralateral hemiparesis

Posterior cerebral artery is correct. The patient presents with an ipsilateral third nerve palsy and contralateral hemiplegia. The sudden onset and history of hypertension and AF suggest a vascular event. This is suggestive of Weber's syndrome, which is caused by infarction of a branch of the posterior cerebral artery in the midbrain.

Basilar artery is incorrect. Infarction as a consequence of basilar artery thrombosis would cause 'locked-in syndrome. This is when the body and most of the facial muscles are paralyzed but consciousness and eye movements are preserved.

Left middle cerebral artery is incorrect. A stroke affecting this region may result in contralateral (i.e. right-sided) weakness, sensory disturbance and speech disturbance.

Left anterior cerebral artery is incorrect. This is characterized by weakness and sensory lossaffecting the lower limbs more than the upper limbs.

Left retinal artery is incorrect. This would cause monocular visual loss.

Question 110 of 409

A 24-year-old woman presents to her general practitioner complaining of frequent headaches, occurring 3-4 times a month. They last for approximately 4 hours at a time. They are throbbing in nature and associated with photophobia, phonophobia, nausea and a visual aura. There is no specific pattern of timing to when the headaches occur. She has no past medical history and does not take any regular medications.

On examination, she has a raised body mass index (32 kg/m²). The neurological examination is unremarkable and she is headache-free at the time of review.

Given the likely diagnosis, what is the most appropriate medication to prevent further headaches?

A. Acetazolamide
B. Propranolol
C. Sumatriptan
D. Topiramate
E. Zolmitriptan

B. Propranolol

Propranolol is preferable to topiramate in women of childbearing age (i.e. the majority of women with migraine)

Propranolol is the correct answer. This woman presents with typical features of migraine (episodic throbbing headache associated with aura, photophobia, phonophobia and nausea). The first choice of prophylaxis of migraine in a woman of childbearing age is propranolol.

Acetazolamide is incorrect. While she does have a raised BMI, which is a risk factor for idiopathic intracranial hypertension, the headache characteristics are far more suggestive of migraines. Additionally, we are told the neurological examination is normal suggesting an absence of papilloedema.

Sumatriptan is incorrect. This is used to treat acute migraine rather than prevent migraine. Topiramate is incorrect. This can be used for prophylaxis of migraine but is potentially teratogenicand therefore less preferable in women of child-bearing age.

Zolmitriptan is incorrect. This can be used as prophylaxis for menstrual migraines. However, we are told her headaches have no predictable pattern and therefore this is not indicated.

Question 1 of 299

A 64-year-old lady presents with worsening anxiety to the emergency department. She has no history of mental health issues but her husband is worried that over the past month she has become very nervous and developed panic attacks. In addition, she is not able to carry out her daily tasks. She is retired and usually keeps active but over the past few weeks has become more disengaged and no longer is arranging to see friends or leave the house. She has terrors in the night where she screams and takes several hours of calming from her husband.

She is normally well and takes ramipril and a simvastatin for high blood pressure.

On examination, she is withdrawn but alert. She is haemodynamically stable and afebrile. Her abbreviated mental test score is 3/10 and she does not know where she is or the time of day. She has mildly increased tone in both arms with occasional myoclonic jerks and the power is 4/5 in all muscle groups. There is intact coordination and sensation.

Hb	140 g/l
Platelets	398 * 10 /l
WBC	7.6 * 10 /l
CRP	5mg/l
Na	134 mmol/l
K	4.2 mmol/l
Urea	4.3 mmol/l
Creatinine	67 µmol/l

CT head	brain atrophy, no intracranial haemorrhage or mass effect
MRI head	Hyperintense signals in the left thalamus with some similar changes in both putamen and right thalamus

What treatment should be initiated?

A. Supportive
B. Cyclophosphamide
C. Methylprednisolone
D. Intravenous antibiotics
E. Venlafaxine

لم يجلوب السؤال في الملف

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A 32-year-old man had a compressive pituitary macroadenoma surgically removed in France 4 weeks ago and has been on pituitary hormone replacement since, including growth hormone. Surgery was uncomplicated and he initially made a good recovery. He has no other past medical history. He has now developed, over the last 1-2 weeks, poor balance, with a broad-based gait. He has also become extremely forgetful. On examination, he has an ataxic gain and you also observe occasional myoclonic limb movements. His MMSE score is 22/30. Clinical examination is otherwise unremarkable, as are routine blood investigations.

Given the likely diagnosis, what might you expect to find on his MRI brain?

- A. Areas of demyelination in the cerebellum
- B. An area of high T2 signal in the right middle cerebral artery territory with restricted diffusion on diffusion-weighted sequences
- C. High T2 signal in the posterior thalamus
- D. Leptomeningeal enhancement
- E. Loss of grey-white matter differentiation

The diagnosis is Creutzfeldt-Jakob disease (CJD) from an iatrogenic source.

The use of prion-contaminated medication, grafts and instruments may result in iatrogenic CJD. The current worldwide total of growth-hormone-associated cases of CJD is 226. Most cases occurred in France (119 cases/1,880 recipients; attack rate 6.3%), the United Kingdom (65 cases/1,800 recipients; attack rate 3.6%), and the United States (29 cases/7,700 recipients; attack rate 0.4%). No new case has been identified since 2008 but would be important to recognise this condition and how it presents if it did, as it is invariably fatal -so patients need to be made aware of their prognosis.

The clinical features of the disease vary according to the route of inoculation.

Central inoculation, eg from infected neurosurgical instruments, results in a rapidly progressive neurodegenerative disease similar to sporadic CJD, characterised by ataxia, dementia, myoclonus, rigidity, and akinetic mutism. Survival is 2-12 months.

Peripheral inoculation, eg from the use of the old human growth hormone products, results in a 24% 15% 37% 13% 11%slightly less acute disease -characterised by progressive cerebellar ataxia and dementia. Survival is 8-18 months.

Question 2 of 299

A 42-year-old woman presents with a headache over the past week. She describes it as left sided, excruciating and it makes it difficult for her to sleep at night. The pain is worse when lying down and she notices that her nose is blocked on that side. She has vomited but has been feeling achy all over for the last few days. She has tried paracetamol and ibuprofen without benefit. She is otherwise well and only takes the combined oral contraceptive pill.

On examination, she appears lethargic. She has normal eye movements and fundoscopy is normal. She is able to move all her limbs with a power of 5/5 and has normal reflexes. She walks with a slow but steady gait. She is coryzal and she will not let you examine her face when you press over the frontal area.

She was seen in the emergency department a few days prior with this headache and the report is below.

CT head:- There is no evidence of bleeding, mass effect or hydrocephalus. It is noted that there is a homogeneous collection of moderately enhancing material in the frontal sinus.

What is the most likely diagnosis?

- A. Idiopathic intracranial hypertension
- B. Sinusitis
- C. Subarachnoid haemorrhage
- D. Hemicrania continua
- E. Meningioma

B. Sinusitis

Severe constant headache on a background of rhinitis is likely sinusitis

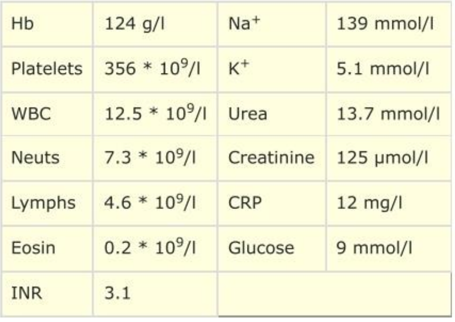
The history is an acute headache. Although there are features of raised intracranial pressure, a CT head excludes any meningioma. A subarachnoid haemorrhage typically is the most severeheadache that reaches maximal intensity over a few seconds to minutes. Hemicrania continua is a constant headache that often has autonomic features associated with it that are not seen here. Idiopathic intracranial hypertension is possible given her combined OCP use but there are no other features and fundoscopy shows no papilloedema. Examination is normal but the patient is anxious to be examined due to the exquisite tenderness of the facial sinuses in sinusitis. Furthermore, the CT head reports a collection of mucus in the sinuses seen incidentally.

Question 3 of 299

An 87-year-old lady normally resident at a nursing home is admitted with a short history of diarrhoea and vomiting, generalised malaise, stiffness, tremor, pyrexia and hallucinations. She has been previously admitted repeatedly with aspiration pneumonias, however no further details of her prior medical history are known, except her current medication including co-beneldopa, warfarin, amlodipine, amitriptyline, metformin and lactulose.

Examination is limited due to the agitated status of the patient, however chest auscultation and abdominal examination are unremarkable. A chest x-ray is unremarkable with a urine dipstick positive for ketones and containing a trace of blood.

Blood sample and blood cultures are taken and the results are provided below:



Hb	124 g/l	Na⁺	139 mmol/l
Platelets	356 * 10^9/l	K⁺	5.1 mmol/l
WBC	12.5 * 10^9/l	Urea	13.7 mmol/l
Neuts	7.3 * 10^9/l	Creatinine	125 µmol/l
Lymphs	4.6 * 10^9/l	CRP	12 mg/l
Eosin	0.2 * 10^9/l	Glucose	9 mmol/l
INR	3.1		

Taking in consideration her co-morbidities what would be your next step:

A. Initiate insulin
B. Start Tazocin
C. Prescribe rotigotine patch
D. Hold amlodipine
E. Prescribe 5 mg vitamin K orally

C. Prescribe rotigotine patch

Abrupt cessation of antiparkinsonian medication (dopamine agonists) can lead to neuroleptic malignant syndrome

This is a common situation in hospital medicine. A lady that suffers from Parkinson's disease (even though not explicitly stated - but can be assumed given the frequent aspiration pneumonias and her medication) presents dehydrated with diarrhoea and vomiting. It is possible that this lady may have gastroenteritis and it hence important to rehydrate her. It is also however important to ensure that she continues to take her anti-parkinsonian medication. Given her inability to eat and drink at the moment the most appropriate alternative would be a parenteral or topical solution. Given her blood picture and inability to isolate a source of infection, one should be cautious about starting broad spectrum antibiotics

Question 4 of 299

You are asked to review a patient on the surgical ward due to nursing concerns. The patient is a 21-year-old female who is normally fit and well and takes no medications. She was admitted three days ago for suspected appendicitis. So far she has been managed conservatively and was seemingly improving until this morning when her nurse went to perform her observations and discovered she was not responding and appeared 'stiff'.

Current management from the surgical team this admission: intravenous metronidazole, amoxicillin, and gentamicin, and regular paracetamol, oramorph, metoclopramide, and cyclizine for symptom control.

On examination, the patient's neck is in fixed extension with eyes fixed and deviated to the right. You noticed her jaw is clamped shut and limbs are rigid. Observations are stable.

What immediate medical management should be given in this situation?

A. Dantrolene
B. Naloxone
C. Diazepam
D. Adrenaline
E. Procyclidine

E. Procyclidine

Acute dystonic reactions are an adverse effect of antipsychotic medications

This patient is having an acute dystonic reaction secondary to metoclopramide. Reactions typically present with an extrapyramidal disorder and are managed with the withdrawal of the offending medication and administration of an anticholinergic such as procyclidine. Acute dystonic reactions should be considered in patients taking antipsychotic medication or other centrally acting dopamine agonists.

Question 5 of 299

A 72-year-old man presents to the emergency department. He had experienced slurring of his speech and right-sided facial drooping earlier that day following his lunch. He reports that the symptoms were mostly resolved ten minutes following their onset but took another five minutes to fully resolve.

On examination, there is no residual dysphasia or a facial droop and he has a normal neurological examination.

What imaging modality would be preferred for this patient?

A. CT angiogram head
B. Diffusion-weighted MRI head
C. Fluid attenuation inversion recovery (FLAIR) MRI head
D. T1-weighted MRI head
E. T2-weighted MRI head

B. Diffusion-weighted MRI head

MRI brain with diffusion-weighted imaging is the preferred modality in patients with suspected TIA who require brain imaging

The patient has focal neurological symptoms that lasted for only 10 minutes, consistent with a transient ischemic attack. The preferred imaging modality for TIA is a diffusion-weighted MRI head which is very sensitive to small ischaemic lesions.

CT angiogram is most commonly used to image an acute ischaemic stroke (where symptoms are ongoing). It is useful as a quick method to detect an occlusion which can then be treated. It is also used to detect intracranial aneurysms when subarachnoid hemorrhage is suspected.

FLAIR MRI head is a T2 weighted technique that dampens CSF signal and can be a superior method of detecting a white matter brain lesion and can be used to assess for leptomeningealdiseases.

T1-weighted MRI head is a sequence of MRI that provides the best contrast for paramagnetic contrast agents such as gadolinium-containing compounds. The sequence also is good for demonstrating anatomy and shows white matter as bright whilst fluid-filled compartments appear dark. It would not be the most effective imaging sequence to investigate a TIA.

T2-weighted MRI head is a sequence of MRI that lights up fluid-filled compartments, whilst white matter appears dark. Hence lesions that are associated with increased water content such as oedema will show up bright making this a good sequence to detect these lesions. It again would not be the most effective imaging sequence to investigate a TIA.

Question 6 of 299

A 57-year-old woman is seen on the acute medical take with a worsening frontal headache, nausea and distal right upper limb pins and needles.

She was born in India and emigrated 3 years ago to the United Kingdom (UK). Her past medical history includes tuberculosis treated 10 years ago and rheumatoid arthritis for which she has recently been started on methotrexate and sulfasalazine alongside a short course of oral prednisolone. Her chest X-ray was normal and quantiFERON test negative on entry to the UK.

On examination, she looks well and is alert and orientated. Her temperature is 36.8ºC, oxygen saturation 97% on air, heart rate 80 beats per minute, respiratory rate 18 breaths per minute and blood pressure 143/75 mmHg. There is symmetrical swelling in the small joints in both hands and neurological examination reveals 4/5 MRC grade of power in the distal right upper limb. Examination of the cardiovascular, respiratory and abdominal system reveals no additional abnormalities.

Her investigation results are as follows:

Hb	124 g/L
Platelets	398* 10 /L (150 - 400)
WBC	12 * 10 /L (4.0 - 11.0)
Na	135 mmol/L (135 - 145)
K	3.6 mmol/L (3.5 - 5.0)
Urea	5 mmol/L (2.0 - 7.0)
Creatinine	63 µmol/L (55 - 120)
CRP	24 (< 5)

You arrange a contrast CT head which shows the following- A 3-4cm diameter homogeneous contrast enhancing round lesion adjacent to the meningeal membrane situated in the left frontal lobe with evidence of surrounding oedema and mass effect. No additional lesions noted. No evidence of acute ischemia, haemorrhage or collection.

What is the most likely diagnosis?

A. Tuberculosis
B. Aspergilloma
C. Meningioma
D. Neuroblastoma
E. Lung metastasis

C. Meningioma

Meningioma primary brain tumors show contrast enhancement on a CT scan

The key to this question is the interpretation of the CT head findings. A meningioma will show homogeneous contrast enhancement on a CT scan.

The description and location of this space occupying lesion fit better with a meningioma than neuroblastoma.

Similarly, you may expect local or systemic signs and symptoms with metastatic lung cancer and CT findings fits better with the diagnosis of a meningioma. However, a screening CT scan of the chest, abdomen and pelvis may be appropriate to rule out this possibility.

We are told that this lady had active tuberculosis while living in India. However, on entry to the UK, there was no evidence of an active infection or a latent infection which could have been reactivated when initiated on immunosuppressive medications.

Although aspergilloma would be a risk with immunosuppression, this would be rarer and the CT head findings are more concordant with the diagnosis of a meningioma.

Question 7 of 299

A 56-year-old man is brought into the emergency department of a major stroke unit by his wife who reports that for the last 2-hours her husband has been unable to move his right side and is struggling to speak. He is known to have type 2 diabetes mellitus and hypercholesterolemia, for which he takes metformin and atorvastatin respectively. In addition, he has had a recent provoked lower limb deep vein thrombosis (DVT) for which he was started on apixaban. He works as an accountant, is an ex-smoker and drinks occasionally.

On examination, there is a dense right hemiparesis with expressive dysphasia. He scores 22 on the National Institute of Health Stroke Score (NIHSS).

Following rapid assessment, a CT brain and CT intracranial angiogram are performed, showing a proximal occlusion within the M2 segment of the middle cerebral artery with no major new ischaemic changes. An MRI head with MR angiography confirms these appearances.

Given this patient's presentation, which of the following would be the best intervention?

A. Thrombolysis immediately
B. Thrombectomy within 6 hours
C. Thrombolysis within 6 hours
D. Thrombectomy within 12 hours
E. Thrombolysis within 12 hours

B. Thrombectomy within 6 hours

The standard target time for thrombectomy in acute ischaemic stroke is 6 hours

This patient meets the criteria for a mechanical thrombectomy:

- thrombectomy can be achieved within 6 hours of symptom onset (if this cannot be achieved, consideration for thrombectomy is given in patients with sufficient salvageable brain tissue identified on imaging for up to 24 hours after symptom onset, although this is assessed on a case-by-case basis).

- he is anticoagulated, thus thrombolysis is contraindicated
- Imaging demonstrates a proximal occlusion
- There are no new major ischaemic changes on imaging
- his NIHSS is more than 5 he was independent prior to symptom onset

Question 8 of 299

You are asked to see a 57 year-old female who has presented acutely to eye clinic with deteriorating vision. On questioning she has experienced sudden blurring of the temporal field in her right eye. Her past medical history is noteworthy for hypertension and type 2 diabetes mellitus.

What is the likely diagnosis?

A. Branch retinal artery occlusion
B. Nasal retinal detachment
C. Central retinal vein occlusion
D. Temporal branch retinal vein occlusion
E. Nasal branch retinal vein occlusion

E. Nasal branch retinal vein occlusion

This case describes visual blurring and not total visual loss. Retinal artery occlusions lead to amaurosis fugax, often describes as a 'black curtain' descending over the vision. The most likely explanation for visual blurring is a retinal vein occlusion. The temporal field corresponds to the nasal retina. Both hypertension and diabetes are risk factors for a retinal vein occlusion.

Question 9 of 299

A 55-year-old man presents to the emergency department with a four-hour history of an acutely painful left eye. He also describes photophobia and reduced visual acuity associated with the pain.

On examination, diffuse redness is noted throughout the left eye. The left pupil is smaller than the left and he is unable to count fingers in the left eye although visual acuity in the right eye is unimpaired. It is also noted that there is some pus in the anterior chamber.

What treatment is most likely to be indicated?

A. Intravenous antibiotics
B. Intravenous diuretic
C. Steroid + mydriatic eye drops
D. Topical antibiotics
E. Topical beta blocker

C. Steroid + mydriatic eye drops

Anterior uveitis is most likely to be treated with a steroid + cycloplegic (mydriatic) drops

This man has presented with a case of anterior uveitis, as confirmed by the typical findings on examination. As per NICE guidance, the management of anterior uveitis is dependant on whether an infective or non-infective cause is established. As a result, all cases must be diagnosed and managed by ophthalmology in secondary care. The most common therapy is steroid + mydriatic drops and therefore this is the correct answer.

Intravenous antibiotics may be indicated in cases of infective anterior uveitis as directed by ophthalmology however treatment is more likely to be topical steroids and cycloplegics.

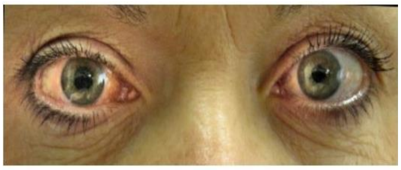
Carbonic anhydrase diuretics (for example acetazolamide) are of use in acute angle-closure glaucoma however as this is not the diagnosis here, this answer is incorrect.Topical antibiotics again may be indicated in infective cases but are not the most likely treatment.

Topical beta-blockers are again indicated in cases of acute angle-closure glaucoma which is not the diagnosis here.

Question 10 of 299

A 48-year-old woman presents to the eye casualty department with several hours of blurred vision in her right eye, accompanied by seeing 'haloes' around bright lights. She also describes nausea and numerous episodes of vomiting.

On examination, the eye appears as shown below:



What is an appropriate initial choice of treatment?

A. Topical brinzolamide
B. Topical dexamethasone
C. Topical ganciclovir
D. Topical gentamicin
E. Topical latanoprost

A. Topical brinzolamide

This patient's presentation of blurred vision, the description of 'haloes' around lights and nausea is suggestive of acute angle-closure glaucoma (AACG). This is supported by the photo of the right eye, showing erythema and a semi-dilated pupil, relative to the left eye. Immediate management involves a combination of eye drops, typically including a carbonic anhydrase inhibitor such as topical brinzolamide.

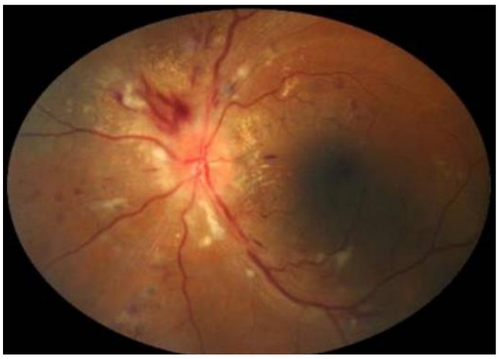
Topical dexamethasone is a corticosteroid used to reduce inflammation in a number of eye diseases. It does not routinely play a role in AACG.

Topical ganciclovir is used in the management of dendritic/herpetic keratitis. Rather than a fixed, dilated pupil, fluorescein staining would reveal a branching pattern.Topical gentamicin is used to manage bacterial infections of the eye, such as bacterial keratitis. The pupil would not be fixed and dilated as seen here, but the eye would be generally red in appearance.

Topical latanoprost may be used to treat residual angle-closure glaucoma after laser peripheral iridotomy. It is not used in the acute setting.

Question 11 of 299

A 76-year-old man is routinely reviewed as part of a health checkup. During this process, his eyes are examined using retinal photography and the following image is noted:



He reports generally reduced visual acuity and sometimes double vision.

He takes a number of medications, including amlodipine, losartan, salbutamol, metformin, paracetamol and codeine.

Given the photo above, what is the most likely diagnosis?

A. Central retinal artery occlusion
B. Central retinal vein occlusion
C. Diabetic retinopathy
D. Hypertensive retinopathy
E. Vitreous haemorrhage

D. Hypertensive retinopathy

The findings on the photo above are indicative of hypertensive retinopathy. The photo shows some generalised arterial narrowing and some evidence of silver wiring - a silvery/white appearance of retinal arterioles if they have become occluded. A flame haemorrhage is also visible in the top left area, as well as exudates scattered across the image. Together, these are all featuresof hypertensive retinopathy.

Central retinal artery occlusion classically presents with retinal whitening and a cherry-red spot opacification of the nerve fibre layer as it becomes oedematous due to ischaemia, with the cherryred spot being due to fovea sparing (no nerve fibre layer over the fovea).

Central retinal vein occlusion would also include features such as flame haemorrhages, as well as cotton-wool spots - fluffy white patches on the retina caused by damage to nerve fibres and the resulting accumulation of material within the nerve fibre layer. However, the other findings visible here, such as silver wiring and arteriovenous (AV) nicking, are more indicative of hypertensive retinopathy.

Diabetic retinopathy may have some similar findings as hypertensive retinopathy on fundoscopy and can include haemorrhages and cotton-wool spots. Hypertensive retinopathy can be distinguished by the presence of silver wiring and AV nicking, which are both present in this case. Furthermore, with advanced proliferative retinopathy, neovascularisation would be observed.

The haemorrhage visible in this case is a flame haemorrhage, rather than a vitreous haemorrhage. Vitreous haemorrhages appear as a well-defined accumulation of blood within the vitreous body, obscuring the retina, rather than on the retina itself. They normally present with a sudden, painless loss or blurring of vision.

Question 12 of 299

A 75-year-old man is referred to you for the management of tremor and mobility issues. On examination, he has a noticeable resting tremor that is worse in the right hand when compared to the left. He also is quite bradykinetic when mobilising and displays mild rigidity. His speech and cognitive function do not appear to be affected. His blood pressure is 125/80 mmHg without any significant postural drop.

You suspect idiopathic Parkinson's disease. Further history taking reveals that this gentleman has had a previous significant gambling problem which is now well controlled.

Which of the following medications, in particular, should be avoided in this patient?

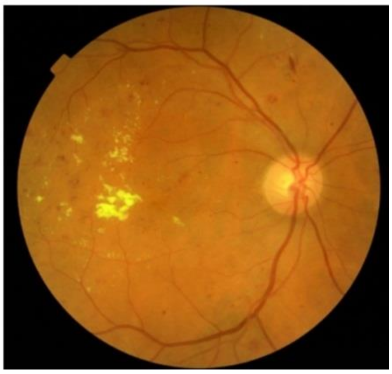
A. Rasagiline
B. Levodopa/carbidopa
C. Pramipexole
D. Entacapone
E. Amantadine

C. Pramipexole

There is an established link between the use of dopamine receptor agonists and serious impulse control disorders. For patients who have a history of habitual behaviours associated with increased risk, this group of medications should be avoided.

Question 13 of 299

A 48-year-old man with a history of type II diabetes mellitus attends the routine diabetic eye screening. He reports good compliance with his diabetes medications. His fundoscopy showed:



What is the most appropriate management for this patient?

A. Intra-vitreal VEGF inhibitor therapy
B. Optimise glycaemic control
C. Routine 2-yearly eye screening
D. Urgent trabeculectomy
E. Vitreoretinal surgery

B. Optimise glycaemic control

The fundoscopic findings of microaneurysms, hard exudates, and blot haemorrhages suggest severe non-proliferative diabetic retinopathy (NPDR). Besides optimising glycaemic control, antihypertensives and statin therapy, pan-retinal laser photocoagulation should be considered in severe NPDR.Optimise glycaemic control should be considered in all patients with non-proliferative or proliferative diabetic retinopathy. Furthermore, tight control of blood pressure and lipid levels may help slow the progression of diabetic retinopathy.

Intra-vitreal VEGF inhibitor therapy is recommended for patients with maculopathy or may be considered in combination with pan-retinal laser photocoagulation to treat severe proliferative diabetic retinopathy which may present with extensive neovascularisation around the disc or elsewhere.

Routine 2-yearly eye screening is incorrect. All patients aged 15 or over with diabetes are advised to have routine diabetic eye screening at least once a year.

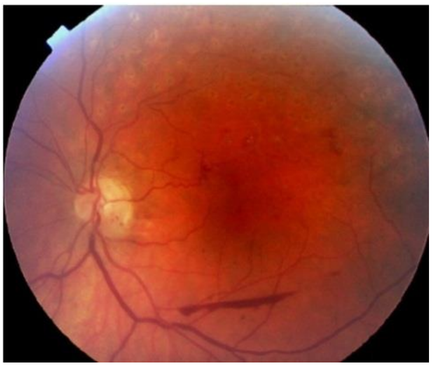
Urgent trabeculectomy , the surgical intervention used to reduce intraocular pressure in acute angle-closure glaucoma. Glaucoma leads to the classical fundoscopic appearance of cupping or increased vertical cup-to-disc ratio, which is not seen in this patient.

Vitreoretinal surgery is recommended for severe or vitreous haemorrhage, which is often a complication of extensive neovascularisation seen in proliferative diabetic retinopathy (PDR). However, this patient does not show any PDR or vitreous haemorrhage features, which may be seen as a red haze of haemorrhage into the vitreous associated with dot-blot haemorrhages, neovascularisation elsewhere or boat-shaped pre-retinal haemorrhages.

Question 14 of 299

A 60-year-old woman with history of proliferative diabetic retinopathy presents to the emergency department with a sudden painless loss of vision in the left eye. On examination, there is reduced visual acuity in the left eye.

Fundoscopy shows:



Given the fundoscopy findings, what is the most likely diagnosis?

A. Central retinal artery occlusion
B. Central retinal vein occlusion
C. Hypertensive retinopathy
D. Retinal detachment
E. Vitreous haemorrhage

E. Vitreous haemorrhage

The central red haze of haemorrhage into the vitreous is characteristic of vitreous haemorrhage. Looking closely at this patient's retina, there are dot and blot haemorrhages (seen as round blots of haemorrhages with indistinct margins) in the upper periphery of the retina. There is alsoevidence of neovascularisation which is seen best in the mid-zone with multiple fine vessels leaving the optic disc into the vitreous. Additionally, in the lower quadrant of the retina, there is a pre-retinal haemorrhage. Together, these features are suggestive of proliferative diabetic retinopathy.

Vitreous haemorrhage is characterised by a red haze of haemorrhage into the vitreous. The associated findings of dot blot haemorrhages, neovascularisation and pre-retinal haemorrhages suggest proliferative diabetic retinopathy, the most common cause of vitreous haemorrhage in adults.

Central retinal artery occlusion is characterised by retinal whitening and thin arteries. Associated findings may include sluggish blood flow (boxcar shape of the arteries), Hollenhorst plaquecholesterol (refractile lesions within the arteries) or large calcific plaques (white lesions within the arteries).

Central retinal vein occlusion results in diffuse retinal 'blood and thunder' haemorrhages. However, this is less common and is not usually associated with other features of proliferative diabetic retinopathy seen in this patient. Risk factors for retinal vein occlusion include advancing age (>55y), hypertension, diabetes, glaucoma, sickle cell anaemia, oral contraceptives and smoking.

Hypertensive retinopathy can cause retinal vascular damage, resulting in flame-shaped haemorrhages associated with arteriolar constriction, arteriovenous nicking, cotton-wool spots, yellow hard exudates, and optic disk oedema, which are not seen in this patient.

Retinal detachment appears as the retina detaches from the typical structure of the eye. In the case of rhegmatogenous retinal detachment, a large bullous separation of the retina may be seen.

Question 15 of 299

A 46-year-old Ghanaian woman was flying from Ghana to San Francisco, USA when she was noticed to be confused and behaving inappropriately on the plane. She was talking loudly to herself, complaining of a headache, and also appeared to be hearing voices. She had one episode of incontinence on the plane. During the transit in London, she was brought to the nearest hospital for investigations.

On examination, she was drowsy and slow to respond to questions. Her temperature was 37.3ºC, heart rate of 98 bpm, blood pressure of 138/92 mmHg, respiratory rate of 16, and oxygen saturations were 100% on air. Her pupils were 3mm bilaterally, equal and reactive. Her neck was supple and there was mild photophobia. Her abbreviated mental test score was 6/10.

Her investigations revealed:

C Reactive protein	24 mg/l
Haemoglobin	128 g/l
White cell count	11.6 x 10^9/L
HIV antibody serology	positive
HIV viral load	19000 copies/ml
CD4+ T lymphocyte count	35 cells/mm³
Na+	136 mmol/l
K+	4.9 mmol/l
Urea	7.2 mmol/l
Creatinine	108 µmol/l
Corrected calcium	2.32 mmol/l
Plasma glucose	5.8mmol/l

Chest X-Ray: Lung fields clear

Computer Tomography (CT) head scan: Hypodense lesions involving the medial temporal regions. Lesions enhance with contrast.

Cerebro-spinal fluid (CSF) analysis:

Opening pressure	20 cmH2O
Protein	1.2 g/L
White cell count	50 per mm³ (predominantly mononuclear cells)
Red cell count	5 per mm³
Glucose	4.8 mmol/l
Gram stain	No organisms seen

What is the next most appropriate management step?

A. Intravenous acyclovir
B. Oral prednisolone
C. Highly Active Anti-Retroviral Therapy (HAART)
D. Intravenous fluconazole
E. Pyrimethamine and Sulfadiazine

A. Intravenous acyclovir

This lady is immunocompromised with a new diagnosis of HIV, and has an altered conscious level, with psychotic symptoms suggestive of encephalitis. Her CT head scan shows the involvement of the medial temporal region, which is highly suggestive of herpes encephalitis, and this is supported by the LP results with raised protein levels, and a predominantly mononuclear white cell picture. Viral polymerase chain reaction (PCR) is the gold standard in the diagnosis of herpes encephalitis, and management for Herpes encephalitis is high-dose intravenous acyclovir to achieve central nervous system penetrance.

Although this lady has advanced HIV infection, there is some evidence that starting HAART may cause an immune reconstitution syndrome, resulting in an initial deterioration in the patient's condition as the immune system is boosted. Thus while she would require HAART, the initial management should be to decrease the burden of infection with acyclovir to prevent a deterioration in her condition.

There is limited evidence for steroid use in HSV encephalitis. There is no evidence of cryptococcal or toxoplasmosis infection from the CT and CSF results, which rules out options (d) and (e) respectively.

Question 16 of 299

A 57-year-old man presents with slurred speech. He was brought in by ambulance as his daughter was concerned when she came to visit and found his speech to be abnormal. On questioning, the patient has not noticed any changes; but the daughter has noticed that he has been drooling and been having difficulty eating for several months. She has not noticed a change in his memory or cognitive function but has been concerned that he has been laughing out of place in conversations. She feels that the changes in her father have been slowly happening over that time. On examination, the patient has slight slurred speech, mild dysarthria and the tongue appears to move abnormally. His limbs are normal on examination, as is the remainder of his cranial nerve examination.

What is the most likely diagnosis?

A. Myotonic dystrophy
B. Stroke
C. Motor neuron disease
D. Multiple sclerosis
E. Syringobulbia

C. Motor neuron disease

The correct answer is motor neuron disease. This a male patient in his 50s who has developed evidence of bulbar signs without any sensory or peripheral involvement. The chronic history makes a stroke less likely, whilst the progressive nature, his age and sex makes multiple sclerosis less likely. The absence of sensory loss or weakness of the arms makes syringobulbia less likely whilst myotonic dystrophy would have more peripheral signs. This is motor neuron disease presenting with bulbar features.

Question 17 of 299

An 82-year-old female presents to Parkinson's disease clinic with increasing number of episodes when she is unable to move at all, each lasting for around 20 minutes and up to 4 times a day. She was diagnosed with Parkinson's disease five years ago and has since been taking Sinemet 125 only.

Which of the therapies is most appropriate to reduce motor fluctuations?

A. Entacapone
B. Deep brain stimulation (DBS) of globus pallidus
C. Reducing L-dopa dose
D. Change to ropinirole
E. Change to selegiline

A. Entacapone

The patient describes increasing frequency of 'on-off' fluctuations, a common complaint in patients who have been diagnosed with PD and medicated with L-dopa for a long time. Selegiline, a monoamine oxidase B inhibitor, while useful as an adjunct, has a very poor evidence base as a monotherapy. There is little evidence that in a more elderly patient, changing to a dopamine agonist has any benefit. The patients L-dopa load should be kept as low as possible but the main problem is the lack of a smooth profile. DBS is premature. Entacapone is a catechol-Omethyltransferase (COMT) inhibitor, which reduces the peripheral breakdown of L-dopa and increases the serum half-life of L-dopa. It has little therapeutic benefit when used alone but is a successful adjunct in smoothing out motor fluctuations.

Question 18 of 299

An 80-year-old male presents to the Parkinson's disease (PD) clinic complaining of jerking and flailing movements of his arm. He says he has no control of them and finds them embarrassing and disabling. However, he reports very few other PD symptoms, with minimal problems with 'off' periods. His past medical history includes PD, diagnosed 8 years ago, and type 2 diabetes. He currently takes Sinemet 250 for Parkinson's disease 5 times a day and metformin 500mg TDS, he has been on the same dose of Sinemet for past 18 months.

What is your most appropriate management?

A. Reduce dose of Sinemet
B. Increase dose of Sinemet
C. Start entacapone
D. Start ropinirole
E. Deep brain stimulation (DBS)

A. Reduce dose of Sinemet

The patient describes involuntary movements on a background of chronic and substantial use of L-dopa, a phenomenon known as L-dopa dyskinesias (LID). The pathophysiology of LIDs remains unclear but the key is to minimise L-dopa dosage if at all possible. As the patient reports minimal other motor symptoms, this is possible in this case. Entacapone smooths the motor profile of Ldopa but does not help with LIDs. DBS is a possible treatment for LIDs changes to L-dopa dose is not possible or if symptoms remain refractory despite L-dopa dose changes.

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A 14-year-old presents to your neurology clinic reporting 9 months of subtle and gradual onset, progressive lower limb weakness. For the past 18 months, he has noticed a difficulty in keeping up with his peers in PE lessons, which he initially put down to 'not being very sporty'. However, he feels weak whenever he walks and has particular difficulty getting up from a chair.

On examination, you notice significantly hypertrophied calves on inspection. Formal examination of power reveals 4- out of 5 bilaterally in shoulder abduction, adduction and normal 5 out of 5 distally. 4- out of 5 is also noted in hip flexion and extension, 4+ in knee flexion and extension, 5 out of 5 in ankle plantar and dorsiflexion. The weaknesses demonstrated are not fatiguable and are persistent. Reflexes are present in all areas, plantars are downgoing. He has no other past medical history. His family history is unknown as he was adopted.

What is the most likely diagnosis?

A. Duchenne muscular dystrophy
B. Becker muscular dystrophy
C. McArdle syndrome
D. Spinal muscular atrophy
E. Inclusion body myositis

B. Becker muscular dystrophy

Progressive proximal muscle weakness appears to be the most important symptoms in this teenage patient. The key differentials can be separated by the age of the patient. Duchenne muscular dystrophy (DMD) is a more severe form of Becker muscular dystrophy (BMD) and patients are normally wheelchair bound by early teens. Similarly, spinal muscular atrophy produces a flaccid paralysis of proximal musculature with absent reflexes, presenting from newborn to under the age of 3.

McArdle disease is a metabolic disorder affecting proximal muscles caused by myophosphorylase deficiency. Patients frequently report a 'second wind' caused by a change in fatty acid metabolism, weakness is hence classically not persistent. Inclusion body myositis classically produces distal to proximal weakness in middle-aged patients. BMD is hence the most appropriate answer.

BMD is an X-linked disorder of dystrophin, a component of skeletal muscle that reinforces the sarcolemma-glycoprotein complex and prevents degradation. Respiratory, cardiac and bulbarmanifestations are common. Diagnosis is made on clinical suspicion, raised CK, EMG suggestive of small polyphasic potentials, a muscle biopsy suggestive of dystrophinopathy and subsequent genetic testing.

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A 76-year-old right-handed female presents with sudden onset flaccid right upper and lower paralysis with complete dysphasia. Her daughter reports her to have been well two hours ago.

On examination, the patients score 0/5 on her right upper and lower limb, at least 4/5 on both left limbs (examination was difficult due to her dysphasia), with a loud carotid bruit. She is also now in atrial fibrillation, a new diagnosis for her. She is well known to the stroke team: 6 weeks ago, she was admitted with a right middle cerebral artery ischaemic stroke, leaving her with minimal residual weakness on her discharge.

During her admission, she was found to have 85% carotid stenosis in her right internal carotid artery and 75% in her left internal carotid artery, for which she declined surgery. Her other past medical history includes hypertension, type 2 diabetes mellitus and dyslipidaemia. She does not take any anticoagulants. A CT head demonstrates a hypodensity in the left middle cerebral artery area distribution, consistent with an acute ischaemic stroke with no areas of haemorrhagic transformation.

What is the most appropriate next course of action?

A. Intravenous alteplase
B. Clopidogrel 75mg
C. Aspirin 300mg
D. Treatment dose low molecular weight heparin
E. Warfarin

C. Aspirin 300mg

This patient has clearly had a new ischaemic stroke. Her NIHSS score and presentation within 4.5 hours would be sufficient to warrant consideration of thrombolysis. However, the key here is to identify the contraindication to thrombolysis: refusing carotid surgery recently is NOT a contraindication. However, a recent stroke or head trauma within 3 months is. The appropriate management would be to load the patient with 300mg aspirin. Clopidogrel 75mg is preferred 14 days after an ischaemic stroke. Anticoagulation is typically started 14 days after the acute stroke, possibly sooner in patients with small infarct sizes and hence a low risk of haemorrhagic transformation.

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A 72-year-old admitted to hospital with VF arrest. The ambulance crew reported an out-of hospital downtime for 3 minutes before CPR was commenced. In A&E resus, CPR was successful and the patient was intubated after the return of spontaneous circulation. 3 days later, on extubation, the ITU consultant notes confusion and bilateral upper limb weakness, confirmed to be new with a collateral history. A CT head, followed by MRI head, demonstrates bilateral small areas of ischaemic change in bilateral posterior parietal lobes, between middle and posterior cerebral artery territories. CT angiography demonstrates 45% RICA stenosis, 60% L ICA stenosis. The cardiac monitor demonstrates atrial fibrillation and echocardiogram demonstrates septal akinesia, consistent with recent MI.

What is the likely cause of this patient's stroke?

A. Cardiac mural thrombus
B. Left carotid artery stenosis
C. Right carotid artery stenosis
D. Hypotension during cardiac arrest
E. Cardioembolic disease

D. Hypotension during cardiac arrest

The neuroimaging describes a posterior watershed infarct, in which the areas of ischaemia lie in the most vulnerable regions of the brain in between two cerebral artery territories. Embolic disease and cardiac thrombi can produce bilateral symptoms. However, the specific areas involved suggest regions most prone to ischaemia during the period of systemic hypoperfusion. The two broad categories of differentials to consider include cardiac pump failure and hypovolaemia. In this case, hypoperfusion due to cardiac arrest is most likely.

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A 52-year-old male is brought to the emergency department by his concerned wife after tripping and falling down a flight of 12 stairs at home, hitting his head on the way down. The patient himself is not concerned and believes he could have stayed at home.

He denies having a headache, reports no nausea or vomiting, no seizures and did not lose consciousness between the fall and when you examine him. He is not taking any regular medications including anticoagulants and remembers the whole episode except for about 20 seconds after landing at the bottom of the floor. On examination, he has no limb weakness or loss of sensation. His pupils are equal and reactive bilaterally.

What is the most appropriate management?

A. Discharge, no further investigations required
B. Discharge, outpatient CT head within 72 hours
C. Observe for 24 hours and discharge if no deterioration
D. CT head immediately
E. CT head within 8 hours of injury

E. CT head within 8 hours of injury

CT head within 8 hours of injury is the correct answer according to NICE guidelines for head injury assessment. This patient has experienced a dangerous mechanism of injury (fall down >3 steps) and has retrograde amnesia (loss of memory for 20 seconds after the event), which are both risk factors that warrant a CT head scan within 8 hours, even in the absence of other concerning features.

CT head immediately would be indicated if the patient had additional concerning features such as GCS <13, focal neurological deficit, seizure, vomiting, or was on anticoagulation. As none of these are present, scanning within 8 hours rather than immediately is appropriate.

Observe for 24 hours and discharge if no deterioration would be excessive for this patient. Inpatient observation is typically reserved for patients with more concerning features or those who have had abnormal imaging. This would be an inefficient use of resources for this patient.

Discharge, outpatient CT head within 72 hours is inappropriate as the timeframe is too long. If imaging is indicated in head injury, it should be performed within 8 hours to identify potentiallyserious pathology that may require intervention.

Discharge, no further investigations required would be unsafe. While the patient appears well, the mechanism of injury and presence of retrograde amnesia mandate further investigation as per NICE guidelines. Missing an intracranial injury could have serious consequences.

The key principle in head injury management is to identify patients who require imaging based on risk factors, even if they appear well, as early intervention for intracranial pathology leads to better outcomes.

Question 23 of 299

A 19 year old woman presents to the general medical clinic for review. She has been referred due to drooping of her eyelids, first on the right and now bilaterally. On further questioning she reveals that she has been struggling to read in dim light and recalls that two family members have required insertion of cardiac pacemakers. Examination reveals partial bilateral ptosis and a generalised ophthalmoplegia in all directions of gaze. Fundoscopy demonstrates central areas of dark pigmentation on a pale fundus.

From the options given which investigation is most likely to reveal the diagnosis?

A. Serum for anti-acetylcholine receptor antibodies

B. Serum for anti-voltage gated calcium channel receptor antibodies
C. Muscle biopsy
D. Karotyping
E. Thyroid function tests

C. Muscle biopsy

A triad of chronic progressive external ophthalmoplegia, pigmentary retinopathy and cardiac conduction defects makes Kearns-Sayer syndrome likely.

Kearns-Sayer syndrome is an example of a mitochondrial disease, a group of rare conditions which many find confusing. Despite their rarity, those candidates who do a rotation in neurology are likely to encounter mitochondrial disease in one form or another and so a basic knowledge is useful.

Kearns-Sayer syndrome is characterised by ptosis and external ophthalmoplegia occurring in the first or second decades of life. Cardiac conduction defects usually occur later and are usually in the form of atrio-ventricular nodal block, often requiring cardiac pacing. Multiple endocrinopathies may be associated and a proximal myopathy may be evident. Serum elevation of lactate and pyruvate is frequently seen, as is the case with many mitochondrial conditions.

The diagnosis should be suspected from the history and clinical features as listed above and diagnosis is confirmed by muscle biopsy which demonstrates characteristic 'ragged red fibres', this is often supplemented by polymerase chain reaction analysis of mitochondrial DNA for mutation detection.

Anti-acetylcholine receptor antibodies are seen in myasthenia gravis, and would be involved in the work-up of any patient with an unexplained bilateral ptosis, but this wouldn't explain the pigmented retinopathy or the family history of pacemaker insertion. This is also the case for antivoltage gated calcium channel antibodies, found in the Lambert-Eaton myasthenic syndrome.

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A 70-year-old man with a history of hypertension and benign prostatic hypertrophy is brought in by ambulance after a fall. He reports he felt dizzy after standing up from his arm chair, stumbled and tripped over his cat. His wife, who witnessed the fall, reports that he then hit his head on the coffee table a lost consciousness for around 1 minute.

She describes no abnormal movements or incontinence. On regaining consciousness he was oriented immediately. He remembers regaining consciousness. He has no headache, dizziness, nausea or vomiting.

On examination, he has a small laceration on his forehead. His pupils were equal and reactive to light. He had no focal neurological deficits. He was a 15 on the Glasgow Coma Scale. His abbreviated mental test score was 10/10.

ECG: Sinus rhythm. 70 beats per minute. No T wave or ST segment changes.

- Blood pressure (lying): 135/75 mmHg
- Blood pressure (standing): 110/60 mmHg
- Haemoglobin 135 g/dl
- Troponin T 1 ng/L
- Urine dip: trace of protein

What is the most appropriate course of action?

A. Admit for CT head within 1 hour
B. Admit for CT head within 8 hours
C. Admit for CT head within 24 hours
D. Discharge to return for CT head next day
E. Discharge with outpatient follow-up, no imaging required

B. Admit for CT head within 8 hours

Nice Guidelines on head injury state that: For adults who have sustained a head injury and have any of the following risk factors, perform a CT head scan within 1 hour of the risk factor being identified:

- GCS less than 13 on initial assessment in the emergency department.
- GCS less than 15 at 2 hours after the injury on assessment in the Emergency Department.
- Suspected open or depressed skull fracture.
- Any sign of basal skull fracture (haemotympanum, 'panda' eyes, cerebrospinal fluid leakage from the ear or nose, Battle's sign).
- Post-traumatic seizure.
- Focal neurological deficit.
- More than 1 episode of vomiting.'

The patient does not meet any of these criteria, so CT head within 1 hour is not mandatory. The guideline then also states:

'For adults with any of the following risk factors who have experienced some loss of consciousness or amnesia since the injury, perform a CT head scan within 8 hours of the head injury:

A. Age 65 years or older.
B. Any history of bleeding or clotting disorders.
C. Dangerous mechanism of injury (a pedestrian or cyclist struck by a motor vehicle, an occupant ejected from a motor vehicle or a fall from a height of greater than 1 metre or 5 stairs).
D. More than 30 minutes' retrograde amnesia of events immediately before the head injury.'

As this gentleman had a period of unconsciousness and is over 65, he should have a CT head within 8 hours.

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A 32-year-old man had a compressive pituitary macroadenoma surgically removed in France 4 weeks ago and has been on pituitary hormone replacement since, including growth hormone. Surgery was uncomplicated and he initially made a good recovery. He has no other past medical history. He has now developed, over the last 1-2 weeks, poor balance, with a broad-based gait. He has also become extremely forgetful. On examination, he has an ataxic gain and you also observe occasional myoclonic limb movements. His MMSE

score is 22/30. Clinical examination is otherwise unremarkable, as are routine blood investigations.

Given the likely diagnosis, what might you expect to find on his MRI brain?

A. Areas of demyelination in the cerebellum
B. An area of high T2 signal in the right middle cerebral artery territory with restricted diffusion on diffusion-weighted sequences
C. High T2 signal in the posterior thalamus
D. Leptomeningeal enhancement
E. Loss of grey-white matter differentiation

C. High T2 signal in the posterior thalamus

The diagnosis is Creutzfeldt-Jakob disease (CJD) from an iatrogenic source.

The use of prion-contaminated medication, grafts and instruments may result in iatrogenic CJD. The current worldwide total of growth-hormone-associated cases of CJD is 226. Most cases occurred in France (119 cases/1,880 recipients; attack rate 6.3%), the United Kingdom (65 cases/1,800 recipients; attack rate 3.6%), and the United States (29 cases/7,700 recipients; attack rate 0.4%). No new case has been identified since 2008 but would be important to recognise this condition and how it presents if it did, as it is invariably fatal -so patients need to be made aware of their prognosis.

The clinical features of the disease vary according to the route of inoculation.

Central inoculation, eg from infected neurosurgical instruments, results in a rapidly progressive neurodegenerative disease similar to sporadic CJD, characterised by ataxia, dementia, myoclonus, rigidity, and akinetic mutism. Survival is 2-12 months.

Peripheral inoculation, eg from the use of the old human growth hormone products, results in a slightly less acute disease - characterised by progressive cerebellar ataxia and dementia. Survival is 8-18 months.

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A 75 year old man with Parkinson's disease is brought to see you in clinic by his son. He has become increasingly concerned that his neighbours have been watching him, and have put wiring throughout his walls to monitor his movements. He has also been describing visual hallucinations of animals climbing up his walls. His son is concerned that he has become increasingly anxious. He is currently on co-careldopa, ropinirole, and rasagiline.

What is the best course of action?

A. Initiate a 'drug holiday' withholding all but co-careldopa for 1 week
B. Reduce ropinirole
C. Reduce rasagiline
D. Refer for cognitive behavioural therapy
E. Start quetiapine

B. Reduce ropinirole

This gentleman is displaying psychotic symptoms. Psychosis in Parkinson's disease (PD) can be due to the disease process itself (non-motor feature of PD), or medication side effect. It is sensible to perform a medication review and alter any potentially precipitating drugs.

Ropinirole is a dopamine agonist and is associated with psychosis, and in particular visual hallucinations. It is also important to warn all patients initiated on dopamine agonists of the potential for impulsive behaviour disorders including gambling and excessive spending.

Abrupt discontinuation of medication is never indicated in Parkinson's disease since this can lead to rapid deterioration and worsening of symptoms.

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You are asked for advice on a 57-year-old man who presented to primary care 12 weeks after an inferior myocardial infarction. He has had symptoms of persistent low mood that varies during the day, tearfulness and hopelessness. He has lost 6kg of weight in the past 3 months and his BMI is currently 19. He has fleeting thoughts of suicide but would not act on this as he does not want to hurt his family. You are told that 20 years ago, following an overdose of paracetamol, he was prescribed Citalopram and diagnosed with depression.

The GP tells you that other than the recent MI, he also has a diagnosis of Atrial Fibrillation, for which he is on warfarin and he has suffered from epigastric pain for the past 2 years. He had an OGD 1 year ago where treatment of a duodenal ulcer with adrenaline was required. Since then his symptoms of epigastric pain have decreased but are still present. There has been no repeat OGD.

What would be the safest course of action?

A. Prescribe Phenelzine
B. Prescribe Sertraline
C. Prescribe Mirtazapine
D. Prescribe Imipramine
E. Refer for 2 month wait CBT

C. Prescribe Mirtazapine

The answer is C Mirtazapine. E is an unsafe option because of the significant amount of weight loss to a point where is BMI is close to him being underweight. 2 months is too long to wait to offer an intervention

Sertraline is an SSRI. SSRIs are known to interfere with platelet aggregation and increase the risk of a GI bleed. As this individual has a history of a bleeding duodenal ulcer and continuing symptoms, Sertraline would be an unsafe option. SSRIs also interact with warfarin

Phenelzine is a MAO inhibitor and Imipramine is a TCA. TCAs have been linked to ischaemic heart disease and sudden cardiac death. TCAs are contraindicated in those with a history of ischaemic heart disease. TCAs are also dangerous in overdose which may be a significant risk for this individual. MAO inhibitors are thought to be arrhythmogenic and decrease LVF. They are notrecommended in those with cardiovascular disease.

Mirtazapine is a good choice as it is not known to cause cardiac conduction disturbances and there is good evidence of its safety post-MI. It also causes increased appetite and weight gain which would be advantageous for this individual at this time. Mirtazapine does not interact with warfarin and it does not affect platelet aggregation. Generally speaking, Mirtazapine is a safe drug and is often prescribed for the elderly and people with multiple medical problems. Mirtazapine is also relatively safe in overdose to other antidepressants.

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A 28-year-old female psychiatric nurse presents to the emergency department following a 2minute tonic-clonic generalised seizure, which self-terminated. Whilst taking the history the following morning, she tells you that over the last 48 hours she has become increasingly unwell. She has been feeling increasingly anxious and has been having insomnia and vivid nightmares which wake her from sleep. She says that everything around her no longer looks real but more like a photocopy. Bowel and bladder movements have been normal. She feels generally weak and asks the lights to be dimmed in the examination room. When the nurse bell goes off in the next cubicle, she has to cover her ears.

On examination, she appears anxious, she is perspiring, respiratory rate 16/min, blood pressure 142/86 mmHg, heart rate 115/min regular, sats 98% on air. You notice a fine tremor, especially in the hands and eyelids. GCS = 15. Oriented in time place and person. When you examine her, she tells you that it feels as if her legs are floating off the bed even though they are stationary. There is no flushing of the face

From looking at the computer records, you can see that she has a history of panic disorder treated with PRN lorazepam and sertraline 1 year ago. and has been treated for depression in the past. You also note from your records that she presented to A+E 5 days ago due to stress as she was sacked from her job.

Hb	136 g/l
Platelets	232 * 10 /l
WBC	6.9 * 10 /l
Na	142 mmol/l
K	3.8 mmol/l
Urea	6.2 mmol/l
Creatinine	81 µmol/

What is the most likely diagnosis?

A. Benzodiazepine withdrawal
B. Atypical Panic attack secondary to panic disorder
C. LSD intoxication
D. Benzodiazepine excess
E. Opiate withdrawal

A. Benzodiazepine withdrawal

Benzodiazepine withdrawal can cause physical symptoms such as tachycardia, sweating, fine tremor which is more prominent in the tongue, eyelids and hands. It can also cause nightmares and insomnia, anxiety and phobic symptoms as well as seizures, hypersensitivity to light, sounds and touch. They can also experience derealisation (an unpleasant subjective experience that the outside world does not look real to them) and kinaesthetic hallucinations. Other symptoms include malaise and tinnitus and it sometimes can present as a delirium.

Some factors in the history also point towards this as being a likely diagnosis. She has a history of panic disorder and previous prescription for benzodiazepines when she may have developed dependence. As a psychiatric nurse, she may have access to benzodiazepines off prescription. The fact that she has recently lost her job may mean that she has run out of her supply and is now suffering from withdrawal syndrome.

An overdose of benzodiazepines would not present with tachycardia, sweating and anxiety. LSD intoxication could present with hallucinations, anxiety, tachycardia and sweating, however the duration of symptoms is too long for LSD unless multiple doses were taken and LSD intoxication. LSD is not linked to causing seizures. This presentation has many features of a panic attack but would not explain the seizure or kinaesthetic hallucination.

Opiate withdrawal could present in a similar way, however, you would expect to see a marked degree of GI disturbance in this syndrome and is not linked with kinaesthetic hallucinations or derealisation. In this question, the history of previous benzodiazepine usage makes this answer less likely

Question 29 of 299

A 64 year old man presented to the Emergency Department after becoming unwell at home. His wife reported that the patient experienced a sudden onset and severe headache while watching television. Shortly afterwards she found him to have become confused and drowsy and an ambulance was called. On arrival in the Emergency Department, the patient suffered a witnessed tonic-clonic seizure, self-terminating after two minutes.

The patient had known hypertension and hypercholesterolaemia and had suffered a non-ST elevation myocardial infarction two years before, treated with a drug-eluting stent to the left anterior descending artery. Regular medications included ramipril 10 mg OD, bisoprolol 10 mg OD, bendroflumethiazide 2.5 mg OD, simvastatin 40 mg OD and aspirin 75 mg OD. The patient's wife reported that compliance with anti-hypertensive medications had been inconsistent and that controlling the patient's blood pressure had been an on-going problem over the previous two years. The patient was a retired builder and ex-smoker.

On examination, the patient was drowsy (GCS M4V2E3) but was protecting his own airway. Pupils were equal and reactive. The patient was spontaneously moving all his limbs and had downgoing plantar reflexes. Cardiovascular, respiratory and abdominal examination was unremarkable. Initial observations and investigations are listed below.

Blood pressure 220 / 115 mmHg Heart rate 89 beat / minute O2 sats (15 L O2) 100 % Respiratory rate 19 / minute Temperature 37.1ºC.

CT brain: no extra-axial bleeding or collection; no intracerebral haemorrhage; no evidence acute ischaemic stroke; no subarachnoid blood; normal ventricular system; mild small vessel disease in keeping with patient's age.

Lumbar puncture:

CSF red cells	3/ mm³
CSF white cells	3 / mm³
CSF gram stain	unremarkable
CSF glucose	60 % serum level
protein	0.5 g / L
CSF	negative for bilirubin and xanthochromia

Given persistent reduced GCS and need to obtain blood pressure control, patient was intubated and transferred to the neuro intensive care unit. Following discussion with neurological team further imaging was arranged

MR brain with angiography: structurally normal brain as per previous study; bilateral symmetric vasogenic oedema involving the subcortical white matter in the parietal-occipital, posterior temporal and posterior frontal lobes; MRA unremarkable without any area of stenosis or vasospasm.

What is the correct diagnosis?

A. Reversible cerebral vasoconstriction syndrome
B. Posterior reversible leucoencephalopathy syndrome
C. Acute disseminated encephalomyelitis
D. Cerebral venous thrombosis
E. Pituitary apoplexy

B. Posterior reversible leucoencephalopathy syndrome

Posterior reversible leucoencephalopathy syndrome may present with thunderclap headache, usually followed rapidly by confusion, seizures and visual symptoms. CT brain and lumbar puncture results are usually normal or near normal. The most common causes of PRES are hypertensive encephalopathy (as in this case) and eclampsia. Hypertension is commonly observed. Diagnosis is made by evidence of vasogenic brain oedema on MRI brain. PRES is often associated with reversible cerebrovascular vasoconstriction syndrome with vasospasms on angiography (although not in this case). Acute disseminated encephalomyelitis does not present with thunderclap headache.

Cerebral sinus thrombosis and pituitary apoplexy can both present with thunderclap headache and normal CT brain and LP although the other results and clinical picture in this case are not consistent with these diagnoses. Acute disseminated encephalomyelitis does not present with thunderclap headache.

Question 30 of 299

A 61-year-old man is referred neurology after he noticed a tremor. He has a tremor affecting both his arms and his wife has noticed he is slow with activities and has struggled with his balance more recently. Further questioning revealed the patient has had some issues with urinary incontinence and erectile dysfunction.

His past medical history includes hypertension, myocardial infarction and hypercholesterolaemia. He takes ramipril, bisoprolol, aspirin and simvastatin.

On examination, he is found to increased tone, but power was normal. However, his coordination was impaired. The rest of the physical examination was normal. His GP did some blood tests prior to the referral, the results of which are shown below:

Thyroid stimulating hormone (TSH)	0.4 mU/L	(0.5-5.5)
Free thyroxine (T4)	12 pmol/L	(9.0 - 18)
Hb	125 g/L	(135-180)
WBC	5.7 * 10 /L	(4.0 - 11.0)
Na	139 mmol/L	(135 - 145)
K	4.3 mmol/L	(3.5 - 5.0)
Urea	6.5 mmol/L	(2.0 - 7.0)
Creatinine	148 µmol/L	(55 - 120)

What is the most likely cause of this man's presentation?

A. Idiopathic parkinson's disease
B. Multiple system atrophy
C. Progressive supranuclear palsy
D. Drug induced parkinsonism
E. Hyperthyroidism

B. Multiple system atrophy

Erectile dysfunction is an early autonomic feature of multiple system atrophy

This patient has presented with Parkinsonism (tremor, bradykinesia and rigidity). There are lots of causes of Parkinsonism.

In this case, the clues to working out the diagnosis are the balance issues, coordination problems, urinary incontinence and in particular the erectile dysfunction. This points to a diagnosis of multiple system atrophy.

In Parkinson's disease, the onset of tremor is usually unilateral at the beginning. It is typically described as being present at rest and disappears on movement. Other features include micrographia.

Progressive supranuclear palsy is a Parkinson's plus syndrome. Differentiating features are difficulty with moving the eyes vertically, slurring of speech and difficulty swallowing.

Drug-induced Parkinsonism is usually seen with antipsychotics and is reversible upon cessation of the causative drug. None of the medications listed cause drug-induced Parkinsonism.

Tremor can be a feature of hyperthyroidism. However, there are no other features in the history to support this diagnosis such as weight loss despite increased appetite, heat intolerance, sweating, diarrhoea etc.

Question 31 of 299

A 78-year-old man presents to the acute medical unit with an episode of loss of consciousness. He states that he suffered a headache earlier that day. He describes it as a sudden onset headache at the back of his head which reached 10/10 severity within a few seconds.

You organise the following investigations: CT head Blood in the sulci, fissures, basal cisterns and ventricles

What is the next most appropriate management?

A. Cerebral CT angiography

B. Lumbar puncture
C. MRI head
D. Endovascular clipping
E. Endovascular coiling

A. Cerebral CT angiography

In non-traumatic subarachnoid haemorrhages, a CT angiogram should be performed to identify intracranial aneurysms

The CT scan has confirmed a subarachnoid haemorrhage (SAH). As it is spontaneous, and not traumatic, it is likely to be an aneurysmal SAH. However to confirm this the patient needs urgent angiography - either CT or MRI. Once an aneurysm is confirmed radiologically the patient can undergo endovascular clipping or coiling.

A lumbar puncture is not indicated in this case as the CT has confirmed the diagnosis of SAH. A lumbar puncture should be performed 12 hours after the onset of headache in patients who have a negative CT scan.

MRI head would confirm a haemorrhage, however angiography is required to definitively confirm an aneurysm.

Question 32 of 299

A 18-year-old man presents to the clinic complaining of both motor and verbal tics which are causing embarrassment. He was diagnosed with Tourette syndrome when he was 14-years old. He initially tried habit reversal therapy (HRT), which partially worked however his symptoms are now the worst they have ever been. He has no other significant past medical history and takes no regular medicines.

You decide to prescribe a drug which will block the effects of dopamine in the basal ganglia.

What treatment will you prescribe?

A. Methylphenidate
B. Risperidone
C. Dextroamphetamine
D. Lamotrigine
E. Clozapine

B. Risperidone

Antipsychotics have the greatest efficacy at treating tics

Tics are classed as a hyperkinetic movement disorder consisting of repetitive, stereotyped motor or vocal movements that may be simple or complex. There is an urge to perform the movement with relief after performing the movement. They are caused by enhanced dopamine levels. Therefore dopamine antagonists such as the antipsychotics are first line pharmacological agents. Both risperidone and clozapine are antipsychotics, however due to the potentially life threatening side effects of clozapine (e.g. myocarditis and neutropaenia) it is reserved as a third line agent.

Methylphenidate, dextroamphetamine, and, lamotrigine are known drug induced causes of tics.

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A 24-year-old man presents with lower limb weakness. He states that he had an episode of food poisoning two weeks ago which consisted of severe abdominal pain and profuse non-bloody diarrhoea. Following this he noticed progressive weakness in his legs. His past medical history is largely unremarkable, however, he does admit to intravenous drug use with heroin. He last injected heroin three weeks ago.

On examination, you note weakness of ankle plantar flexion (MRC grade 3), ankle dorsiflexion (MRC grade 3), knee flexion (MRC grade 4) and knee extension (MRC grade 4). The ankle jerk and knee jerk are only present on reinforcement. There is a loss of sensation to the mid-calves. A neurological examination was otherwise unremarkable.

What is the most likely diagnosis?

A. Acute inflammatory demyelinating polyradiculoneuropathy (AIDP)
B. Acute motor axonal neuropathy (AMAN)
C. Botulism
D. Miller Fisher syndrome
E. Tetanus

A. Acute inflammatory demyelinating polyradiculoneuropathy (AIDP)

Worsening lower limb weakness following gastroenteritis - think Guillain-Barre

Acute inflammatory demyelinating polyradiculoneuropathy (AIDP) is correct. The most likely diagnosis is Guillain-Barre syndrome. The clinical features are most in keeping with AIDP which is the most common subtype. The preceding gastroenteritis is classic of this condition with approximately 30% of cases being provoked by Campylobacter jejuni infection.

Acute motor axonal neuropathy (AMAN) is incorrect. This is a less common subtype of Guillain Barre syndrome which presents with motor involvement only. There is often early and severe respiratory involvement. The presence of sensory involvement, in this case, makes this a less likely diagnosis.

Botulism is incorrect. Botulism is characterised by a descending, flaccid paralysis, in contrast to Guillain-Barre syndrome which presents with an ascending paralysis. It is important to note that the patient is an intravenous drug user which is a risk factor for botulism. However, this method of transmission would not explain the gastroenteritis features. These can of course be related to food-borne botulism, however regardless the clinical features of an ascending paralysis are more in keeping with Guillain-Barre syndrome.

Miller Fisher syndrome is incorrect. This subtype of Guillain-Barre syndrome is characterised by areflexia, ophthalmoplegia, and ataxia. Limb weakness in this subtype is uncommon making this a less likely diagnosis.

Tetanus is incorrect. Tetanus is a life-threatening disease characterised by a descending spastic paralysis. Although the causative bacteria Clostridium tetani has been found in contaminated heroin, the clinical features of an ascending paralysis are more suggestive of Guillain-Barre syndrome.

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A 23-year-old man is brought into the Emergency Department with a severe headache.

He was awoken from sleep early this morning with the most severe headache he has ever experienced. He describes an immediate onset posterior headache that has not improved at all. He has felt his vision slightly blurred and vomited 3 times since his arrival in the department. You perform an urgent CT brain which reveals a subarachnoid haemorrhage.

What investigation is now indicated?

A. CT angiogram
B. Digital subtraction angiography
C. Lumbar puncture
D. MRI brain
E. Ophthalmoscopy

A. CT angiogram

In non-traumatic subarachnoid haemorrhages, a CT angiogram should be performed to identify intracranial aneurysms

The next step when a subarachnoid haemorrhage is confirmed is to determine whether the haemorrhage is aneurysmal or not and so the correct answer is a CT angiogram.

Subarachnoid haemorrhage has been confirmed on plain CT and so a lumbar puncture to look for xanthochromia is not necessary and therefore incorrect. This would be the correct answer if CT had not revealed a cause for the headache and you were still suspicious of a subarachnoid haemorrhage.

MRI brain is incorrect. It will not necessarily show an aneurysm so may not be helpful and anangiogram would still need to be performed.

Digital subtraction angiography is incorrect. It is sometimes performed by neurosurgeons or interventional radiology to look for an aneurysm but CT angiography is quicker and safer to confirm this.

Ophthalmoscopy is also incorrect and has little value here as the diagnosis and raised intracranial pressure are already known.

Question 35 of 299

A 35-year-old female presents to the Emergency Department with worsening lower limb weakness over a three day period. She is now unable to walk and feels that her fingers are becoming clumsy. On examination her heart rate is 65 beats per minute and regular, her blood pressure is 125/70 mmHg and her respiratory rate is 20 breaths per minute. She has absent ankle and knee jerks and reduced reflexes in the upper limbs.

Her cerebrospinal fluid (CSF) study shows an elevated protein with a normal white cell count.

Given her likely diagnosis, which of the following parameters is most important to measure throughout her admission?

A. Lower limb power
B. Serial CSF protein levels
C. Forced expiratory volume over 1st second (FEV1)
D. Postural blood pressure
E. Forced vital capacity (FVC)

E. Forced vital capacity (FVC)

FVC is used to monitor respiratory function in Guillain-Barre syndrome

The most common cause of death in patients with the severe Guillain-Barre Syndrome (GBS) is respiratory failure. This is due primarily to weakness in chest wall muscles, but may also be due to bulbar dysfunction and aspiration. Patients may require intensive care admission and ventilatory support.

For this reason, it is of great importance that forced vital capacity (FVC) is measured on a routine basis and results documented so that additional support can be sought as soon as possible. FEV1 may be preserved in early deterioration as it is not a measure of vital capacity or a marker of chest wall expansion. Therefore, a reduction in FVC is a more sensitive marker of deterioration.

Question 36 of 299

A 17-year-old man is referred by the emergency department with weakness of his lower limbs and hands, worsening over the last 2 days. He is having increasing difficulty walking and is becoming very clumsy and dropping things. He burnt his hand yesterday in the kitchen without realising until afterwards that he had done it. He also complains of feeling dizzy on standing but has no chest pain or palpitations. He is normally fit and well, although did suffer a bought of diarrhoea and vomiting 4 weeks ago. He is on no regular medication and denies any alcohol or smoking history.

On examination he is afebrile, his blood pressure is 128/79 mmHg on lying but on standing it drops to 90/60 mmHg. His respiratory rate is 20/min and his saturations are 97% on air. His cardiovascular, respiratory and abdominal examinations are normal. Neurological examination reveals symmetrical distal weakness of his lower limbs with relative proximal sparing and altered sensation to fine touch, vibration and proprioception. He has similar findings in his upper limbs. He is suspected of having Guillain-Barre syndrome and following blood tests, CT head and lumbar puncture is started on IV immunoglobulins. Despite this, he begins to become increasingly short of breath and he is referred to ITU for respiratory support.

What respiratory parameter is used to determine if invasive ventilator support is needed?

A. pO2 on ABG < 8kPa
B. FEV1:FVC ratio < 0.7
C. Saturations on 15 litres O2 less than 93%
D. FVC < 15ml/kg
E. Peak flow of less than 300ml

D. FVC < 15ml/kg

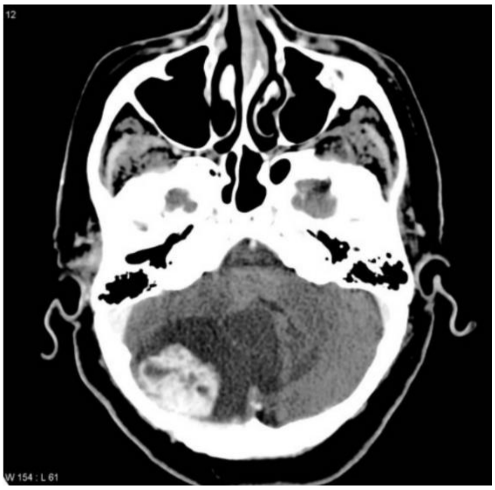
FVC is used to monitor respiratory function in Guillain-Barre syndrome

This man has Guillain-Barre syndrome with autonomic involvement and compromise to his respiratory function. The parameter used to assess whether a patient needs ventilator support is an FVC <15-20ml/kg. low pO2, low saturations and low peak flow are all indicatory of poor respiratory function but FVC is the parameter used in these cases. FEV1:FVC ratio is not used.

Question 37 of 299

A 26-year-old man is admitted for investigation after having increasing trouble walking. On admission he is found to have a severely ataxic gait and is unable to walk in a straight-line. For many weeks he has been complaining of visual problems, dizziness, headaches and clumsiness.

On examination he is noted to have horizontal nystagmus, an intention tremor and past pointing. On fundoscopy a large, erythematous retinal lesion is present surrounded by tortuous, dilated vessels.



His father had similar problems in his 20's. A CT scan is arranged:

What is the most likely underlying diagnosis?

A. Friedreich's ataxia
B. Ataxic telangiectasia
C. Neurofibromatosis
D. Tuberous sclerosis
E. Von Hippel-Lindau syndrome

E. Von Hippel-Lindau syndrome

Von Hippel-Lindau syndrome

Retinal + cerebellar haemangiomas = Von Hippel-Lindau syndrome

The correct answer is Von Hippel-Lindau syndrome. Von Hippel-Lindau (VHL) syndrome is a rare, autosomal dominant disorder characterized by the development of various benign and malignant tumours, including hemangioblastomas in the central nervous system (CNS), retinal angiomas, renal cell carcinomas, and pheochromocytomas. The patient's presentation with ataxia, nystagmus, intention tremor, past pointing, and a retinal lesion surrounded by dilated vessels is highly suggestive of CNS hemangioblastoma and retinal angioma associated with VHL. A cerebellar lesion is also clearly seen in the neuroimaging. The family history of similar problems in his father also supports this diagnosis.

Friedreich's ataxia is an autosomal recessive inherited disorder that primarily affects the spinal cord and peripheral nerves. It typically presents with progressive limb and gait ataxia, dysarthria, loss of deep tendon reflexes, sensory neuropathy, and skeletal deformities such as scoliosis. Although Friedreich's ataxia can cause some similar neurological symptoms to VHL syndrome (e.g., ataxia), it does not explain the retinal lesion or strong family history seen in this case.

Ataxic telangiectasia, also known as Louis-Bar syndrome or Ataxia-telangiectasia (A-T), is an autosomal recessive disorder characterized by cerebellar degeneration leading to progressive ataxia along with oculocutaneous telangiectasias. Patients often present with recurrent sinopulmonary infections due to immunodeficiency. While some features like ataxia overlap with VHL syndrome in this case, A-T would not account for the retinal lesion or CT scan findings.

Neurofibromatosis refers to a group of genetic disorders causing the development of benign tumours, most commonly neurofibromas, in the nervous system. There are two main types: Neurofibromatosis type 1 (NF1) and Neurofibromatosis type 2 (NF2). NF1 is characterized by multiple cafe-au-lait spots, axillary and inguinal freckling, Lisch nodules in the iris, and neurofibromas. NF2 is associated with bilateral vestibular schwannomas (acoustic neuromas), meningiomas, and ependymomas. Neither type of neurofibromatosis would explain the retinal lesion or specific neurological symptoms seen in this case.

Tuberous sclerosis is an autosomal dominant disorder characterized by the formation of benign hamartomas in multiple organs including brain, skin, kidneys, heart, lungs, and eyes. Common features include seizures, cognitive impairment, facial angiofibromas, ash leaf spots on the skin and renal angiomyolipomas. Although some aspects of this syndrome might overlap with VHL syndrome (e.g., CNS involvement), it does not account for the specific retinal lesion described or other clinical findings in this case.

Question 38 of 299

A 32-year-old man is admitted to the neurology ward with a new leg weakness. He describes progressive weakness of his legs and now also his hands, which initially started 7 months ago and has progressed very slowly. He has minimal sensory disturbance in all four limbs but on examination of the legs he has grade 3/5 power throughout with absent reflexes and tone is not raised. He has no past medical history and no recent travel. A set of bloods on admission show the following:

Hb	141 g/l (115-165 g/l)	Na	141 mmol/l (135-145 mmol/l)
Platelets	301 x 10 /l (150-450 x 10 /l)	K	4.1 mmol/l (3.5-5.2 mmol/l)
WBC	6.7 x 10 /l (2-11 x 10 /l)	Urea	4.1 mmol/l (2.5-6.5 mmol/l)
Neuts	5.1 x 10 /l (1.5-7 x 10 /l)	Creatinine	81 µmol/l (60-100 µmol/l)
Lymphs	1.2 x 10 /l (0.5-1.5 x 10 /l)	CRP	0.9 mg/l (<5 mg/l)

A non-contrast CT head and a diagnostic lumbar puncture are also performed which show the following:

CT Head	No mass lesions, haemorrhage or acute major territory infarct seen. Ventricles of normal diameter. No skull vault fractures.

CSF Protein	611 mg/l (140-450 mg/l)
CSF WBC	1 x 10 /l (<5 x 10 /l)
CSF Glucose	2.9 mmol/l (2.3-4.5 mmol/l)
CSF Electrophoresis	Normal pattern of IgG banding

Given the most likely diagnosis, which of the following treatments is most appropriate for this patient?

A. Conservative management
B. Therapeutic lumbar puncture
C. Intravenous antimicrobials
D. Intravenous immunoglobulin
E. High-dose steroids

E. High-dose steroids

CIDP is like GBS but with a more chronic onset and treated with steroids

From the history and clinical findings here, the most likely diagnosis would be chronic inflammatory demyelinating polyneuropathy or CIDP. The two main differentials would be Guillain-Barre syndrome and multiple sclerosis. Guillain-Barre syndrome would be a far more acute presentation with a rapid deterioration and multiple sclerosis would not produce such a global picture and in addition there would also be oligoclonal bands present on electrophoresis of the CSF. The chronic picture and high CSF protein are both in keeping with a diagnosis of CIDP above the others.

CIDP is an autoimmune inflammatory neuropathy which has an ongoing disease process and thus it responds well to immunosuppressive therapy. The most appropriate first line therapy therefore is high-dose steroids as this will achieve the goal of reducing inflammation and immunosuppressing a patient with a single drug. Therapeutic lumbar punctures are not indicated as there is no evidence for this being due to benign intracranial hypertension and antibiotics are also not indicated given the lack of infection markers.

Question 39 of 299

A 27-year-old man is brought to the hospital having become unwell at a party. On arrival, he is drowsy and only responds to voice. His airway appears patent and his chest is clear. Pupils are 4mm and reactive to light. Observations are as follows.

- Respiratory rate 12 breaths /min
- Oxygen saturations 95% on air
- Heart rate 84 beats /min
- Blood pressure 117/65mmHg
- Temperature 36.7ºC

Due to his reduced conscious level, you organise a CT head. While there, a friend phones the hospital and tells you he was seen taking some tablets at the party roughly 90 minutes ago and they have since found empty packets of diazepam in the house.

He returns from CT head which is reported as normal. You are asked to see him urgently as he is now unresponsive and making snoring sounds. These improve with a jaw thrust but recur if this is released. The examination is otherwise the same as on arrival.

What is the best course of action?

A. Activated charcoal
B. Flumazenil
C. Haemodialysis
D. Intubation and ventilation
E. Naloxone

D. Intubation and ventilation

Benzodiazepine overdose is best managed supportively and with airway protection and ventilation if needed. Flumazenil should be avoided unless for reversal of anaesthesia

The correct answer is intubation and ventilation. Drug ingestion leading to CNS depression is common and may be due to a variety of substances. This picture with normal observations and normal pupils should raise the suspicion of benzodiazepine use, especially with the empty packets found at the house. The mainstay of treatment in such a case is supportive, with securing the airway the main priority in the case described.

Activated charcoal is incorrect. This can bind some substances and minimise their effects if given sufficiently early after ingestion. However, its role in benzodiazepine overdose is uncertain. Toxbase advises that it can be considered within the first hour after ingestion provided the airway is protected. In this case, the patient is ou twith this timeframe and the airway is unsafe.

Flumazenil is incorrect. This antidote is effective in reversing the effects of benzodiazepine toxicity. However, reversal of an unknown quantity can lead to rebound seizures which are then refractory to benzodiazepine therapy as the receptor is blocked by the drug. Flumazenil should generally only be used in an iatrogenic overdose where the exact quantity is known. In all other cases, supportive treatment such as airway management is the mainstay.

Haemodialysis is incorrect. This is used for the removal of some toxic agents such as lithium. However, it does not remove benzodiazepines and would not be effective here.

Naloxone is incorrect. This is a highly effective antidote for opiate toxicity and acts quickly enough that it could avoid the need for intubation in such cases. However, the clinical picture is not suggestive of opiate toxicity. This generally presents with other features such as myoclonic jerks and pinpoint pupils. Opiates are also more commonly used by injection rather than as tablets.

Question 40 of 299

A 37-year-old man is being reviewed in the eye clinic for painless loss of vision bilaterally. The symptoms started 1 year ago when he initially developed central scotomas, followed by loss of colour vision and ultimately loss of all vision. He has no significant past medical history.

On examination, visual acuity is 3/60 in both eyes. Fundoscopy reveals optic atrophy in both eyes, with narrow vessels and absent macular reflexes. An MRI scan of the brain shows significantly lower optic nerve volumes than normal. His mother also lost her vision at 34 years of age. His brother and sister, who are both a few years older, developed the same symptoms several years ago.

What is the most likely diagnosis?

A. Autosomal dominant optic atrophy (ADOA)
B. DIDMOAD syndrome
C. Leber's optic atrophy
D. MELAS syndrome
E. Optic neuritis

C. Leber's optic atrophy

30-years-old, central scotoma → loss of colour vision → rapid onset of significant visual impairment ? Leber's optic atrophy

The history of central scotoma, colour blindness, and rapid onset of visual impairment point in an adult male point toward a diagnosis of Leber's optic atrophy in this scenario. A history of a similar illness in the mother and all siblings confirms the mitochondrial pattern of inheritance in which all children of an affected mother inherit the disease. Diagnosis of mitochondrial disease is confirmed by muscle biopsy showing ragged red fibres due to an increased number of mitochondria.

Although optic atrophy is a prominent feature of autosomal dominant optic atrophy (ADOA) , vision loss in ADOA occurs in late childhood or early adolescence. The onset of vision loss in ADOA is gradual and not rapid as in this scenario. The pattern of inheritance described is mitochondrial and not autosomal dominant, therefore this option is incorrect.

MELAS syndrome is characterised by mitochondrial encephalomyopathy lactic acidosis and stroke-like episodes. There is no history of seizures, weakness, metabolic disturbances, or altered consciousness and so MELAS is an unlikely diagnosis here.

The family history in this scenario demonstrates mitochondrial inheritance and effectively rules out acquired/autoimmune causes of vision loss such as optic neuritis. Optic neuritis is usually associated with multiple sclerosis, diabetes mellitus, or recent infection.

Optic atrophy and vision loss in DIDMOAD syndrome occur in childhood or adolescence. Optic atrophy in DIDMOAD syndrome is accompanied by diabetes insipidus, diabetes mellitus, and sensorineural deafness. The unremarkable past medical history of this patient rules out this diagnosis.

Question 41 of 299

A 62-year-old man develops difficulty in breathing seven hours following thyroidectomy for hyperthyroidism. He has been finding it more difficult to breathe over the last one hour and feels like his neck is tighter than it was. He has a past medical history of COPD, ischaemic heart disease and angina. He normally takes aspirin, ramipril, Symbicort, bisoprolol and atorvastatin, but has not been taking aspirin for the last week in preparation for surgery. On examination his chest is clear and heart sounds are normal. The neck wound is dressed and there is a small amount of strikethrough bleeding. His heart rate is 90 bpm, his blood pressure is 156/82mmHg and his oxygen saturations are 89% on room air. His calves are soft and non-tender.

What is the most appropriate plan?

A. Page the surgical and anaesthetic registrar urgently
B. Start salbutamol and ipratropium nebulisers
C. Give a stat dose of aspirin, clopidogrel and fondaparinux
D. Give a stat treatment dose of heparin
E. Request and urgent CT neck

A. Page the surgical and anaesthetic registrar urgently

Difficulty in breathing due to immediate heamatoma is an emergency and needs immediate action

This patient has likely a haematoma following thyroid surgery. This can cause laryngeal oedema which can be fatal. It is critical to secure the airway early, as allowing oedema to progress can make intubation more difficulty, and subsequently for the surgical team to relieve the haematoma. As the chest is clear this is unlikely to be an exacerbation of COPD, and the absence of chest pain makes acute coronary syndrome or pulmonary embolism unlikely.

Question 42 of 299

A 33-year-old female is referred to the neurology outpatient department by her general practitioner. She complains of five months of excruciating right-sided throbbing headaches, localized to the temporal region. The headaches start and stop abruptly and last for approximately 15 minutes at a time. They occur 9-12 times a day and are associated with nasal congestion and rhinorrhoea. There is no significant past medical history and she is not on any regular medications. She has a family history of migraine and smokes ten cigarettes per day. She does not drink alcohol.

Neurological examination at the time of clinic review is unremarkable.

Hb	140 g/L	Male: (135-180) Female: (115 - 160)
Platelets	176 * 10 /L	(150 - 400)
WBC	8 * 10 /L	(4.0 - 11.0)
Na	139 mmol/L	(135 - 145)
K	4 mmol/L	(3.5 - 5.0)
Urea	5 mmol/L	(2.0 - 7.0)
Creatinine	64 µmol/L	(55 - 120)
CRP	3 mg/L	(< 5)

An MRI brain is organized, which is reported as normal.

Given the likely diagnosis, which medication treatment will result in resolution of her symptoms?

A. Carbamazepine
B. High flow oxygen
C. Indomethacin
D. Prednisolone
E. Sumatriptan

C. Indomethacin

Paroxysmal hemicrania is completely responsive to treatment with indomethacin

Indomethacin is the correct answer. Paroxysmal hemicrania is characterized by relatively short-lasting, frequent unilateral headaches associated with ipsilateral autonomic features (such as rhinorrhoea and nasal congestion in this case). It shares many features with cluster headaches but can be differentiated based on its dramatic response to indomethacin, higher prevalence in females, shorter attack duration and higher attack frequency. Brain imaging is useful to exclude a secondary cause of presentation.

Carbamazepine is not the right answer. This is used as a treatment for trigeminal neuralgia. This headache is not characterized by ipsilateral autonomic features and the pain affects the lower part of the face in the distribution of the trigeminal nerve.

High flow oxygen is not the right answer. This treatment is used to abort attacks of cluster headache. Cluster headaches share many of the same features as paroxysmal hemicrania but the attacks tend to be of longer duration (45-90 minutes) and occur less frequently (1-8 times per day on average). Patients often describe circadian rhythmicity to their symptoms, with the headaches occurring at similar times each day.

Prednisolone is not the right answer. High dose prednisolone can be used to treat the temporal headache of giant cell arteritis. However, the young age of this patient essentially excludes this diagnosis.

Sumatriptan is the wrong answer. This is used as a treatment for both cluster headaches and migraines, neither of which is the correct answer here. The duration of migraine is typically longer and more classically associated with visual aura, paraesthesia, photophobia and phonophobia.

Question 43 of 299

A 55-year-old man is referred to neurology clinic by his GP with reports of a six month history of tremor affecting both hands. It worsens with emotional stress. He has no past medical history and does not take any medications. He does not smoke or drink alcohol. He works as a cooper.

On examination, there is a tremor of both hands with an frequency of 5 Hz and a medium amplitude. It is slightly more prominent on the right hand than the left but this is subtle. It is present both posturally and at rest. There are no other discriminating features.

Given the clinical presentation, which test is most appropriate to guide diagnosis?

A. Acetylcholine receptor antibodies
B. Lumbar puncture
C. MRI head
D. Nerve conduction studies
E. 123I-FP-CIT SPECT

E. 123I-FP-CIT SPECT

123I-FP-CIT SPECT can be used to help differentiate essential tremor from Parkinson's

123I-FP-CIT SPECT is the correct answer. In this scenario, there is uncertainty as to whether this tremor represents an essential tremor or a Parkinsonian tremor. The so called 'DaTSCAN' can be used to differentiate between essential tremor and a Parkinsonian tremor if there is diagnostic uncertainty. A DaTSCAN measures the density of nigrostriatial dopamine transporter sites. A radioactive tracer is injected into the blood and binds to the dopamine transporter in the brain. The imaging technique assess for the level of binding observed. In Parkinson's disease, there is a decreased level of binding between the tracer and the dopamine transporter in the brain. In essential tremor, the binding level will be normal.

Lumbar puncture is incorrect. Typically, this investigation is not necessary in differentiating between essential tremor and PD. However, if there was a suspicion of an intra-cerebral infectioncausing tremor, it may be useful.

MRI head is incorrect. This may show vascular changes if vascular parkinsonism is suspected.However, this type of Parkinsonism tends to be lower body predominant and tremor is a less common feature.

Nerve conduction studies is incorrect. This is a test that can help diagnose neuropathies but is little value in determining the cause of tremors.

Acetylcholine receptor antibodies are incorrect. This is an antibody test used to support a diagnosis of myasthenia gravis. This disorder is characterized by fatigable weakness rather than tremor.

Question 44 of 299

A 23-year-old presents to the emergency department. He has arrived with a headache however nursing staff are concerned by the appearance on the ECG taken at triage. He is usually well. He is clear that he does not have any chest pain or shortness of breath. He does report that his head is really hurting and this started suddenly in the morning when he was preparing his breakfast.

On examination, he is a young slim gentleman with normal heart sounds. His lung fields are clear and there is no visible JVP. He is difficult to examine due to agitation and he refuses to open his eyes to the light. His blood pressure is 140/80 mmHg.

ECG ST elevation in leads V4-V6

What is the likeliest cause of the ECG changes?

A. Benign early re-polarisation
B. Left ventricular hypertrophy
C. Subarachnoid haemorrhage
D. Silent myocardial infarction
E. Acute pericarditis

C. Subarachnoid haemorrhage

ST elevation can occur in subarachnoid haemorrhage

This gentleman has ST elevation but no cardiac signs. This could be benign early re-polarisation also called 'high take off' but the severe headache suggests this could be a subarachnoid haemorrhage. His slim physique may suggest a Marfan's syndrome which is a recognised cause of cerebral aneurysm. A silent myocardial infarction is rare particularly in young males and there are no other

symptoms seen like shortness of breath. Acute pericarditis has ST elevation in all leads and sharp chest pains. Left ventricular hypertrophy presents with large volume complexes but not ST elevation.

Question 45 of 299

A 25-year-old gentleman presents with a headache to the emergency department. He describes walking back from the gym and developing an occipital headache that came on suddenly and became a 9/10 severity within seconds. He started to vomit and a bystander called an ambulance.

He is normally fit and well. He takes no medication regularly and denies any recreational substance use. He has been referred to the genetics clinic due to his father having been diagnosed with a phaeochromocytoma and his maternal grandmother had kidney tumours.

On arrival to the emergency department, his glasgow coma scale has dropped from 14 to 6. He has reactive pupils and an intact corneal reflex. His neck is rigid and he screams out when you try to flex his neck. His blood pressure is 180/90mmHg, heart rate 60/min and respiratory rate is 11/min. He has two cafe-au-lait spots on his trunk but there are no palpable masses and no changes seen in the irises. He is 6 foot 5 inches and weighs 95 kilograms. Abdominal examination is unremarkable

Urine dip	blood trace, leucocytes positive
ECG	ST elevation in V1-V4
CT head	large high attenuation signal seen in the left cerebellar hemisphere with surrounding oedema, no mass effect
Lumbar puncture	xanthochromia colour, raised red cells

What is the likely unifying diagnosis?

A. Tuberous sclerosis
B. Neurofibromatosis type 1
C. Von Hippel Lindau
D. Multiple endocrine neoplasia
E. Polycystic kidney disease

C. Von Hippel Lindau

Early age SAH occur in Von Hippel Lindau

This gentleman presents with a subarachnoid haemorrhage at a young age which is likely aneurysmal. He has a family history notable for renal and adrenal malignancy and there is a suspected genetic condition present in the family. Of the above, PKD and VHL are associated with subarachnoid haemorrhage and so these should stand out as possible causes. Polycystic kidney disease usually has palpable bilateral enlarged kidneys or gross haematuria. Therefore, the answer is Von Hippel Lindau syndrome where c3p25 mutation causes 80% to develop cerebellar haemangiomas or haemangioblastomas which can bleed acutely.

Question 46 of 299

An 87-year-old lady presents to the emergency department with unilateral weakness and according to her family difficulty in naming objects. This started about six hours ago.

Examination was consistent with weakness in left arm and leg (arm > leg) with decreased sensation. You also notice that she becomes increasingly tearful and has difficulty in naming objects around her, although she is able to describe their function. A systems review indicates a Heart Rate of 72/min and a blood pressure of 110/84 mmHg. There are no precordial murmurs and no carotid bruits. An ECG is performed showing that the patient is in sinus rhythm.

An urgent CT reveals an area of grey matter differentiation in the right hemisphere. Therefore a diagnosis of Right Partial Anterior Circulation Syndrome (R PACS) is made.

She has previously been on amlodipine 5 mg and mirtazapine 30 mg once per day. She is started on aspirin.

Which other intervention would you consider regarding her treatment.

A. Atorvastatin 80 mg OD to be started 48 hours post onset of symptoms
B. Amlodipine to be raised to 10 mg
C. Anti-embolism stockings to be prescribed
D. Bisoprolol 1.25 mg OD to be initiated
E. Atorvastatin 40 mg OD to be started now

A. Atorvastatin 80 mg OD to be started 48 hours post onset of symptoms

Statins should be initiated for secondary prevention of stroke 48 hours following ischaemic event

This lady has suffered from a cerebrovascular ischaemic episode. As per NICE guidelines, and given the lack of co-morbidities secondary prevention should be started appropriately. In the case of secondary prevention in ischaemic CVA, NICE guidelines suggest the prescription of atorvastatin, 48 hours post-event. The reason behind this is that previous studies have identified the potential for haemorrhagic transformation in early administration of statins. Anti-embolism stockings are not advised to be used in stroke patients and it has been proven that they add no further benefit in the prevention of embolic events in such patients in the acute setting.

Question 47 of 299

A 45-year-old man with a history of hypertension and ischemic heart disease presents to the emergency department following a collapse. He reports that he developed weakness down his left side before suddenly collapsing to the floor. His partner reports that he lost consciousness for around two minutes before recovering. She also noted that the right side of his face was dropping. The patient's symptoms completely resolved after 60 minutes.

Current medication is amlodipine 5mg, bisoprolol 2.5mg, aspirin 75mg, simvastatin 20mg.

Which feature in the history suggests that an alternative diagnosis is more likely than a TIA?

A. Patient's age

B. Timing of symptoms
C. Crossed pattern of weakness
D. Loss of consciousness
E. Current use of anti platelets

D. Loss of consciousness

TIAs do not cause loss of consciousness

Transient ischaemic attacks (TIAs) rarely cause loss of consciousness. Whilst the patient is young for a TIA it does not completely exclude this as a possibility, especially given his risk factors. Whilst anti-platelets are a preventative measure they do not cause a complete risk reduction. Crossed patterns of weakness can occur with a posterior circulation TIA. Finally, the timing of the symptoms is actually supportive of a TIA with sudden onset and quick, complete recovery.

Question 48 of 299

A 32-year-old woman is seen in the neurology clinic with a 6-month history of intermittent headaches. The headaches typically affect her left side and are associated with paraesthesia in her left hand and occasional nausea and vomiting. She says that her symptoms resolve after a period of 4-6 hours each time. She has no past medical history of note and has no regular medications or allergies.

On examination, her observations are recorded within normal limits. There is equal power in all 4 limbs with no sensory or coordination disturbance. Her cranial nerve examination is unremarkable.

What is the most appropriate next step in the long-term management of this patient?

A. Amitriptyline
B. Aspirin
C. Propranolol
D. Sumatriptan
E. Topiramate

C. Propranolol

Propranolol is preferable to topiramate in women of childbearing age (i.e. the majority of women with migraine)

This patient's headache is likely to represent a migraine, given the unilateral presentation associated with photophobia and vomiting and normal neurological examination. To prevent the recurrence of migraines, prophylaxis can be offered to patients. Propranolol is preferred as pharmacological prophylaxis in women of childbearing age, as topiramate is teratogenic and associated with birth defects such as a cleft palate.

Amitriptyline is a third-line agent used in the prophylaxis of migraines. Typically, alternative drugs such as propranolol should be used initially, unless contraindicated. Aspirin is used in the acute treatment of migraines. However, it plays no role in the prophylaxis of migraine.

Sumatriptan is another medication used in the acute treatment of migraines. However, long-term sumatriptan is not used to prevent the recurrence of migraines.

Topiramate is used in the prophylaxis of migraines. However, as this patient is a woman of childbearing age, it is inappropriate to initiate topiramate for long-term use as it is teratogenic and associated with birth defects.

Question 49 of 299

A 60-year-old woman was referred with dizziness, nausea, and vomiting. This had come on insidiously the previous day. She described the room spinning, which was there constantly but worse with any sudden movement. She had suffered flu-like symptoms in the previous week, which were now resolving. On examination, she had horizontal nystagmus with the fast beat towards the right. Speech was normal. There was no past-pointing and finger-nose testing was normal. Gait was normal. Power was 5/5 throughout all limbs. Her hearing was normal.

What is the most likely diagnosis?

A. Benign paroxysmal positional vertigo
B. Meniere's disease
C. Vestibular neuronitis
D. Transient ischaemic attack
E. Vertebrobasilar insufficiency

C. Vestibular neuronitis

Prodromal viral illness with associated vertigo and nystagmus is suggestive of vestibular neuronitis

The presence of horizontal nystagmus in the absence of any other neurological signs suggests a vestibular origin of the acute symptoms in this case. A posterior circulation vascular event would be likely to be associated with other signs of cerebellar dysfunction or other cranial nerve palsies. The preceding probable viral illness and the persistence of the symptoms suggest a viral vestibular neuronitis as the cause of her vertigo rather than benign paroxysmal positional vertigo. There are also none of the additional features that would suggest Meniere's disease such as unilateral deafness, severe nausea, and a pressure sensation in the ear.

Question 50 of 299

A 53-year-old man presents to the emergency department with a 1-day history of bilateral leg weakness. He is now struggling to stand up from a chair. Whilst waiting to be seen in the department, the patient starts to experience finger paraesthesia.

The patient reports no medical history of note and is in good health aside from a recent bout of diarrhoea. He takes no regular medications, smokes 20 cigarettes per day and drinks up to 4 pints of beer 'most nights'. He works as a plumber.

On clinical examination, there is a marked weakness of the lower limbs, absent reflexes at the ankle, knee, and elbow with a grossly normal sensory assessment. He is dysarthric and coughs significantly on a bedside swallow assessment.

An MRI whole spine is ordered and reported as normal. Blood tests show a raised ALT, but no other abnormalities.

Given the likely diagnosis, assuming he receives standard treatment, what is the probability of long term weakness

A. 30%
B. 40%
C. 50%
D. 1%
E. 15%

E. 15%

Up to 15 percent of individuals with Guillain-Barre syndrome will experience long-term weakness

This patient presents with classical Guillain-Barre syndrome(GBS). Early recognition and treatment with intravenous immunoglobulin and/or plasma exchange both increase the speed of recovery and are important in reducing the incidence of long term sequelae.

The most common long term sequelae of GBS is fatigue, although, depending on the clinical course, pain and persistent neurological deficits may also be problematic, with long term neurological difficulties reported in 7-15% of patients.

Question 51 of 299

A 27-year-old caucasian woman is 28 weeks pregnant. She has been epileptic since the age of 7 and takes lamotrigine, which she has continued throughout the pregnancy. She has not had a seizure for 2 years.

What must you consider in pregnant patients taking lamotrigine?

A. Serum lamotrigine levels fall in the second trimester
B. She must not breastfeed while taking lamotrigine
C. She should be advised to stop lamotrigine
D. Folic acid 400mcg should be taken throughout pregnancy
E. It is protein bound and therefore drug levels increase in pregnancy

A. Serum lamotrigine levels fall in the second trimester

Due to the increased plasma volume and enhanced renal/hepatic drug clearance, lamotrigine levels decrease in the second trimester of pregnancy. It may be necessary to increase the dose of lamotrigine at this time (normally increased two or three fold). A baseline serum drug level is useful to establish compliance and inform future changes in drug doses.

Breastfeeding is not contraindicated in women taking lamotrigine. However, the infant should be monitored for signs of accumulation such as a rash or drowsiness.

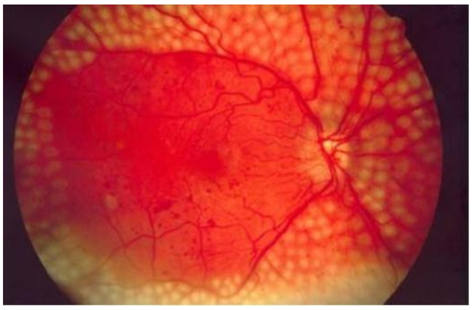
Patients should not stop their anti-epileptic medications unless a joint decision has been made in conjunction with an obstetrician/obstetric physician doctor. If a patient has been seizure-free for >2 years and is on dual therapy then it may be beneficial to reduce/stop one of the medications.

Patients on anti-epileptic medications should take folic acid 5mg throughout pregnancy.

Lamotrigine has low levels of protein binding and therefore is not affected by the decreased serum protein levels in pregnancy.

Question 52 of 299

A 52-year-old man presents for a routine eye screening appointment. He has a past medical history of hypertension, type 2 diabetes mellitus and hyperlipidaemia. The eyes are examined using an ophthalmoscope and the following view is seen of the left eye:



What is the most common complication of the treatment that this patient has received?

A. Chronic eye pain
B. Development of floaters
C. Development of infection
D. Reduced central vision
E. Reduced night vision

E. Reduced night vision

The photo shows clear evidence of diabetic retinopathy - proliferation of small vessels is present, as well as haemorrhage. Importantly, this photo also shows evidence of panretinal laser photocoagulation (PRP) - the numerous circular yellow spots around the periphery are 'burn scars' from the treatment. PRP aims to prevent further proliferation of vessels. Of the complications listed here, the most common adverse effect associated with PRP is a reduction in night vision. This is because PRP leads to scarring of peripheral retinal tissue, where rods are situated (rods being responsible for vision in low light). As such, the peripheral visual field is also often reduced.

Chronic eye pain is incorrect. Some temporary pain may arise following the procedure, but this improves within 24-48 hours.Development of floaters is incorrect. Floaters develop due to the underlying condition, and due to macular oedema that may result from diabetic eye disease. PRP itself is not generally associated with causing further floaters.

Development of infection is incorrect. PRP is not associated with the development of infection.

Reduced central vision is incorrect. As PRP targets the periphery, the central vision is typically spared.

Question 53 of 299

A 68-year-old man develops a left-sided hemiparesis and facial droop whilst out shopping with his wife at 14:35pm. His initial CT head scan is done at 15:40pm and reveals no intracranial haemorrhage or space occupying lesion. He is given alteplase in the emergency department at 15:50pm.

His post-thrombolysis CT head at 09:00 am the following morning reveals a right middle cerebral artery infarction and no evidence of intracranial haemorrhage or haemorrhagic transformation. A 12 lead ECG done in the stroke unit shows atrial fibrillation of 110 beats per minute. He has a persisting dense left-sided weakness several hours post thrombolysis.

Which of the following drugs should be added next?

A. Clopidogrel
B. Aspirin
C. Warfarin
D. Unfractionated heparin
E. Rivaroxaban

B. Aspirin

Aspirin 300mg orally for the initial two weeks should be commenced in patients with a disabling stroke and atrial fibrillation. Following this, these patients should be considered for anticoagulant treatment. The post-thrombolysis CT head revealed no acute intracranial haemorrhage or haemorrhagic transformation, therefore, it is safe to start aspirin.

Question 54 of 299

A 67 year-old woman presents to hospital after a collapse at home. One week earlier she had seen her own doctor after feeling generally unwell with fever, myalgia, and coryza. These symptoms were attributed to a viral infection and have now resolved. However, for the past few days she has felt unsteady on her feet and has had to hold on to the furniture whilst walking around the house. For the last two days in particular she has also noticed that her vision has deteriorated, and she has been seeing double. Today she tried to hold onto the sofa whilst walking around the room, but misjudged the distance and lost her grip, causing her to fall to the floor. Her daughter who is also present adds that she feels her mothers speech is slurred compared to normal, and has been so for the past three days.

Her past medical history includes hypertension, hypothyroidism, type 2 diabetes, hyperlipidaemia, angina, and recurrent urinary tract infection. She takes ramipril, amlodipine, doxazosin, laevoythyroxine, metformin, gliclazide, simvastatin, atenolol, nicorandil, and nitrofurantoin. She does not smoke but admits to enjoying a glass of sherry on most nights. Usually she is independently mobile, without aids. She lives on her own and is self-caring.

On examination, there are some bruises on the left shoulder where she fell, but no suggestion of any fracture. Observations are normal and she is afebrile. Cardiovascular, respiratory, and abdominal examination is unremarkable. She is fully alert and oriented. Pupils are equal and reactive to light. Eye movements are grossly impaired in all directions. There is no facial asymmetry. Muscle power is normal in all limbs, but finger-nose pointing is impaired and she is unable to walk in a straight line. Reflexes are unobtainable. Sensation is normal.

What is the most likely diagnosis?

A. Wernickes encephalopathy
B. Viral cerebellitis
C. Myasthenia gravis
D. Miller Fisher syndrome
E. Posterior circulation stroke

D. Miller Fisher syndrome

There has been a subacute deterioration in global cerebellar function, as suggested by gait ataxia, bilateral upper limb dysmetria, and dysarthria, progressing over a period of days. There is also complete ophthalmoplegia, and absence of reflexes. This combination of features occurring after a recent viral infection suggests a diagnosis of the Miller Fisher variant of Guillain-Barre syndrome. One might also expect to find descending weakness, which was not present in this case. Confirmation of the diagnosis is by lumbar puncture which demonstrates raised protein with normal cell count, and positive serum anti-GQ1b antibodies. Treatment is as for Guillain-Barre syndrome, with supportive care and intravenous immunoglobulin or plasmapheresis.

Wernickes encephalopathy presents with ataxia and ophthalmoplegia but also confusion, and typically occurs in malnourished alcoholics.

Viral cerebellitis is more common in children and would not explain the ophthalmoplegia and areflexia.

Myasthenia gravis would be unlikely to develop so rapidly, and though a cause of complex ophthalmoplegia, it would not explain the areflexia or the cerebellar signs.

Posterior circulation syndromes can cause acute cerebellar dysfunction but stroke of any sort is unlikely as this is a subacute presentation of neurological features not referable to one part of the brain.

Question 55 of 299

A 44 year-old man presents to the neurology clinic. He complains of weakness in the right hand, which causes difficulty with tasks such as writing and dressing, and has also noted 'twitching' in his right forearm and hand. He cannot recall exactly when the symptoms first began but feels that they have certainly been present for at least six months, and that they have got worse.

He is otherwise fit and well. His only past medical history is well-controlled asthma. There is no family history of neurological disease.

Cranial nerve examination is normal. On examination of the right hand, there is weakness of extension of the the middle and ring fingers (2/5 on the MRC scale), but no discernible muscle wasting. Fasciculations are visible in the dorsal aspect of the forearm. On examination of the left hand, there is also some subtle weakness of extension of the ring finger (4/5 on the MRC scale). There is no weakness of wrist extension on either side and power in all other muscle groups is normal. For both hands, all the fingers extend when the wrist is passively moved into palmar flexion. All reflexes are normal and there are no sensory signs.

What is the most likely diagnosis?

A. Multifocal motor neuropathy
B. Chronic inflammatory demyelinating polyneuropathy
C. Motor neuron disease (amyotrophic lateral sclerosis)
D. Inclusion body myositis
E. Ruptured tendon of extensor digitorum

A. Multifocal motor neuropathy

The history is typical for multifocal motor neuropathy (MMN). The condition presents with asymmetric muscle weakness, which is slowly progressive, and without sensory signs. Differential weakness of finger extension is a typical presentation, and reflects a pathological process which selectively affects particular motor fibres within a peripheral nerve (in this case the posterior interosseous branch of the radial nerve). Weakness without wasting is another typical feature. Nerve conduction studies show areas of conduction block outside usual areas for compression. Intravenous immunoglobulin can produce a rapid improvement in weakness.

MMN may sometimes mimic motor neuron disease (MND). Both feature weakness, wasting, and fasciculation, with relatively preserved reflexes, and normal sensation. However, certain features suggest one diagnosis over the other. For example, bulbar weakness is common in MND but rare in MMN. Depending on the form of MND, variable upper motor neuron signs may occur, contrary to MMN which is a purely lower motor neuron condition occurring in a distribution correlating with named peripheral nerves. Finally, whereas MND is usually fatal within 5 years, MMN is slowly progressive over decades and does not typically cause respiratory failure.

Chronic inflammatory demyelinating polyneuropathy(CIDP) typically causes a progressive sensory and motor neuropathy with loss of reflexes. CSF protein is elevation and nerve conduction studies show evidence of demyelination.

Inclusion body myositis characteristically causes weakness of finger flexion, as well as knee extension and ankle dorsiflexion. Affected muscles are wasted, and fasciculations do not occur as there is no denervation.

Rupture of extensor tendons often occurs in rheumatoid arthritis. The affected finger cannot be actively extended, and does not passively extend when the wrist is passively moved into palmar flexion.

Question 56 of 299

A 50-year-old man was transferred to a tertiary referral centre with dense right-sided weakness and a Glasgow Coma Score of 8. It was estimated that the onset of this event was 8 hours prior to being found. He had bilaterally up-going plantar responses. He was haemeodynamically stable and not known to have any major co-morbidities.

A CT Scan of his brain revealed a moderate area of established infarct within left middle cerebral artery (MCA) territory with massive cerebral oedema and mid-line shift.

In this circumstance, which of the following interventions may benefit the patient in the acute period?

A. Decompressive hemicraniectomy
B. Intra-arterial clot retrieval
C. Intravenous thrombolysis
D. Intravenous dexamethasone
E. Aspirin through nasogastric tube

A. Decompressive hemicraniectomy

For individuals aged up to 60 years who suffer an acute MCA territory ischaemic stroke complicated by massive cerebral oedema, surgical decompression by hemicraniectomy should be offered within 48 hours of stroke onset.

Question 57 of 299

An 86-year-old lady was admitted to the acute medical admissions unit having been referred by her GP with acute confusion. Three days ago she was treated by her GP with trimethoprim 200mg BD for a presumed urinary tract infection, her nursing home having complained of new urinary incontinence and offensive smelling urine. Unfortunately, she continued to deteriorate and on the day of admission had become rather confused and unable to mobilise, leading to the admission. Her past medical history comprised Alzheimer's disease, hypertension, hypercholesterolaemia and osteoarthritis for which she was prescribed donepezil 10mg OD, amlodipine 5mg OD, atorvastatin 20mg ON and paracetamol 1g QDS. She was usually able to mobilise independently and suffered from intermittent confusion, though she was able to hold lucid conversations with her care workers. She had a daughter who resided abroad who had not made contact with her in several years and did not have a lasting power of attorney or advanced directive in place.

Examination revealed a very unwell elderly lady. Her temperature was 39.6ºC, heart rate 122 bpm, respiratory rate 26/min and blood pressure of 97/58 mmHg. Examination of her cardiovascular system revealed the presence of a bounding peripheral pulse and a capillary refill time of three seconds. Examination of her respiratory system revealed tachypnoea. Examination of her abdomen revealed no abnormalities. She was not compliant with formal neurological examination; her GCS was 14 (E4 S4 M6). She was very confused and unable to maintain a formal conversation; her speech did not resemble any form.

When the doctor proceeded to perform venous cannulation, she became very aggressive and hostile. She refused all forms of treatment, stating that she did not require medical treatment and requested to leave the ward immediately. She did not appear to comprehend the risks of refusing medical admission and was unable to recall the information disseminated.

What is the next best management option?

A. Seek opinion from family members
B. Apply for formal Deprivation of Liberty Order and proceed with treatment once obtained
C. Respect the wishes of the patient and arrange for urgent GP follow up
D. Section under 5(2) of the Mental Health Act (MHA) and proceed with treatment

E. Proceed with treatment under the Mental Capacity Act (MCA)

A. Proceed with treatment under the Mental Capacity Act (MCA)

This lady has evidence of a life-threatening acute organic illness requiring immediate treatment, but is refusing treatment and lacks the capacity at present to make a decision regarding her medical care, and does not have an advanced directive of lasting power of attorney. Responsibility for decisions regarding medical care, therefore, is bestowed to the medical team, who are entitled to treat the patient under the Medical Capacity Act. Seeking a family opinion here is good practice but impractical here given the paucity of family contact and the emergency situation, and the Mental Health Act is not applicable here as the primary illness is most likely organic rather than relating to a mental health disorder. It is worth mentioning that under the MCA it is possible to obtain an emergency DOLS; a formal DOLS application must be made at the same time but in an emergency situation awaiting a formal DOLS should not prevent treatment.

Question 58 of 299

A 70-year-old male is evaluated at the neurology outpatient department for an episode of left sided body weakness which lasted for around 30 minutes and resolved completely. He described the episode as an inability to move the left side of his body and an associated numbness and tingling sensation in the area involved. He remained conscious throughout and was able to communicate verbally with his family members during the episode.

He suffers from hypertension and has hypercholesterolaemia. He is also a smoker with a 40 pack year history and is known to be a heavy drinker.

He has also noted a tremor in both his hands which tends to improve after he has a drink. He also feels unsteady while walking and feels the need to grip something otherwise he may lose balance. His medication includes amlodipine 5mg daily and atorvastatin 20mg daily.

On examination, his blood pressure is 150/95mmHg and his pulse is 86bpm regular. A carotid bruit is audible over both sides of the neck. Neurological examination reveals impaired sensations in a glove and stocking distribution. The remaining clinical examination is normal.

Investigations reveal:

ECG: deep S waves in lead V1-V3 and tall R waves in V4-V6

CXR: Enlarged cardiac silhouette with flecks of calcification around the aorta

Carotid artery Doppler studies reveal 85% occlusion in the right external carotid. 50% occlusion in the right internal carotid. Left internal carotid is 80% occluded while there is a 60% occlusion in the left external carotid artery.

Which of the following is the most suitable treatment option in this patient?

A. Left internal carotid endarterectomy
B. Optimizing medical management
C. Right external carotid endarterectomy
D. Percutaneous stenting of the right internal carotid artery
E. Percutaneous stenting of the left internal carotid artery

B. Optimizing medical management

In the aforementioned scenario, the patient has suffered from a right sided TIA and has recovered completely. The question aims to assess the candidates knowledge pertaining to intervention in patients with TIAs and carotid stenosis.

Carotid endarterectomy or endovascular stenting is recommended in patients with symptomatic stenosis of the affected vessel >70%. In the patient described above, the symptoms involve the left side of the body, hence the right carotid is involved. The degree of stenosis in the right internal carotid is 50 % and therefore the treatment of choice would be optimization of medical therapy with improved blood pressure control and antiplatelet agents rather than intervention.

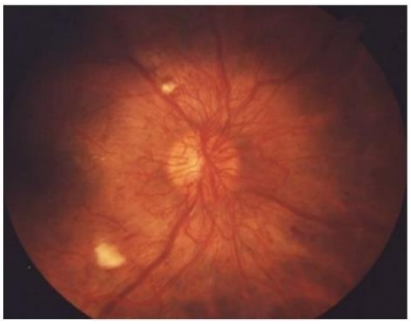
The carotid stenosis in the left internal carotid is asymptomatic therefore not an indication for endarterectomy.

The external carotids do not form part of the circle of Willis and do not contribute to the blood supply of the brain.

The reference to his benign essential tremor is of no relevance to his clinical condition

Question 59 of 299

A 50-year-old woman presents to the optician with a progressive decline in visual acuity. The optician refers her for medical review. The fundoscopic examination shows:



Given the fundoscopic findings, what is the most appropriate treatment?

A. Intra-vitreal VEGF inhibitor therapy
B. Optimise glycaemic control
C. Pan-retinal laser photocoagulation
D. Urgent trabeculectomy
E. Vitreoretinal surgery

C. Pan-retinal laser photocoagulation

The fundoscopy shows extensive neovascularisation in all four quadrants obscuring the optic disc, which is suggestive of advanced proliferative diabetic retinopathy commonly seen in type I diabetes. The soft exudates, seen as 'cotton wool spots' on the left upper and lower quadrant, represent areas of retinal infarction, suggesting advanced diabetic retinopathy.

Pan-retinal laser photocoagulation is the most appropriate treatment for this patient with extensive neovascularisation to improve retinal oxygenation and reduce the risk of retinal or vitreous haemorrhage. Pan-retinal photocoagulation aims to prevent haemorrhage and furtherneovascularisation, not to regain lost vision.

Optimise glycaemic control should be considered in all patients with non-proliferative or proliferative diabetic retinopathy. However, this patient, with extensive neovascularisation and impending haemorrhage, requires urgent photocoagulation.

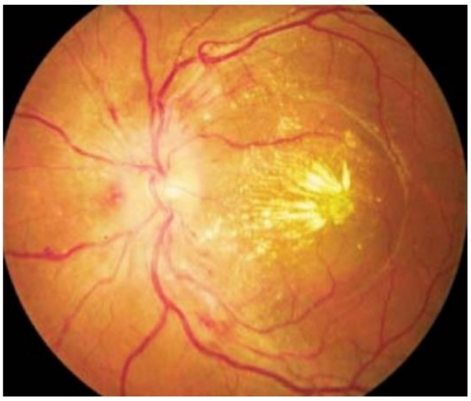
Urgent trabeculectomy is the surgical intervention used to reduce intraocular pressure in acute angle-closure glaucoma. Glaucoma leads to the classical fundoscopic appearance of cupping or increased vertical cup-to-disc ratio, which is not seen in this patient.

Intra-vitreal VEGF inhibitor therapy is recommended for patients with maculopathy or may be considered in combination with pan-retinal laser photocoagulation to treat severe proliferative diabetic retinopathy. While VEGF as an adjunctive therapy can help in the regression of neovascular vessels, definitive treatment with photocoagulation is essential to prevent impending retinal or vitreal haemorrhage.

Vitreoretinal surgery is recommended for severe or vitreous haemorrhage, which is often a complication of extensive neovascularisation. However, the fundoscopic findings in this patient do not suggest severe or vitreous haemorrhage, which may be seen as a red haze of haemorrhage into the vitreous associated with dot-blot haemorrhages, neovascularisation elsewhere or boatshaped pre-retinal haemorrhages.

Question 60 of 299

A 75-year-old man visited the optician for a progressive decline in vision in both eyes and a headache. The optician was concerned about his fundoscopic appearance and referred him to the emergency department for further workup. He has hypertension and diabetes and reports good compliance with his medications. He currently takes perindopril, amlodipine and hydrochlorothiazide for hypertension; metformin, linagliptin and dapagliflozin for diabetes management



What is the next best step in his management?

A. Add basal-bolus insulin therapy
B. Escalate antihypertensive treatment
C. Monitor visual acuity weekly
D. Refer for urgent trabeculectomy
E. Start broad-spectrum antibiotics

B. Escalate antihypertensive treatment

The flame and blot haemorrhages collected around the fovea result in a characteristic macular star pattern. The associated arteriolar narrowing and AV nipping are suggestive of hypertensive retinopathy. The more ominous sign of papilloedema indicates stage IV hypertensive retinopathy.

Escalate antihypertensive treatment is the next best step in his management. Stage IV hypertensive retinopathy is indicative of uncontrolled malignant hypertension. Prompt review and escalation of hypertensive treatment are vital in reducing ophthalmologic and systemic complications.

Add basal-bolus insulin may be considered if the patient has poorly controlled diabetes despite optimised oral therapy. Although the fundoscopic features suggest hypertensive rather than diabetic retinopathy, persistent hypertension can accelerate the progression of diabetic eye disease.

Monitor visual acuity weekly is inappropriate as this patient with stage IV hypertensive retinopathy requires prompt treatment to control blood pressure to reduce ophthalmologic and systemic complications.

Refer for urgent trabeculectomy , which is the most common surgical intervention for glaucoma, is not needed in this patient. Glaucoma leads to the classical fundoscopic appearance of cupping or increased vertical cup-to-disc ratio, which is not seen in this patient.

Start broad-spectrum antibiotics like azithromycin or ciprofloxacin would be the appropriate treatment for the cat-scratch disease, which is the most common cause of infectious neuroretinitis. Although macular star pattern and papilloedema may also be seen in neuroretinitis, the onset is typically acute and often limited to one eye.

Question 61 of 299

A 21-year-old man is referred to the ophthalmology clinic. He has a past medical history of type 1 diabetes mellitus which has been poorly controlled as he has recently started university and is living away from home for the first time. This has caused him to monitor his blood sugars less frequently as he is embarrassed about his medical problem and is keen to fit in. He has no visual symptoms but fundoscopy demonstrates microaneurysms and cotton wool spots. He has no other known medical problems and uses biphasic insulin. His observations are all within normal range visual acuity is normal.

Apart from advising and support in managing his diabetes, how should he be managed?

A. Referral for retinal laser treatment
B. Annual digital retinal photography
C. Six monthly digital retinal photography

D. Start ramipril
E. Start amlodipine

B. Annual digital retinal photography

The correct answer is annual digital retinal photography. This patient has developed preproliferative diabetic retinopathy and strict diabetic control is important. Apart from that, regular retinal photography can help find proliferative retinopathy in which case laser treatment will be needed. There is no evidence of hypertensive changes.

Diabetic retinopathy in type 1 diabetes

People with type 1 diabetes are at high risk of diabetic retinopathy. On diagnosis, they should be referred to screening services and from that point either undergo annual review or be referred to an ophthalmologist. If signs of proliferative retinopathy occur then the patient should be referred for retinal laser treatment.

Question 62 of 299

A 73-year-old patient is brought in by ambulance to the emergency department. He has had a sudden onset of right-sided weakness and dysphasia which started five hours ago. He undergoes a CT scan and an ischaemic stroke is confirmed with no evidence of acute bleeding.

Due to the delay in presentation to hospital, he is not thought to be suitable for thrombolysis.

He is started on aspirin and admitted to the stroke ward. He has significantly reduced mobility due to his weakness. He is considered to be at risk of haemorrhagic transformation.

How should his risk of venus thromboembolism be managed?

A. Low molecular weight heparin
B. Unfractionated heparin
C. Anti-embolic stockings
D. Intermittent pneumatic compression
E. Warfarin

D. Intermittent pneumatic compression

The correct answer is intermittent pneumatic compression. Patients admitted with stroke are very likely to be at high risk of developing a thromboembolic event due to reduced mobility, but low molecular weight heparin is associated with haemorrhagic transformation. NICE advises that patients admitted within three days of an acute stroke should have intermittent pneumatic compression considered. This should be provided for 30 days or until the patient is mobile or discharged.

Question 63 of 299

A 63-year-old woman returns to neurology clinic for review with her husband. She was diagnosed with Parkinson's disease two years ago and was started on ropinirole six months ago as her symptoms were becoming difficult to manage. She was mainly concerned with the rigidity of her movements. Since then she has improved remarkably, and her movements are much better, with reduced rigidity on examination. Her mood has also been improving with the relief from her symptoms.

However, her husband has become concerned that she has been increasingly spending large amounts on shopping, something which has not done before and that he feels is out of character.

What is the most likely explanation?

A. Normal behaviour
B. Progression of Parkinson's disease
C. Lewy-body dementia
D. Dopaminergic dysregulation syndrome
E. Impulse control disorder

A. Impulse control disorder

The correct answer is impulse control disorder. This is a patient recently started on a dopamine agonist who has subsequently developed reduced impulse control. This is not an unimportant symptom and can have significant implications, especially for

with a history of gambling. Before starting a dopamine antagonist this risk has to be explained to the patient. This symptom does not fit any of the other diagnoses described: there is no cognitive decline as would be expected with dementia, no worsening of Parkinsonian symptoms and no mood changes as would be expected in dopaminergic dysregulation syndrome.

Question 64 of 299

A 55-year-old man is reviewed in the psychiatric clinic. He has been referred by his GP who has been unable to manage his depression. He has a past medical history of hypertension, previous acute coronary syndrome one year ago, high cholesterol and depression. He mentions that his mood has worsened and that he is having persistent thoughts about taking his own life to the point where he is scared that he might 'do something'. There are no effects on his cognition, concentration or any sleep disturbance.

What advice should he be given in regards to driving?

A. No restrictions on driving
B. No restrictions on driving but must inform the DVLA
C. Can drive a car but not lorry or bus
D. Can drive a car but not lorry or bus and must inform the DVLA
E. Must not drive and must inform the DVLA

E. Must not drive and must inform the DVLA

The correct answer is that he must not drive and must inform the DVLA. He is a patient with depression and active suicidal thoughts and therefore should not drive.

Question 65 of 299

A 72-year-old male presents to the Parkinson's clinic with his wife, reporting increasing frequency and duration of 'off' periods. The couple finds these episodes extremely debilitating and occurs up to

11 times a day. The patient was diagnosed with Parkinson's disease 12 years ago. As a relatively young patient on diagnosis, he was commenced on ropinirole, which he continued for 5 years, before being prescribed Sinemet 6 times a day and entacapone for the following 7 years. Over past 2 years, the 'off' episodes have gradually increased in frequency in addition to the development of very mild involuntary jaw movements. He is very low in mood and has presented to the emergency department with two episodes of attempted paracetamol overdoses.

He would like a more effective treatment. What would you recommend?

A. Deep brain stimulation
B. Subcutaneous apomorphine
C. Reintroduce ropinirole at higher doses
D. Palliative care involvement
E. Trihexyl

B. Subcutaneous apomorphine

The treatment of 'off' symptoms is particularly important in patients who have been diagnosed with Parkinson's disease for a long time. The introduction of ropinirole would not help off periods. Trihexyl is an anti-cholinergic therapy for PD patients where tremor is the predominant symptom. In the context of suicidal behaviour, the patient would not be a candidate for deep brain stimulation, which for unknown reasons, increases the risk of suicide. Subcutaneous apomorphine is a reasonable option, administered as an infusion after a test bolus, which can be used to alleviate but not cure off symptoms.

Question 66 of 299

A 62-year-old male presents to the emergency department via blue light ambulance 90 minutes after sudden onset right sided weakness, expressive and receptive dysphasia. He is known to be T2DM, hypertensive on 3 agents and has a 40 pack year smoking history. On examination, you demonstrate a dense 0 out of 5 right hemiparesis. The patient has a complete expressive and receptive dysphasia, which you confirm to be new following a collateral history from his wife. The patient scores an NIHSS score of 7. He has no history of any recent surgery or head trauma. He is not on an anticoagulant and has no past medical history of coagulation disorders. His CT head demonstrates no areas of haemorrhage and a likely evolving area of ischaemic in the left middle cerebral artery territory. The radiologist calculated his ASPECT score to be 8. His observations on arrival are: temperature 36.7 degrees, heart rate 90/min and regular, blood pressure 220/150 mmHg, sats 99% on air, respiratory rate 20/min.

What is the most appropriate first-line treatment?

A. IV thrombolysis
B. Aspirin 300mg OD
C. PO amlodipine 2.5mg
D. MRI head with diffusion weighting sequences
E. IV labetalol

E. IV labetalol

The patient has multiple vascular risk factors with a sudden onset negative speech and motor syndrome this is strongly suggestive of an acute vascular event. The patient has a likely left MCA territory ischaemic infarct and is within the time window for thrombolysis (<4.5 hours in accordance with ECASS 3 trial results). The size of his infarct is also sufficiently small (calculated by ASPECT score, thrombolysis possible for all scores over 7) and his NIHSS score within range.

Thrombolysis is however not indicated in patients with blood pressures over 185/110 mmHg. Time is brain in the context of acute stroke the longer the delay to cerebral arterial reperfusion, the greater the size of infarct and subsequent functional deficits. The blood pressure is thus lowered rapidly with intravenous labetalol and not oral calcium channel blockers in this case.

Question 67 of 299

A 59-year-old male presents with 4-day history of sudden onset visual disturbance in his right eye. He reports it to be his first episode with no previous history of visual problems. His past medical history includes type 2 diabetes mellitus, hypertension and raised BMI. He is an active smoker of 40 pack years.

On examination, a right relative afferent papillary defect is detected. Pupils were equal in size. A visual field defect is demonstrated in the inferior nasal field of the right eye without a precise quadrantanopia or altitudinal pattern. Temporal arteries are non-tender and not thickened. Visual acuity on Snellen chart in left eye was 6/6, 6/18 in right. Colour vision on Ishihara plates were 17/17 on left, 5/17 on right. Fundoscopy is unremarkable. Examination of the upper and lower limbs are unremarkable, no language deficits are noted. Auscultation revealed normal heart sounds and no bruits.

His blood tests are as follows:

Hb	154 g/l
Platelets	190 * 10 /l
WBC	7.8 * 10 /l
ESR 5 mm/hr	
Na	141 mmol/l
K	3.9 mmol/l
Urea	5.6 mmol/l
Creatinine	80 µmol/l

What is the most likely diagnosis?

A. Right retinal infarct
B. Right temporal arteritis
C. Right non-arteritic ischaemic optic neuropathy
D. Left middle cerebral artery ischaemic stroke
E. Left occipital ischaemic stroke

C. Right non-arteritic ischaemic optic neuropathy

Visual field defects are a common presentation in clinical practice and MRCP part 2. The first feature to notice here is the monocular presentation, which indicates the lesion must be anterior to the optic chiasm. Next, fundoscopy is unremarkable, which would be unusual for a retinal infarct, most likely secondary to central or branch retinal artery occlusion.

Typically, an acute retinal infarct should reveal a classic cherry red spot, pale fundus and possible visualisation of the embolus. On locating the lesion to be at the optic nerve, the cause must be established: typically is this optic neuritis, arteritic or non-arteritic anterior ischaemic optic neuropathy. Optic neuropathy typically produces a correlated deficit in visual acuity with colour vision, allowing differentiation from optic neuritis, where colour vision is frequently better preserved.

Lastly, this patient has a normal ESR, is under 60 and has no local or systemic features of vasculitis, particularly temporal arteritis. In addition, he has multiple vascular risk factors, making a nonarteritic ischaemic anterior optic neuropathy most likely. Management involves optimisation of vascular risk factors and there is limited evidence for a tapering dose of oral prednisolone. Most patients can expect to improve by 3 lines of a Snellen chart by 6 months.

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A 69-year-old male presenting with a 4-month history of progressive 'droopiness' of his left eyelid. He reports that in the last 2 weeks, he has been unable to lift his eyelid at all, which he says is fortunate considering he gets double vision now when he lifts his eyelid with his fingers. He underwent a renal transplant in 1988, which continues to function well, and previously had a squamous cell and malignant melanoma resected 2 and 5 years ago respectively.

On examination, you note complete ptosis, loss of vertical eye movements and loss of adduction in his left eye. On head tilt to the right, he is unable to depress left eye. His right eye demonstrates full eye movements. The left pupil is unreactive and larger in diameter compared to the right. There is also loss of sensation at the left nasal skin fold and left forehead with sparing of his left chin. He has no facial weakness, hearing loss or palatal asymmetry. He denies headache or retroorbital pain.

Where is the likely lesion?

A. Left ocular neuromuscular junction
B. Left cavernous sinus
C. Left superior orbital fissure
D. Cerebral aqueduct
E. Left midbrain

B. Left cavernous sinus

The patient has deficits of his oculomotor nerve with parasympathetic involvement, involvement of the trochlear nerve and also the first and second branch of the trigeminal nerve, hence sparing of his left chin as supplied by the mandibular branch of the trigeminal nerve. Loss of pupillary reflexes in the left eye is a result of third nerve involvement and not necessarily retinal or optic nerve pathology. Ability to abduct the left eye suggests the abducens nerve is intact. The only anatomical location involving all these structures is the left cavernous sinus.

The possible cause for this cavernous sinus syndrome is interesting: the insidious gradual course goes against an acute cavernous sinus thrombus. A significant, expanding internal carotid aneurysm, which runs inside the cavernous sinus with the parasympathetic nerves wrapped around it, is a possibility. However, the key is the recognition that the patient is chronicallyimmunosuppressed due to his renal transplant, increasing his risk of opportunistic infections such as fungi and also dermatological malignancies.

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A 66-year-old female is brought into Emergency Department by her distraught husband, who reports that she has not been out of bed for 3 days and seems unable to recognise him. He reports no recent fevers or rigors, she has not complained of cough, dysuria, sore throat or diarrhoea and vomiting prior to being unwell. She is well known to local neurologists: she is undergoing off-label rituximab for difficult to manage relapsing-remitting progressive multiple sclerosis, with previous white matter lesions in her corpus callosum, periventricular white matter, and lower thoracic transverse myelitis.

Over the past 6 years, she had continued to relapse despite interferon beta, natalizumab, mitoxantrone and cyclophosphamide. At baseline, she is weak in her lower limbs and is able to stand only with assistance of a turning apparatus, then able to walk about 3 steps. She is normally cognitively intact and fully orientated, normally responsible for organising the couples finances. Her past medical history includes type 1 diabetes mellitus, autoimmune thyroiditis and previous deep vein thrombosis 5 years ago (warfarin since discontinued). Her medication history includes lantus, novomix, levothyroxine and 3 monthly infusions of rituximab, which had stably maintained her MS for 4 years.

On examination, she is not orientated to time or place and appears extremely irritable. Halfway through the examination, she becomes extremely tearful. There is a full range of neck movements and no photophobia.

Neurological examination is difficult due to patient cooperation: pupils are equal and reactive, there is a full range of eye movements and no obvious facial asymmetry. Power in her upper limbs were at least 3/5, lower limbs are unable to counteract gravity. Both plantar responses are upgoing. Her bloods are as follows:

Hb	134 g/l
Platelets	268 * 10 /l
WBC	7.8 * 10 /l
Na	137 mmol/l
K	4.4 mmol/l
Urea	4.5 mmol/l
Creatinine	60 µmol/l
CRP	5 mg/l
HIV	negative
Glucose	7.4 mmol/l

A lumbar puncture was performed:

WBC	16 /mm^3
RBC	150 /mm^3
Protein	0.71 g/l
Glucose	3.5 mmol/l
PCR and gram staining	awaited

What is the most appropriate management step?

A. Intravenous aciclovir
B. Intravenous methylprednisolone
C. Intravenous immunoglobulin
D. Intravenous rituximab infusion
E. Psychiatric input

A. Intravenous aciclovir

This is a difficult question regarding the side effect of rituximab and opportunistic infections. The neurological signs are difficult to interpret. Firstly, there are no hard clinical signs that her MS has deteriorated: her plantars are likely to be upgoing at baseline due to previous transverse myelitis while a symmetrical bilateral mild reduction in power is expected as a result of decompensation during acute illness. As a result, further aggressive immunosuppression would not be appropriate.

Rituximab results in B cell depletion through incompletely understood processes. An acute deterioration in cognition and agitation, in the context of an immunosuppressed, should raise strong suspicions of encephalitis or meningitis. The patient might not raise a strong immune response, hence relatively modest number of white cells in CSF and CRP, but the raised CSF protein would be consistent with viral encephalitis. A CSF:serum glucose of around 1:2 lowers the risk of bacterial CNS infection. The patient should be covered for possible herpes simplex or varicella-zoster encephalitis with intravenous aciclovir while awaiting CSF PCR.

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A 57-year-old male presents with a 3 months history of increasing clumsiness in his hands and arms. He has a complicated past medical history: 22 years ago, he underwent a renal transplant after a progressive deterioration in his renal function following diagnosis with autosomal dominant polycystic kidney disease aged 25. His transplant has functioned well since but the patient has since undergone two resections of squamous cell carcinomas and one serious lengthy hospital admission for a systemic fungal infection. He stopped working a year ago as a wine merchant, after complaining that he was no longer able to differentiate 'the smells of his wines as he got older'.

On examination, he is alert and orientated. Questioning was challenging due to his hearing impairment, despite bilateral hearing aids. He scored 17/30 on a mini-mental examination. Pupils were reactive with a full range of eye movements. Facial power and sensation were normal, with symmetrical palatal elevation and no tongue deviation. He was profoundly deaf bilaterally. Tone, power and sensation were normal, reflexes were present with downgoing plantars. However, you note significant bilateral finger-nose dysmetria and heel-shin mal co-ordination.

Which investigation is likely to produce the unifying diagnosis?

A. Lumbar puncture for cerebrospinal fluid including 14-3-3, S100
B. EEG
C. MRI head
D. Neurogenetic testing
E. Nerve conduction studies and EMG

C. MRI head

The patient has presented with anosmia, bilateral symmetrical ataxia, cognitive impairment and sensorineural hearing loss, classical symptoms of superficial siderosis, which would be demonstrated on MRI head, with siderosis typically prominent in the posterior fossa. The underlying cause for iron deposition is thought to be due to chronic or previous intracerebral haemorrhages. In the case of this patient, the association of polycystic kidney disease with brain aneurysms are likely to be significant. In other patients, previous neurosurgery or head trauma can similarly result in siderosis. There is no current treatment for superficial siderosis but case reports have demonstrated possible improvements with lipid-soluble iron chelators.

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A 78-year-old male presents to you with progressive weakness in his arms. He first saw his GP about 4 months ago complaining of a weakness in his right wrist and was told he had carpal tunnel syndrome, for which he was placed on a waiting list to see the orthopaedic surgeons. Over the next 4 months, he noticed progressive weakness in his right wrist and in bending his right elbow. He noted his right hand was becoming increasingly wasted. Two months ago, he noted similar weakness in his left wrist. He reports no changes to sensation, normal speech and swallowing. On examination, cranial nerves were unremarkable.

Both hands were markedly wasted with significant loss of musculature and fasciculations bilaterally. Reflexes were muted in both upper limbs with negative Hoffman's sign. Sensory examination in the upper limbs were unremarkable, he was unable to perform the finger-nose examination. Examination of the lower limbs was also unremarkable but the patient felt a subjective weakness in both hip flexion. MRI imaging of the patients head and spine demonstrated no significant lesions. Nerve conduction studies demonstrated f waves and H-reflexes, multiple focal areas of demyelination and motor neuropathic blocks.

What is the most appropriate management?

A. Intravenous cyclophosphamide
B. Intravenous immunoglobulin
C. Intravenous methylprednisolone
D. Plasma exchange
E. Riluzol

B. Intravenous immunoglobulin

The patient has presented with a pure motor peripheral neuropathy with multifocal areas of demyelination and motor block, with f waves and H-reflexes suggestive of late motor responses to electrical stimuli, suggestive of multifocal motor neuropathy with conduction block (MMN-CB). MMN-CB is an immune-mediated disorder, commonly associated with anti-GM1 antibodies, typically presenting with asymmetric bi-brachial motor weakness without sensory or bulbar involvement, allowing the key distinction from motor neurone disease. MMN-CB is not responsive to steroids or plasma exchange, unlike chronic demyelinating polyneuropathy (CIDP), which typically involves sensory nerves too.

Cyclophosphamide may have some efficacy but is generally not used in MMN-CB due to its toxicity profile.

Riluzole is a drug for motor neurone disease.

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A 45-year-old female presents with small multiple ischaemic infarcts in the right cerebellar hemisphere confirmed on MRI, associated with small haemorrhages within two areas of infarct. A subsequent CT angiogram 24 hours into the admission demonstrated a right vertebral artery dissection with a free flowing thrombus visualised.

What is the optimal management?

A. IV heparin infusion
B. 300mg aspirin 14 days followed by clopidogrel 75mg
C. Load with warfarin
D. Treatment dose low molecular heparin
E. Hold off antiplatelet and anticoagulation

A. IV heparin infusion

It is generally accepted that visualised thrombus requires anticoagulation to prevent it increasing in size and consequent possible embolic showering. However, the patient is still in the hyperacute phase with haemorrhagic transformation within small areas of infarct. The optimal management in this scenario is to offer anticoagulation with the greatest reversibility should intracranial haemorrhage occurs: intravenous heparin is tricky to manage but can be reversed the fastest compared to subcutaneous low molecular weight heparin and warfarin. The patient will require close hourly neurological monitoring and urgent repeat imaging if GCS decreases or new or deteriorating focal neurology presents.

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A 65-year-old lady was referred to the neurology clinic for assessment. She reported a 3-month history of progressive weakness. She had initially noticed difficulty opening jars but reported that over the past month she had also had difficulty walking up stairs. She did not have any pain and reported no changes in sensation. Past medical history included osteoporosis, type 2 diabetes mellitus and hypertension.

On neurological examination there were no fasciculations, the tone was normal and the sensation was intact. Power was reduced in finger flexion (3/5), wrist flexion (4/5), knee extension (3/5) and hip flexion (4/5) bilaterally. Other movements were relatively spared. Upper limb reflexes were present but diminished, but the knee jerk was absent. The plantar response was flexor bilaterally. There was no tenderness over any muscle groups. Cranial nerve examination was unremarkable.

Blood results are shown below:

Haemoglobin	122 g/l
White cell count	8.2 x 10^9/l
Platelets	376 x 10^9/l
C reactive protein	7 mg/l
Erythrocyte sedimentation rate	39 mm/hr
Creatine kinase	272 (24-170 U/l)

What is the most likely diagnosis?

A. Diabetic amyotrophy
B. Inclusion body myositis
C. Motor neuron disease
D. Myasthenia gravis
E. Polymyositis

B. Inclusion body myositis

Inclusion body myositis is the most common primary myopathy in this age group. It manifests as a slowly progressive weakness, usually affecting finger and wrist flexion initially. Lower limbweakness may also occur. Reflexes are usually diminished as in other myopathies. Creatine kinase levels are usually normal or only mildly raised, in contrast to polymyositis (where creatine kinase levels are usually markedly elevated). Muscles are often tender in polymyositis, and the distal muscles are usually not affected until the disease is advanced.

Motor neuron disease is an important differential to consider, but the lack of upper motor neuron signs and fasciculations goes against this. There is no description of fatigability that would lead one to consider myasthenia gravis, and no evidence of bulbar, facial or ocular weakness. Diabetic amyotrophy is characterised by painful wasting of the proximal lower limb muscles

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A 45 year old man develops weakness of the extensors in the wrist and digits of his left upper limb over a week. Over the next 2 months he also notices weakness of the small muscles of hands on the right side, and that he is progressively unable to dorsiflex his right foot. On examination, he has wasting and 2/5 power in the left wrist and digit extensors. On the right he has clawing of the right ring and little fingers along with wasting of the small muscles of the hand on that side (except the thenar eminence and the first two lumbricals, which are spared). He has a right foot drop along with wasting of the anterior tibial and perineal muscles on that side. Fasciculations are seen in all of the areas of weakness. Sensory examination and reflexes are normal, along with no clonus. Plantars are down-going. The remainder of the examination are normal. Nerve conduction studies show conduction block.

What is first-line maintenance treatment for this condition?

A. Riluzole
B. Intravenous immunoglobulin
C. Pyridostigmine
D. Steroids
E. Beta-inteferon

B. Intravenous immunoglobulin

The answer here is the condition called multifocal motor neuropathy with conduction block (MMNCB). These patients are usually younger to middle-aged men who develop focal arm weakness in the distribution of a named nerve. It usually happens quite suddenly (e.g. over a week) and they are often mistaken as a stroke at first presentation. However, over several months additional named motor nerves become involved asymmetrically such that MMNCB may eventually look like motor neurone disease (MND) - the principle differential here (specifically the lower motor neurone progressive muscular atrophy form rather than the mixed upper/lower motor neurone ALS form). For exam purposes, the key difference between these two conditions is the nerve conduction studies. MMNCB shows conduction block. MND does not. MMNCB is a demyelinating condition, much in the same way Guillain Barre or chronic inflammatory demyelinating polyneuropathy (CIDP) are. However, in MMNCB this demyelination is in segments of a nerve rather than affecting the whole nerve. Therefore, the action potentials (as well as being slowed because of demyelination) actually don't get past the areas of conduction block. The importance of doing nerve conduction studies is to decide whether a motor neuropathy is axonal (i.e. the axon itself getting damaged) or demyelinating. The demyelinating ones tend to be more easily and similarly treatable. MMNCB, Guillain Barre, and CIDP are all good examples ofdemyelinating neuropathies, all of which therefore respond to intravenous immunoglobulin (IVIG).

Note that the nerves involved in this particular case are the left radial nerve, right ulnar nerve, and right common perineal nerve.

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A 45-year-old lady has had weight loss over the last eight months. Over the last 3 months, she has become confused according to her family, forgetting things she would normally remember like telephone numbers or what she did during the day. She has now presented with a second episode of what sounds like a generalised tonic-clonic seizure in the last week. She has no other past medical history of note. Clinical examination shows MMSE of 23/30, cachexia, a bulky left adnexal region, and nil else. Routine blood tests are unremarkable. You organise a CT chest, abdomen, pelvis and head which reveal a suspicious lesion in the left ovary but are otherwise normal. You organise a biopsy of the left ovary, send off a paraneoplastic blood screen, and order an MRI of the brain.

What would you expect the paraneoplastic screen to return showing?

A. Voltage-gated potassium channel antibodies
B. Anti-Hu antibodies
C. Anti-GAD
D. Anti-Ma2 antibodies
E. NMDA receptor antibodies

E. NMDA receptor antibodies

The diagnosis is limbic encephalitis. Limbic encephalitis represents a group of autoimmune conditions characterised by inflammation of the limbic system and other parts of the brain. People with limbic encephalitis typically presents with a subacute development of memory impairment, confusion, and alteration of consciousness, often accompanied by seizures and temporal lobe signal change on MRI.

It is important to diagnose the underlying autoimmune aetiology of limbic encephalitis as this might point you toward the underlying diagnosis of cancer. This is to say that limbic encephalitis may often present as a paraneoplastic syndrome, although not always. The antibodies that cause limbic encephalitis and their associated malignancies are:

- NMDA receptor antibodies = ovarian cancer
- Voltage-gated potassium channel antibodies = Thymoma or small cell lung cancer
- Anti-Hu antibodies = Small cell lung cancer
- Anti-GAD = Thymoma

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A 47 year old female with relapsing-remitting multiple sclerosis (MS) describes increasing difficulty with fatigue, and has had to give up her work as a medical secretary. She has always been careful about ensuring a balanced diet, gentle exercise and early treatment of infection. On review and questioning her mood is good. Her most recent bloods are shown below:

Hb	120g/dl
Platelets	150 * 10 /l
WBC	5.2 * 10 /l
Na	130 mmol/l
K	3.8 mmol/l
Urea	6.0 mmol/l
Creatinine	68 µmol/l

B12, folate and TFTs are normal.

What is the best course of action?

A. Advise that she increase caffeine intake
B. Advise that increasing exercise may benefit
C. Trial of vitamin D
D. Trial of amantadine
E. Trial of ropinirole

D. Trial of amantadine

The first step in her management should be to exclude anaemia, thyroid dysfunction, sleep or mood disturbance. With the above results and knowledge that she is living a well balanced lifestyle, the next step would be a trial of amantadine.

Amantadine is an anti-viral agent that slows viral replication. It is also known to have some dopaminergic effects; the mechanism of action for reducing fatigue in MS however, remains unclear. It is currently recommended by NICE as a potential treatment, although a Cochrane review in 2006 was inconclusive.

Increasing caffeine intake and levels of exercise would not be recommended in this patient. Similarly, vitamin D has not be proven to be of benefit in MS. Ropinirole is a dopamine agonist used in parkinsons disease but not in MS.

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A 26-year-old female from the travelling community presents to A&E following a seizure. History from her family suggests that she was suffering with headaches over the last 24 hours and this morning was feverish and vomiting. Her partner says she was unwell 1 month ago with a fever and whole body rash that spontaneously resolved. There is no other significant past medical history.

On examination she appears drowsy. She has a left sided hemiparesis with bilateral nystagmus. Fundoscopy reveals papiloedema. There are no skin rashes.

What is the most likely underlying diagnosis?

A. Meningococcal septicaemia
B. Meningitis secondary to herpes simplex virus
C. Progressive multifocal leukoencephalopathy (PML)
D. Multiple sclerosis
E. Acute disseminated encephalomyelitis (ADEM)

E. Acute disseminated encephalomyelitis (ADEM)

In the case given there is no history of preceding neurological symptoms and the acute onset of the neurology points away from the diagnosis of MS. In the examination findings it is specified there is no rash making meningococcal septicaemia very unlikely. PML is only seen with significant immunosupression and presents with a subacute picture. This leaves HSV as a plausible diagnosis however ADEM secondary to measles is more likely given the previous full body rash in a member of the travelling community who may not have been vaccinated.

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A 65-year-old man, originally from Pakistan is referred to the rapid access chest clinic by his GP with a one month history of weight loss (of roughly 5 kg) and a dry, irritating cough. Over the past two days he has started to have loss of sensation in both of his feet and lower legs.

Examination reveals heart sounds 1 and 2 present with no added sounds, reduced air entry in the mid and lower left zones of the chest, a non-tender liver edge that extends 3cm below the costal margin and loss of sensation for pain, light touch and temperature in both feet up to the mid shin.

A chest X-ray is performed which shows a unilateral hilar mass, but no other consolidation or features. His past medical history includes mild hypertension and diabetes mellitus which is diet controlled. His only medication is enalapril for his hypertension. Bloods reveal no obvious abnormality.

What is the most likely diagnosis?

A. Squamous cell carcinoma
B. Small cell carcinoma
C. Adenocarcinoma
D. Tuberculosis
E. Sarcoidosis

B. Small cell carcinoma

Small cell carcinoma is the correct diagnosis in this case. The presentation of rapid onset neurological symptoms (sensory neuropathy) alongside constitutional symptoms and a hilar mass on chest X-ray is highly suggestive of small cell lung cancer (SCLC) with paraneoplastic syndrome. SCLC is strongly associated with paraneoplastic syndromes, particularly Lambert-Eaton myasthenic syndrome and peripheral neuropathies. The rapid progression of symptoms is also characteristic of SCLC, which typically has a more aggressive course than other lung cancers.

Squamous cell carcinoma typically presents as a central mass and can cause hilar lymphadenopathy, but it is less commonly associated with paraneoplastic syndromes. While it can present with constitutional symptoms like weight loss, the neurological manifestations would be unusual for this type.

Adenocarcinoma is typically peripherally located in the lung and would be less likely to present as a hilar mass. While it can metastasise to cause neurological symptoms, these would more commonly be due to direct spread rather than paraneoplastic phenomena.

Tuberculosis, while common in patients from Pakistan and capable of causing hilar lymphadenopathy, typically presents with more systemic symptoms such as night sweats and haemoptysis. The neurological symptoms would be unusual for TB unless there was direct CNS involvement, which would typically present differently.

Sarcoidosis can present with hilar lymphadenopathy but is typically bilateral rather than unilateral. While neurological involvement can occur in sarcoidosis, it typically affects the cranial nerves rather than causing a peripheral neuropathy. The weight loss and rapid progression would also be unusual for sarcoidosis.

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A 64-year-old man is referred to you from a psychiatrist for a second opinion. He initially presented with a 3-month history of low mood, apathy and suicidal ideation. In addition he was asked to retire early from his job as an accountant as he was performing poorly at work. He is also sleeping an average of 14 hours per day.

When the psychiatrist assessed him he noted abnormal jerky movements in the lower limbs as well as a broad based gait. An MMSE was performed and he scored 15/30. This is corroborated by your examination and you also note hyperreflexia in the lower limbs and nystagmus.

He has no history of cognitive impairment or any psychiatric history. There is no family history of any neurological or psychiatric conditions and his only past medical history is an appendectomy 20 years ago which was complicated by a large intraperitoneal bleed.

Which findings are you most likely to find on investigation?

A. Increased T2 and FLAIR signal intensity in the putamen and head of the caudate on T2 weighted MRI, 14-3-3 protein on CSF
B. CAG trinucleotide repeats on the short arm of chromosome 4
C. Increased T2 and FLAIR signal intensity in the putamen and head of caudate on T2 weighted MRI and oligoclonal bands on CSF
D. CAG trinucleotide repeats on the short arm of chromosome 12
E. Cortical atrophy, most prominent in the frontal lobes

A. Increased T2 and FLAIR signal intensity in the putamen and head of the caudate on T2 weighted MRI, 14-3-3 protein on CSF

The diagnosis here is Sporadic Creutzfeld Jakob Disease. CJD is characterised by a rapidly progressive dementia along with neuropsychiatric changes. Death usually occurs within a year of diagnosis. Most common psychiatric symptoms are depression and apathy although euphoria, anxiety and emotional lability can also be seen. Myoclonus, ataxia, nystagmus and hyperreflexia are the most common neurological symptoms.

A represents the most likely findings for sporadic CJD. Sporadic CJD normally has a longer clinical course than nvCJD and is normally seen in older people (although nvCJD is possible in this individual as he may have has a blood transfusion 20).B represents a finding seen in Huntingtons chorea. The lack of family history makes this diagnosis less likely. The oligoclonal bands in C are seen in association with Multiple Sclerosis not CJD. D is not a recognised investigation finding and E would be more consistent with a diagnosis of Picks Disease. The very rapid onset of symptoms and presence of myoclonic jerks make this answer less likely

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A 55-year-old man presents to the Accident and Emergency department and a medical referral is requested. He reports that for the past week he has been hearing voices. He states that he cannot recognise who the voices are as they are whispering quietly but he thinks that they are making derogatory comments about him. He denies any visual hallucinations and you cannot elicit any delusional beliefs. His mood appears euthymic.

He tells you that he has a long history of alcohol dependence lasting for 30 years drinking approximately 10 units of alcohol a day on average. He tells you that in the past he has tried to stop drinking alcohol but this has caused admission to hospital due to seizures. He is particularly worried because he has cut down on alcohol since the hallucinations because he is worried that he is going mad. He is now only drinking 2 units a day and has not drunk any alcohol for 24 hours. On one previous occasion where he abstained from alcohol, he said that he had hallucinations and had to be admitted to hospital for a few days and put on a drip and was told that he almost died.

Na	144 mmol/l
K	3.6 mmol/l
Urea	14.1 mmol/l
Creatinine	119 µmol/l
Bilirubin	36 µmol/l
ALP	199 u/l
ALT	92 u/l
γGT	271 u/l
Albumin	36 g/l

He has a family history of alcohol dependence and depression but no other psychiatric problems. His medications include Omeprazole, Vitamin B, Thiamine and Diazepam.

On examination his GCS is 15, there is no tremor or sweating Pulse 80 regular BP 138 / 74 chest clear, abdo soft non tender, no peripheral focal neurology MMSE 28/30 He is commenced on chlordiazepoxide and observed for 24 hours. His GCS remains at 15 and his repeat physical examination remains unchanged and the hallucinations are still present

What is the most likely diagnosis?

A. Late onset schizophrenia
B. Delerium tremens
C. Alcohol withdrawal syndrome
D. Alcoholic hallucinosis
E. Hepatic encephalopathy grade 2

D. Alcoholic hallucinosis

Alcoholic hallucinosis is a rare condition that can occur during intoxication or withdrawal, but occurs without a clouding of consciousness. Characteristically there are auditory hallucinations which can be vague at first but then can develop into clear voices with a derogatory or persecutory content. The hallucinations normally resolve after less than 6 months.

Delirium tremens will present with a clouding of consciousness and a fluctuating or reduced GCS should be seen when the patient is observed. Also in Delirium Tremens, visual hallucinations are more common, with Lilliputian hallucinations being characteristic of this condition.

Alcohol withdrawal syndrome does not typically cause auditory hallucinations. In addition there is no evidence of a withdrawal syndrome as his pulse is normal there is no sweating or tremulousness. It is possible he has been self medicating with diazepam during his period of abstinence from alcohol.

Schizophrenia is unlikely as a diagnosis as the symptoms are too acute. In addition, affective changes and delusions and family and personal history would be more likely with a diagnosis of schizophrenia.

Hepatic encephalopathy is not the diagnosis. Although there are deranged LFTS, there is no reduction in GCS and there is no liver flap .In grade 2 encephalopathy there should be definite impairment of concentration and attention. However, this is not seen in this individual as evidenced by the high score on the MMSE

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A 65-year-old man was referred to neurology outpatient clinic with a six month history of double vision. He had first noticed this when reading before going to bed but had more recently been occurring earlier during the day whenever the patient concentrated on an activity. The patients wife also reported that she had been struggling to hear the patient when he spoke to her and that on several occasions the patients eyelids had drooped during late in the day. The patient denied experiencing any weakness in his arms or legs but stated that his general mobility was normally very limited due to osteoarthritis of both his knees. Other past medical history included hypercholesterolaemia, hypertension and diverticular disease. Regular medications included bendroflumethiazide 2.5 mg OD, simvastatin 40 mg OD, co-dydramol as required. The patient was a retired builder and lived alone with his wife. He was an ex-smoker who rarely consumed alcohol.

Examination showed normal pupillary reflexes, visual acuity and visual fields. No ptosis was observed. Assessment of eye movements demonstrated a complex ophthalmoplegia with diplopia in all directions of gaze. There was significant weakness of bilateral facial muscles with bilateral involvement of the forehead. Examination of the tongue and palette was normal. Peripheral nerve examination was unremarkable except for evidence of fatiguable muscle weakness in the upper limbs.

Chest x-ray: clear lung fields with no effusion; no cardiomegaly; possible mediastinal widening

Haemoglobin	13.5 g / dL
White cell count	8.9 * 10 /l

Platelets	409 * 10 /l
Urea	4.7 mmol / L
Creatinine	95 micromol / L
Sodium	137 mmol / L
Potassium	4.8 mmol / L
Calcium (adjusted)	2.5 mmol / L

What is the single most sensitive investigation for the likely diagnosis?

A. Repetitive nerve stimulation neurophysiology
B. Serum acetylcholine receptor antibodies
C. Serum muscle specific tyrosine kinase antibodies
D. CT mediastinum
E. Single fibre electromyography

E. Single fibre electromyography

The patient is presenting with symptoms and signs consistent with myasthenia gravis. All of the above investigations would be appropriate in this context but have variable sensitivities for myasthenia gravis as listed below:

- Single fibre electromyography: sensitivity 92-100 %
- Repetitive nerve stimulation neurophysiology: sensitivity 70 %
- Serum acetylcholine receptor antibodies: sensitivity 85 %
- Serum muscle specific tyrosine kinase antibodies: sensitivity 40-70 %

The Edrophonium (Tensilon) test has a high sensitivity for myasthenia gravis but is no longer used due to risk of fatal cardiac arrhythmia. Around 10 % of patients with myasthenia gravis also have a thymoma and therefore CT scanning is an important part of assessment of these patients. However, this investigation will no yield a diagnosis.

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A 65-year-old man underwent an elective inguinal hernia repair. Due to the list running late into the evening, the patient was admitted for an overnight stay. During the night after the operation the patient was observed to have an increasing oxygen requirement and the following morning was referred to the oncall medical registrar.

The patient reported feeling progressively more short of breath since the operation, particularly when he had tried to lie down to sleep. He denied any cough, chest pain, leg swelling or palpitations. Prior to the operation the patient had been generally well although he had found that he frequently experienced double vision when reading especially in the evening. He also had noticed some difficulties when chewing tough foods in recent weeks. Past medical history was unremarkable and the patient took no regular medications.

Examination revealed a regular pulse, no elevation of jugular venous pressure and normal heart sounds. Both calves were soft and non-tender. The patient had a shallow respiratory effort and was unable to speak in full sentences. Chest expansion was reduced bilaterally, chest was resonant with vesicular breath sounds throughout. The patient had bilateral weakness of facial muscles and ptosis on prolonged upward gaze. Power of neck flexion and extension was reduced, graded as 4/5.

Basic observations:

- Blood pressure: 120 / 76 mmHg
- Heart rate: 115 beats / min
- Respiratory rate: 32 breaths / min
- Temperature: 36.8ºC

Portable CXR: technically poor film due to poor inspiratory effort; clear lung fields; no pleural effusion; no upper lobe blood diversion; no free air under diaphragm.

Arterial blood gas analysis (35 % O2)

pH	7.29
PaCO2	6.6 kPa
PaO2	8.7 kPa
Bicarbonate	18 mmol / L (reference 20.0-26.0)
Lactate	2.1 mmol / L

Bedside forced vital capacity: 1.9 L

What is the most important next step in management?

A. Pyridostigmine
B. Referral to intensive care unit
C. Reduce percentage of supplemental oxygen
D. High-dose corticosteroids
E. Intravenous immunoglobulin

B. Referral to intensive care unit

The patient has symptoms and signs of previously unrecognised myasthenia gravis. The physiological stress of surgery has precipitated a myasthenic crisis associated with type 2 respiratory failure, tachycardia, tachypnoea and reduced forced vital capacity. The priority in management is to refer the patient to an intensive care unit for respiratory support.

Corticosteroids and pyridostigmine are mainstays of treatment of myasthenia gravis with intravenous immunoglobulin also used in severe cases. However, these treatments cannot be expected to restore respiratory muscle function rapidly in this emergency situation.

The patient has no history of obstructive lung disease with type 2 respiratory failure due instead to failure of ventilation due to respiratory muscle weakness. Therefore, reducing percentage of supplemental oxygen would not be beneficial and would cause additional hypoxia.

Question 83 of 299

A 48-year-old woman presented to the emergency department with a severe headache. Symptoms had started early that day while the patient had been walking around her office with the headache reaching maximal intensity within a few minutes. The pain was felt across the entirety of the patient's head and was much improved when she lay down flat. There were no associated symptoms and the patient had been constitutionally well in the preceding days.

The patient had no past medical history and was nulliparous. She was a non-smoker who consumed 15 units of alcohol per week.

Clinical examination demonstrated no evidence of focal neurological deficit and no signs of meningism. Simple analgesia given in the emergency department had limited impact on the patient's headache.

CT brain with venogram: no evidence of intra-axial or extra-axial bleeding; no space occupying lesion; no hydrocephalus; no evidence of venous sinus thrombosis

Lumbar puncture: opening pressure 5 mmHg; red cells 8 mm³; white cells 1 / mm³; no xanthrochromia

What is the next best investigation to confirm the likely diagnosis?

A. Digital subtraction myelography
B. Cerebral angiography
C. CT brain with contrast
D. MRI whole spine with STIR
E. MRI brain with gadolinium

E. MRI brain with gadolinium

The clinical presentation and initial investigations are suggestive of spontaneous intracranial hypotension. The key aspect of the history is the strong postural relationship to pain with the headache worsening within a few seconds of upright posture and easing within a minute of lying horizontal. Spontaneous intracranial hypotension can cause either a headache of insidious or rapid onset. A low opening pressure on lumbar puncture (< 6 cmCSF) is one of the diagnostic criteria.

Diagnosis is confirmed by MRI with gadolinium that demonstrates distinctive dural gadolinium enhancement and downward displacement of brain on sagittal views.

In some cases of spontaneous intracranial hypotension, investigations such as MRI whole spine with STIR sequences or digital subtraction myelography may be used to identify the location of CSF leak in order to guide blood patch treatment.

Question 84 of 299

A 55-year-old male attends the clinic with low mood. He also describes anhedonia and anergia. On mental state examination you note passive suicidal ideation, psychomotor agitation, and poverty of thought. He was recently started on a new medication for treatment of Huntington's chorea.

What drug may have been the cause of this presentation?

A. Citalopram
B. Venlafaxine
C. Mirtazapine
D. Risperidone
E. Reserpine

E. Reserpine

VMAT inhibitors such as reserpine can cause drug induced depression

Important for me Less important

The clinical features are suggestive of depression. Before diagnosing a mental health disorder it is extremely important to rule out organic and drug induced causes. For example, organic pathology such as hypothyroidism can cause depression. Several drugs have been implicated in drug induced depression including isotretinoin and vesicular monoamine transporter (VMAT) inhibitors (e.g. reserpine).

Therapeutic options for Huntington's chorea include dopamine-depleting agents (e.g. reserpine, tetrabenazine) and dopamine-receptor antagonists (e.g. neuroleptics). It is likely that the patient has been started on reserpine which has resulted in drug induced depression.

Question 85 of 299

A 68-year-old man presents with a weak left leg. He states that he first noticed it a few weeks ago. On examination you note weakness of left ankle plantar flexion.

Nerve conduction studies of the tibial nerve are as follows:

- Conduction velocity 25 metres per second (normal >50) Amplitude of 21mV (normal >17)

What is the most likely cause?

A. Uraemia
B. Multiple myeloma
C. HIV
D. Vitamin B12 deficiency
E. Diabetes mellitus

B. Multiple myeloma

When interpreting NCS, a reduced conduction velocity but normal amplitude is indicative of demyelinating pathology

Peripheral neuropathies can result from axonal degeneration, demyelination, or a combination of the two. Generally, axonal neuropathies are caused by metabolic, toxic or heritable causes. Demyelinating neuropathies are generally caused by autoimmune processes, paraproteinaemia or the result of an inherited disorder of myelin

It is very difficult to distinguish between the two on clinical grounds, hence why nerve conduction studies (NCS) are very useful for making the distinction.

When interpreting NCS, a reduced amplitude but normal conduction velocity is indicative of axonal pathology. Causes of a predominant sensory axonal peripheral neuropathy include diabetes mellitus (commonest cause), B12 deficiency, uraemia, carcinoma (paraneoplastic) and HIV.

The NCS in this case confirm a demyelinating process. Multiple myeloma is the only given option which causes demyelination. The other options are causes of an axonal peripheral neuropathy.

Question 86 of 299

A 54-year-old man presents with seizures. His wife states that they recently returned from Kenya. Observations are as follows: heart rate 96 beats per minute, blood pressure 130/85 mmHg, respiratory rate 16 breaths per minute, SpO2 96% (on air), temperature 38.6ºC.

An MRI scan is performed:

MRI head General oedema of the medial temporal lobes on T1

What is the most likely pathogen?

A. Coxsackie
B. Herpes simplex virus type 1
C. Herpes simplex virus type 2
D. Varicella zoster virus
E. West Nile virus

B. Herpes simplex virus type 1

CT head showing temporal lobe changes - think herpes simplex encephalitis

Herpes simplex virus type 1 is correct. The clinical ad radiological features are consistent with herpes simplex encephalitis. HSV-1 is responsible for 95% of cases of viral encephalitis in adults.

Coxsackie is incorrect. Coxsackie or Echovirus groups of enteroviruses are the most common cause of viral meningitis. The patient in this clinical case has clinical features of encephalitis (e.g. seizures) rather than meningitis (headache, neck stiffness, and photophobia).

Herpes simplex virus type 2 is incorrect. It is herpes simplex virus type 1 which is more commonly implicated in viral encephalitis.

Varicella zoster virus is incorrect. Although this remains within the differential diagnosis, the most common cause of viral encephalitis is herpes simplex virus type 1 making this the preferred answer.

West Nile virus is incorrect. Although this virus remains within the differential diagnosis given the travel history, it is a rare infection in travellers, and thus a less likely diagnosis. However, a consult with infectious diseases and appropriate testing should be conducted in order to exclude it.

Question 87 of 299

A 22-year-old man attends with a 3-month history of low mood. He also states that he has reduced energy levels and does not get enjoyment from things that he usually would. On mental state examination, he has no evidence of delusions or thoughts of self-harm. He is started on sertraline.

When should the patient be reviewed?

A. 1 week
B. 2 weeks
C. 3 months
D. 4 weeks
E. 6 months

A. 1 week

Patients ≤ 25 years who have been started on an SSRI should be reviewed after 1 week

1 week is correct. Patients under the age of 25 years or who are at increased risk of suicide (e.g. psychosis, previous self-harm) should be reviewed after 1-week following the initiation of antidepressant therapy. This is because some evidence suggests that selective serotonin reuptake inhibitors (SSRIs) may cause worsening of suicidal ideas in vulnerable patients, particularly in the very early stages of treatment..

Question 88 of 299

A 54-year-old woman presents to the Emergency Department with a five hour history of headache and visual disturbance. She states the headache came on quickly and she has vomited ten times since this pain started. She denies any past medical history and states she wears glasses for reading.

On examination there is a mid-dilated pupil in the left eye. The right eye direct pupillary reaction was satisfactory. There is no tenderness over the left temporal artery and there is no other neurological deficit.

Her observations revealed a blood pressure of 141/90 mmHg, heart rate 90 bpm, temperature 36.4ºC, oxygen saturation 96% on room air and respiratory rate 12/min.

Her blood results are as follows:

Hb	140 g/L	Male: (135-180) Female: (115 - 160)
Platelets	450 * 10 /L	(150 - 400)
WBC	11.0 * 10 /L	(4.0 - 11.0)
Na	135 mmol/L	(135 - 145)
K	4.6 mmol/L	(3.5 - 5.0)
Urea	5 mmol/L	(2.0 - 7.0)
Creatinine	80 µmol/L	(55 - 120)
CRP	8 mg/L	(< 5)

Topical pilocarpine was the applied to the eye.

What other medication should be given urgently to help manage this condition?

A. Intravenous acetazolamide
B. Topical cyclopentolate
C. Oral nimodipine
D. Topical tropicamide
E. Oral beta blocker

A. Intravenous acetazolamide

Alongside eye drops, IV acetazolamide is used in the initial emergency medical management of acute angle-closure glaucoma

The answer here is acetazolamide. This patient presents with acute angle-closure glaucoma. The acute onset headache, mid-dilated pupil with no other neurological deficit makes acute angle closure glaucoma the most likely pathology. After administering eye drops such as timolol and pilocarpine the optimal intravenous treatment is acetazolamide; a carbonic anhydrase inhibitor which decreases the level of aqueous humour in the eye.

Cyclopentolate is and acetylcholine receptor antagonist antagonist which allows mydriasis, this would cause further closure of the iridocorneal angle and therefore worsening this patients symptoms.

Nimodipine is a calcium channel blocker used in the treatment of subarachnoid haemorrhage to combat the effects of cerebral vasospasm.

Tropicamide is a dilating eye drop which is used may reduced the iridocorneal angle and therefore drainage of aqueous humour, potentially worsening the acute angle closure glaucoma.

Pilocarpine and timolol are used in the treatment of acute angle-closure glaucoma but are used as eye drops rather than intravenously. Pilocarpine causes miosis, thereby increasing the rate at which aqueous humour leaves the eye and decreasing intraocular pressure. The exact mechanism of timolol is not known but it is thought that through inhibition of beta receptors, the production of aqueous humour is reduced. Timolol is a beta blocker but is used topically, there is no known role for oral beta blockers in acute angle closure glaucoma.

Question 89 of 299

A 27-year-old lady was admitted to the Emergency Department due to acute shortness of breath. She had been completely normal that morning. Her sister mentioned that this was not the first time she had suffered such an attack and she improved every time after staying in hospital for a couple of hours and taking some nebulisers. The patient is not known to have any medical condition. Her father is on long term prednisone tablets and nebulisers for chronic obstructive pulmonary disease.

On examination: blood pressure 123/86 mmHg, pulse rate 110/min, respiratory rate 35/min. Oxygen saturation 98%. The chest was clear with good air entry.

Blood investigations showed:

Hb	128 g/l
Platelets	320 * 10 /l
WBC	8 * 10 /l
Na	137 mmol/l
K	4.2 mmol/l
Urea	6 mmol/l
Creatinine	100 µmol/l

A 3-month-old pulmonary function test was normal.

What is the next step in management?

A. Discharge home
B. Refer to repeat pulmonary function tests
C. Nijmegen questionnaire
D. Serial ECG
E. Psychiatric review

C. Nijmegen questionnaire

Normal patient observations and examination ruled out any significant pathology. The history of repeated admissions without a diagnosis and rapid recovery are all pointers towards hyperventilation syndrome (HVS). If a doctor encounters such presentation, the Nijmegen questionnaire can be used to test whether or not the patient has HVS. This questionnaire involves asking about 16 different symptoms such as chest pain and tingling fingers. Each one of these symptoms should be assigned a number from 0 to 4 according to how often it is felt. A score of more than 23 out of 64 is diagnostic of HVS.

Question 90 of 299

A 62-year-old woman presents with a blurring of the vision in her left eye and associated pain. She has not experienced this before but does recall that, 5 years prior, she experienced reduced sensation of the left leg, which eventually completely recovered spontaneously.

On examination, her vision is markedly worse in the left eye, with worse colour discrimination also. She has pain on eye movement in all planes. Neurological examination of the limbs is unremarkable.

Which of the following is associated with a poor prognosis in this patient's most likely diagnosis?

A. Female sex
B. Long interval between relapses
C. Older age of onset
D. Relapsing-remitting course
E. Sensory symptoms only

C. Older age of onset

Older age of onset is associated with a poor prognosis in multiple sclerosis

The diagnosis here is likely to be multiple sclerosis, given the two distinct episodes over time. The first episode presented with weakness of the left leg some years ago, recovered fully, and the recent episode is indicative of optic neuritis. It is worth remembering that the 'typical' patient carries a good prognosis - of the feature of this patient her age on diagnosis is atypical; older age is associated with a poorer prognosis. Usually, patients would present in their 20s or 30s.

Female sex is associated with a good prognosis in multiple sclerosis.

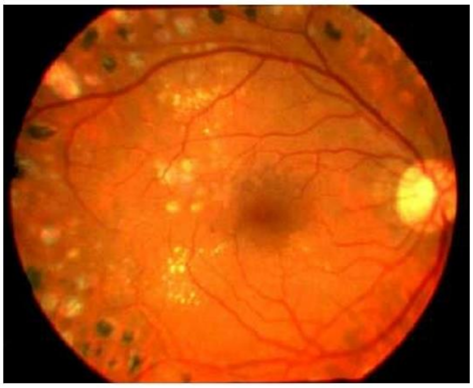
A 5-year interval between episodes, as seen here, is a significant amount of time between the first two episodes and is considered a marker of good prognosis, not poor.

This patient also demonstrates a relapsing-remitting course - complete recovery between episodes. This is also indicative of a good prognosis, not poor.

The presence of only symptoms, as opposed to motor symptoms too, is also considered to be a sign of good prognosis, not poor.

Question 91 of 299

A 64-year-old man presents with visual loss. He is on holiday in the United Kingdom; his English is limited and obtaining a history is difficult. He appears generally well in himself and does not appear to be in any obvious pain. Retinal photography is undertaken and shown below:



Which of the following is the most likely diagnosis?

A. Age-related macular degeneration
B. Central retinal artery occlusion
C. Central retinal vein occlusion
D. Diabetic retinopathy
E. Anterior ischaemic optic neuropathy

D. Diabetic retinopathy

The correct answer is diabetic retinopathy. This photo is classic of the condition. Microaneurysms are visible, as well as some central macular oedema. A number of exudative areas - 'cotton-wool spots' - are also visible. As the condition progresses, more neovascularization is expected.

Age-related macular degeneration is incorrect. The 'dry' form would characteristically appear with deposits of drusen around the macula, rather than the vascular changes seen here. The 'wet' form will have some vascular changes, but this will appear as choroidal neovascularisation rather thanthe various microaneurysms seen here.

Central retinal artery occlusion is not consistent with the photo above - it would typically show a cherry red spot, where the centre of the macula is red, but surrounding retina is pale due to lack of blood flow. The retina in general may appear pale.

Central retinal vein occlusion would present with widespread flame haemorrhages rather than the smaller changes seen here, as well as dilated and tortuous veins, which are not really seen in this photo.

Ischaemic optic neuropathy would also look different to the photo above, presenting with a pale, oedematous optic disc. The vascular changes and exudates seen here would not be present.

Question 92 of 299

A 45-year-old teacher is reviewed in a routine nephrology clinic. She has end stage renal failure with focal segmental glomerular sclerosis (FSGS) on biopsy and is receiving renal replacement via haemodialysis. She requires hearing aids for bilateral sensorineural hearing loss and is of short stature. She mentions that she has been feeling more tired and lethargic in the last few months. A fasting blood sugar taken by her GP is 8.0mmol/l. She has a family history of diabetes in her mother and maternal grandmother, both of whom also required hearing aids at an early age.

Given the above features, what is the most likely cause of her end stage renal failure?

A. Autosomal dominant polycystic kidney disease
B. Alport syndrome
C. Liddle syndrome
D. Maternally inherited diabetes and deafness (MIDD)
E. Granulomatosis with polyangiitis (Wegener granulomatosis)

D. Maternally inherited diabetes and deafness (MIDD)

Alport syndrome and GPA could also cause end stage renal failure and deafness. However, diabetes is not a feature of Alport syndrome and GPA is not a directly heritable disease.

ADPKD causes end stage renal failure with an autosomal dominant pattern of inheritance but would not explain the other features.

Liddle syndrome is a genetic cause of hypertension due to mutations in the epithelial sodium channel in the distal convoluted tubule.

Question 93 of 299

A 55-year-old female is referred to the neurology clinic with fatigue and muscle weakness, particularly affecting the lower limbs causing difficulty walking. The patient has had several similar episodes in the past with one such episode associated with painful, reduced visual acuity. Previous episodes would typically last a few weeks before spontaneously resolving.

On examination today the patient has spasticity weakness of the lower limbs with reduced sensation distally. Cranial nerve and spinal examination are both normal.

An MRI brain is performed showing evidence of disseminated brain lesions and further tests confirm unpaired oligoclonal bands in the CSF, but not in the serum.

Which of the following is associated with a poor prognosis in the patient's most likely diagnosis?

A. Female sex
B. Long interval between first two relapses
C. Older age of onset
D. Presence of oligoclonal bands in the CSF at time of diagnosis
E. Relapsing-remitting disease

C. Older age of onset

Older age of onset is associated with a poor prognosis in multiple sclerosis

This patient meets the definition for multiple sclerosis (MS) with the dissemination of lesions of the CNS, brain and/or spinal cord (motor and sensory disruption, optic neuritis, etc.) in time and space with characteristic findings on MRI and of unpaired oligoclonal bands in the CSF. Older age onset is associated with a poorer prognosis in MS when compared to patients who present at a younger age.

Females generally have a better prognosis in MS, this patient's likely diagnosis, when compared to male patients.

Longer intervals between a patient's first two relapses are associated with an improved prognosis in MS.The presence of CSF oligoclonal bands at the time of MS diagnosis has not been linked to the condition's progression or prognosis.

Of the four MS subtypes/patterns, relapsing-remitting is associated with the best prognosis (other subtypes: secondary progressive disease, primary progressive disease, progressive-relapsing disease).

Question 94 of 299

An eighty-year-old lady is reviewed. She has a complex past medical history of chronic kidney disease, hypertension, type II diabetes, osteoporosis, Paget's disease and epilepsy, taking ramipril, atorvastatin, sodium bicarbonate, linagliptin, zoledronic acid and phenytoin. On examination, she has asymptomatic cervical lymphadenopathy, with no other abnormal findings.

Which medication could be causing this?

A. Phenytoin
B. Sodium bicarbonate
C. Atorvastatin
D. Linagliptin
E. Zoledronic acid

A. Phenytoin

Phenytoin can cause lymphadenopathy

Phenytoin is known to be an iatrogenic cause of lymphadenopathy, as well as carbamazepine, allopurinol and sulphonamides. Be aware, that a more systemic drug reaction is more often noted with medications, with lymphadenopathy as an associated, but not isolated, sign e.g. DRESS and serum sickness.

Sodium bicarbonate, atorvastatin, linagliptin and zoledronic acid are not commonly associated with lymphadenopathy.

Question 95 of 299

A 62-year-old man is reviewed following a thyroidectomy for follicular thyroid cancer without metastatic spread. He started developing first twitching and then cramps following the procedure, primarily affecting his legs. He finds this to be quite painful and has never experienced anything like this previously. He reports that the surgical registrar told him that the procedure went well without complication. He also has a past medical history of hypertension, vitamin D deficiency, depression and gout. He was taking ramipril, cholecalciferol with calcium and allopurinol. All his regular medication have been held for one day due to surgery.

What is the most likely cause of his cramps?

A. Hypocalcaemia
B. Hypercalcaemia
C. Hypophosphataemia
D. Hyperphosphataemia
E. Hypokalaemia

A. Hypocalcaemia

Following thyroid removal, checking calcium is important to assess for hypoparathyroidism

The patient has undergone an thyroidectomy and developed symptoms consistent with hypocalcaemia soon after. This is most likely due to iatrogenic primary hypoparathyroidism as the parathyroid glands can easily be removed or have vascular damage from thyroid surgery. The patient would likely have low parathyroid levels, as well as low calcium and high phosphate. The low calcium would need to be replaced and then symptoms would resolve.

Question 96 of 299

An 82-year-old female is brought into your falls clinic by her daughter after her third fall this year. No fractures were sustained and she appears to have no significant head injuries. The fall appears mechanical in nature. She currently lives with her daughter, who reports the patient's mobility to be progressively deteriorating, from full independence and no exercise limitations 1 year ago to restrictions at 50-70 yards now, limited by knee pain secondary to osteoarthritis. Her other past medical history includes hypertension, type 2 diabetes mellitus, chronic kidney disease and previous gallstones.

You note she is withdrawn and makes little eye contact. Her voice is quiet. When you ask her whether she is low in mood, she does not respond. She reports no suicidal ideations but has little hope for the future. She asks you to 'not worry about it', as she 'has been the same way for several months now'. However, the patient does seem amenable to some kind of treatment.

On the Beck depression scale, she scores 11/63 (0-13 = no or minimal depression), on the geriatric depression scale, she scores 11/15 (greater than 10 = indicative of depression) and mini-mental state examination, she scores 19/30 (20-26 = mild cognitive impairment, 10-19 = moderate cognitive impairment). Routine investigations including B12, folate, thyroid function, liver function tests and bone profile are unremarkable.

What is the most appropriate treatment pathway?

A. Citalopram
B. Amitriptyline
C. Mirtazapine
D. Donepezil
E. No treatment required

A. Citalopram

Depression in elderly can depress cognitive function, hence cognition may be inaccurately depressed on measurement scales. Donepezil is thus not indicated on this measure alone. In elderly patients, GDS is more appropriate than Becks depression scale, as

the latter focuses heavily on somatic symptoms that frequently under-score depression in elderly patients. This patient is describing depression without suicidal ideations. Tricyclic antidepressants and SSRIs are similarly efficacious in elderly patients but the first line of treatment for depression in the elderly is a SSRI such as citalopram, mainly due to the lack of interactions with primarily P450 enzymes . TCAs produce significantly greater anticholinergic side effects. MIrtazapine and sertraline failed to demonstrate a significant benefit compared to placebo in Alzheimers patients with depression

Question 97 of 299

A 46-year-old white Caucasian female presents to the Emergency Department after waking up unable to walk and complains about problems with vision in her right eye. The patient says she is normally extremely healthy and the only time she has ever visited her GP was when she had an episode of diarrhoea and vomiting about a week ago. She is understandably distressed as she has not experienced any symptoms before and has not been previously diagnosed with any medical conditions. She has no drug or family history. Nursing staff report to you that she has been given a pad on her bed as she is incontinent of urine, which she is tearfully embarrassed by.

On examination of her cranial nerves, a right relative afferent papillary defect is noted. Visual acuity and colour vision are 6/6 with 17/17 Ishihara plates on the left, 6/60 with 0/17 Ishihara plates on the right. She reports no diplopia with a full range of eye movements and normal facial sensation. Facial movements are normal. Fundoscopy was unremarkable. Examination of the patient's upper and lower limb power demonstrated normal tone with 2-/5 power on the left arm and leg in all movements; and 4-/5 all movements in right arm and leg. There is a patchy loss of sensation to cotton wool on right lateral wrist and anterior aspect left lateral shin. Both plantars are upgoing. Reflexes are brisk at knee and ankles bilaterally. Anal tone and saddle sensation are intact. The patient's cognitive state is normal on superficial examination.

An urgent MRI head and whole spine demonstrates abnormal high signal in the cervical cord from C3 to C7. Her blood tests were mislabelled and hence unavailable. A lumbar puncture was performed with the following results:

WCC	12 mm³
RBC	<1 mm³
Protein	0.9 g/l
Glucose	5.2 mmol/l (10.2 mmol/l serum)
Oligoclonal bands	awaited
Viral PCR	awaited

What is the most likely diagnosis?

A. Idiopathic intracranial hypertension
B. Transverse myelitis
C. Miller-Fisher syndrome
D. Multiple sclerosis
E. Devic's disease

E. Devic's disease

A first episode of an optic neuritis and myelitis occurring together is suggestive of a central neuroinflammatory cause. A full range of eye movements makes Miller-Fisher syndrome unlikely (and any ataxia demonstrated is likely secondary to limb weakness). Although there is the temptation of diagnosing multiple sclerosis in a patient with features disseminated in place, the keys to the answer are the sites of the lesion (optic nerve and cervical cord) and the long length of cord involvement beyond 3 vertebral levels, much more suggestive of neuromyelitis optic or Devic's disease. The diagnosis would be clinched by a positive aquaporin 4 antibody.

Question 98 of 299

A 42-year-old woman presents with 6 days of drowsiness and a gradual onset headache, initially starting in the occipital region and radiating to the apex. She has recently returned from Australia in holiday one week ago but has complained of poor appetite after a cough and cold since landing. She has no past medical history, takes no regular medications except the oral contraceptive pill. She is a non-smoker and drinks minimally. On examination, the patient has a full range of neck movements and no photophobia. Examination of her limbs is unremarkable. You request a CT head without contrast, which demonstrates two small areas of subarachnoid blood in right convexity. She denies any recent head trauma.

Which investigation will most likely provide the conclusive diagnosis?

A. CT head with contrast
B. MRI head
C. MR venogram
D. Lumbar puncture with xanthochromia
E. CT angiography

C. MR venogram

MR Venogram is the gold standard test for diagnosing venous sinus thrombosis

The patient presents with a non-specific headache on a background of a number of different risk factors for venous thromboembolic disease: recent long-haul flight, likely dehydration from poor oral intake, oral contraceptive pill. A generalised headache is present in almost 90% of all patients with cerebral venous thrombosis (CVT) . The headache is caused by impaired absorption of cerebrospinal fluid due to the sinus thrombus, resulting in increased intracranial pressure. CVTs can also present with seizures, encephalopathy and focal symptoms, but be aware that the presenting clinical features can be extremely variable.

A plain CT head can sometimes demonstrate signs of CVT in one-third of patients. A dense triangle sign is often seen in the posterior superior sagittal sinus. In addition, the empty or negative delta sign due to thrombus obstruction. However, MR venogram (CT venogram in the hands of highly skilled radiologists) is the most sensitive imaging modality.

This scenario is a useful demonstration that not all subarachnoid blood equates to a spontaneously ruptured aneurysm. Cerebral venous thrombosis is a classic cause of intraparenchymal and subarachnoid haemorrhages, particularly if around the convexity . In addition, the description of the headache is atypical for an aneurysmal subarachnoid haemorrhage: the headache is of gradual onset, persists for days, with no meningism. Although it may seem illogical, treatment with anticoagulation will treat the sinus thrombus, resulting in improved CSF flow, while the subarachnoid blood will slowly resolve.

Question 99 of 299

A 57-year-old gentleman was brought to the emergency department by ambulance following a collapse 2 hours earlier. His partner witnessed him fall to the floor and stated that he had not been moving his right arm and leg since. She said that he had been well before this episode. The ambulance crew reported that he had had a generalised tonic-clonic seizure, which was terminated after 6 minutes with 5mg of diazepam. On examination, he was drowsy, with a Glasgow coma score of 10 out of 15, and his blood pressure was 165/92mmHg. He had a right sided facial droop and there was no movement in his right arm or leg. He was known to be a smoker, and he took amlodipine for hypertension. He was not on any other medications.

An urgent CT head scan was performed immediately which demonstrated loss of differentiation between the grey and white matter in the left frontal and parietal lobes, but no acute haemorrhage.

What is the most appropriate initial management?

A. Aspirin 300mg for 2 weeks
B. Intravenous alteplase 900 micrograms/kg
C. Aspirin + dipyridamole 25/200mg
D. Clopidogrel 75mg
E. Warfarin

A. Aspirin 300mg for 2 weeks

This patient presents with a sudden onset of neurological deficit, with lateralising signs. This history is strongly suggestive of a stroke, and the CT head findings suggest an ischaemic cause.

Acute management of ischaemic stroke involves keeping the patient nil by mouth until swallow assessment is performed, nasogastric tube insertion if dysphagia is problematic, and admission to a high dependency stroke unit for close monitoring.

If the presentation is within 4.5 hours of onset thrombolysis with a recombinant tissue plasminogen-activator (such as alteplase) can be considered, which has been shown to improve neurological outcome. This patient presents at 2 hours, but his seizure at the onset of stroke precludes the use of thrombolytic therapy (see table).If the presentation is beyond 4.5 hours, or thrombolysis is contraindicated, aspirin should be commenced at a dose of 300mg for 2 weeks as soon as possible. Lifelong anti-platelet therapy should be continued thereafter. Aspirin in combination with dipyridamole is only recommended if clopidogrel is contraindicated. Warfarin is not indicated in the immediate management of stroke, though may be started if the patient is shown to have atrial fibrillation. This is usually delayed for 2 weeks, due to the risk of haemorrhagic transformation if given before this.

Question 100 of 299

A 45-year-old woman has been brought to the Emergency Department by ambulance. Her 20year-old son found her collapsed on the floor with a note stating that she wished to take her own life. The paramedics have brought all the medication packages that they could find. This included sertraline, atorvastatin and diazepam. Previous medical history includes depression with suicidal ideation and a paracetamol overdose 3 years ago that required admission.

Her 50mg sertraline package shows five empty slots. Her 10mg diazepam package shows 30 empty slots.

On assessment of the patient:

- A - Airway is supported by the anaesthetist with a bag valve mask.
- B - Respiratory rate is being supported up to 12/min. Saturations are maintained at 98% however, the patient desaturates and hyperventilates without assistance.
- C - BP 102/74 mmHg, pulse 94 bpm.
- D - GCS 9, (E1, M5, V3), pupils 4mm bilaterally and reactive.

What is the next step in the management of this patient?

A. Activated charcoal
B. Flumazenil
C. High dose insulin therapy
D. Intubation and ventilation
E. Naloxone infusion

D. Intubation and ventilation

Benzodiazepine overdose is best managed supportively and with airway protection and ventilation if needed. Flumazenil should be avoided unless for reversal of anaesthesia

Intubation and ventilation is correct. The patient is hypoventilating and unable to protect their airway. The cause is likely an overdose of benzodiazepines which requires organ support whilst the drug is metabolised. Further investigations such as CT head, ECG, paracetamol and salicylate levels will also be important to ensure there is no alternative cause for the sudden drop in GCS.Activated charcoal is incorrect because the patient has a low GCS. Although activated charcoal can be used in overdose if the patient presents within 60 minutes of ingestion, it should not be administered to a patient with a decreased GCS. Charcoal is an emetic and increases the risk of aspiration in a patient with a low GCS. In this case, the time of consumption of medication is also unknown.

Flumazenil is incorrect because it is not known if this is an isolated benzodiazepine overdose and the patient may have a dependence on benzodiazepines. Flumazenil is rarely used outside of the operating theatre. It is the antidote to benzodiazepines but must not be used in mixed overdoses. If the patient has any risk of seizures then administration of flumazenil will prevent benzodiazepines from providing seizure control. In this case, the patient has likely ingested a considerable dose of benzodiazepines but may have taken a mixed overdose.

High dose insulin therapy is incorrect because this is used in beta-blocker and calcium channel blocker overdose. High dose insulin therapy is the administration of insulin at 1unit/kg. This therapy may overcome myocardial starvation and increase myocardial function without an increase in oxygen demand.

Naloxone infusion is incorrect because there is no suspicion of an opioid overdose. Naloxone is the antidote to opioid toxicity. Although it is entirely appropriate to trial naloxone in suspected mixed overdose and suspicion of opioids, a naloxone infusion should only be started if the patient has shown a response to an initial naloxone bolus. The initial dose is 400mcg, however experienced clinicians may titrate this to affect in chronic opioid users as this can lead to sudden and severe withdrawal symptoms.

Question 101 of 299

A 72-year-old man is referred to the neurology outpatient department with an insidious onset of tremor by his general practitioner. He has a past medical history of hypertension and recurrent falls. He is on regular amlodipine. He does not smoke or drink alcohol.

On examination, there is bradykinesia and rigidity and a pill-rolling tremor of the right hand. He is unable to make vertical saccades with his eyes when tested. He appears to lung forward when walking.

What is the likely diagnosis?

A. Corticobasal degeneration
B. Dementia with Lewy Bodies
C. Multiple system atrophy
D. Parkinson's disease
E. Progressive supranuclear palsy

E. Progressive supranuclear palsy

Progressive supranuclear palsy: postural instability, impairment of vertical gaze, parkinsonism, frontal lobe dysfunction

Progressive supranuclear palsy is the correct answer. This patient presents with parkinsonism, postural instability and impairment of vertical gaze, which is characteristic of this condition.

Dementia with Lewy Bodies is incorrect. While this can cause parkinsonism, it does not cause gaze palsies and dementia is a prominent feature.

Multiple system atrophy is incorrect. This is another 'parkinson-plus' syndrome but is characterized by autonomic dysfunction and postural instability rather than gaze palsies.

Corticobasal degeneration is incorrect. This is characterized by parkinsonism, apraxia, aphasia and classically 'alien hand syndrome', which results in the failure of the individual to control the movements of their limb resulting from the sensation that the limb is 'foreign'.

Question 102 of 299

A 65-year-old gentleman presents with left sided arm pain that originates in the shoulder and spreads into the ring and little fingers. This has worsened over the past three weeks to the point that he cannot sleep at night. His GP has trialled co-codamol without any benefit.

He is a previous smoker of 40 pack years, has hypertension and a myocardial infarction 5 years previously. He is currently well but on further questioning reveals that he has lost 2 stone in weight over the last 3 months despite increasing his food intake. He has a productive cough which has occasional streaks of blood in.

On examination, he is able to move his left shoulder, elbow and wrist with a full range of motion and no tenderness but reports that the pain is present whilst not moving. He has some weakness of his left biceps with a diminished reflex and sensation is reduced to the fourth and fifth in response to light touch. The limb has normal pulses and capillary refill time with no swelling. There are crepitations on the left upper zone and he has clubbing present.

Hb	103 g/l	Na	137 mmol/l
Platelets	456 * 10 /l	K	4.2 mmol/l
WBC	13.1 * 10 /l	Urea	5.6 mmol/l
Neuts	9.8 * 10 /l	Creatinine	98 µmol/l
Lymphs	0.89 * 10 /l	CRP	89 mg/l
Eosin	0.25 * 10 /l		

Chest x-ray	consolidation seen in the left upper zon

What is the likely diagnosis?

A. Bronchial carcinoma
B. Pneumonia
C. Cervical rib
D. Multiple sclerosis
E. Tuberculosis

A. Bronchial carcinoma

Lung carcinoma can present with a brachial neuritis

This gentleman presents with a neuralgia pain in the left arm and altered neurology in the dermatome of C8. Of these, pneumonia is unlikely to ever cause this. He does have respiratory symptoms and an extensive smoking history and this excludes multiple sclerosis as there are no other areas of altered neurology. The diminished reflexes suggest a lower motor neuron lesion and C8 is classically affected in a Pancoast tumour. Tuberculosis can rarely infiltrate the brachial plexus but bronchial carcinoma is the most likely. A cervical rib is unlikely to present at this age.

Question 103 of 299

A 46-year-old lady is admitted to emergency department with a 2 hour history of dysphasia, visual disturbance and weakness. Examination reveals left homonymous hemianopia, left sided hemiparesis with sensory loss and dysphasia. Observations show she is apyrexial with a heart rate of 102/min and blood pressure of 145/82 mmHg.

An urgent CT head and angiography are booked indicating a large infarct in the territory of the middle cerebral artery in the right cerebrum, while the CT angiography is indicative of proximal occlusion.

There is no significant past medical history and the blood investigations for this lady are normal. Her modified Rankin score is 1 and her national institutes of health stroke scale (NIHSS) is 12.

What is the next most appropriate course of action in her management given that we are now four hours from presentation?

A. Offer intravenous thrombolysis alone
B. Reduce blood pressure via the administration of nifedipine
C. Offer thrombectomy with intravenous thrombolysis cover
D. Load with clopidogrel
E. Treat conservatively with aspirin and secondary prevention

C. Offer thrombectomy with intravenous thrombolysis cover

A combination of thrombolysis AND thrombectomy is recommend for patients with an acute ischaemic stroke who present within 4.5 hours

This question highlights the importance of understanding the timing scales applicable to stroke management. The time limit for offering thrombolysis is 4.5 hours currently, while the thrombectomy window (provided that certain anatomical and functional criteria are met) is that of 6 hours.

This young lady fulfils the criteria for thrombectomy and thrombolysis and it is important to note that thrombolysis is not contra-indicated but actually encouraged en-route to thrombectomy.There is no indication in reducing this lady's blood pressure as it is well below 180/110 mmHg.

There is no place for clopidogrel loading in the acute management of stroke, with the exception of aspirin allergy.

As this lady fulfils all the criteria, has a reasonable pre-stroke score (modified Rankin score <3) and a high stroke score indicating the impact of the occlusion to her functioning (NIHSS >5), thrombectomy with thrombolysis cover is the most appropriate course of action.

Question 104 of 299

A 32-year-old male patient presents with a 1-day history of painful rash on the forehead. The patient does not have any past medical history of note. On examination, there is a vesicular rash in the distribution of the ophthalmic nerve on the right side. There is no pus or discharge. There is currently no ocular involvement.

What is the appropriate treatment?

A. Topical steroid cream for 7-10 days
B. Topical acyclovir for 7-10 days
C. Oral acyclovir for 7-10 days
D. Oral steroid for 7-10 days
E. Topical mupirocin for 7-10 days

C. Oral acyclovir for 7-10 days

Topical antiviral treatment is not given in herpes zoster ophthalmicus

This patient has herpes zoster ophthalmicus. First line treatment is oral acyclovir, started as soon as possible, for 7-10 days. Topical antiviral treatment is not given in herpes zoster ophthalmicus. This patient has no ocular involvement so far, however, if there is any sign of ocular involvement, he should be referred to ophthalmology urgently.

Steroid can be given concurrently but it does not reduce the risk of post-herpetic neuralgia. Topical mupirocin is not a suitable treatment.

Question 105 of 299

A 28-year-old man is admitted to the respiratory ward with sudden onset chest pain and shortness of breath. He has no past medical history of note. On examination, there is reduced air entry in the left lung that is hyper-resonant to percussion. The likely diagnosis is confirmed on a chest x-ray and he is treated with a pleural aspiration and observed on the ward for 24 hours.

During this period of observation, he remains well with normal observations and resolution of his chest pain and shortness of breath. A repeat chest x-ray is taken that appears normal and he is medically fit for discharge.

The patient mentions that he is due to fly to Brazil for a holiday in 2 days and wants to know if this will be safe.

What is the most appropriate advice to give this patient?

A. Able to fly following pleurodesis
B. Able to fly immediately
C. Able to fly in 1 week
D. Able to fly in 6 weeks
E. Unable to fly for life

A. Able to fly in 1 week

Pneumothorax -> can fly 1 week after treatment if chest x-ray shows resolution

This patient's clinical history, examination and investigation findings are in keeping with a pneumothorax. Given that this patient is young with no other past medical history, it is likely that he has had a spontaneous, primary pneumothorax which, if large enough, should be treated with needle aspiration. Current guidance from the British thoracic society states that following complete resolution of the pneumothorax, patients are able to fly 1 week after treatment. If a pneumothorax arises following a traumatic injury, then flying 2 weeks after chest x-ray confirmation of resolution is advised.

A pleurodesis is usually required for recurrent pneumothoraces and can be either chemical or surgical. Given that this patient's chest x-ray shows resolution of the pneumothorax, pleurodesis isnot necessary and would not be an indication for this patient to travel by air.

It is too soon for the patient to fly immediately as guidelines state that patients not air travel until 1 week from the complete resolution of pneumothorax on chest x-ray.

Previous guidelines advised that air travel would be safe following 6 weeks from the resolution of pneumothorax. However, this has since been changed to 1 week.

Unable to fly for life is incorrect. Following a pneumothorax, guidance is given to avoid deep-sea diving for life. However, air travel is still possible following pneumothorax resolution.

Question 106 of 299

A 68-year-old man presents to the acute medical unit with an episode of loss of consciousness. He states that he suffered a headache earlier that day. He describes it as a sudden onset headache at the back of his head which reached 10/10 severity within a few seconds.

You organise the following investigations:

CT head Blood in the sulci, fissures, basal cisterns and ventricles

Cerebral CT angiogram Evidence of ruptured posterior cerebral artery aneurysm

What is the most appropriate initial management?

A. Triple H therapy (hypertension, hypervolaemia, and haemodilution)
B. Labetalol
C. Magnesium sulphate
D. Amlodipine
E. Nimodipine

E. Nimodipine

Nimodipine is used to prevent vasospasm in aneurysmal subarachnoid haemorrhages

The patient has suffered from a subarachnoid haemorrhage (SAH) as confirmed on CT brain. A CT angiography has confirmed that it is aneurysmal. He is now at significant risk of re-bleeding and vasospasm.

To prevent re-bleeding he needs urgent endovascular clipping or coiling.

The gold standard for preventing vasospasm is nimodipine. Alternative calcium channel blockers can be used but the strongest evidence favours nimodipine.

The combination of induced hypertension, hypervolaemia, and haemodilution (triple H therapy) isoften used to prevent and treat cerebral vasospasm after aneurysmal SAH, however the evidence for this approach is inconclusive.

Question 107 of 299

The emergency department registrar asks for your advice regarding a 67 year-old female patient who presented with an acutely painful right eye. On examination the right pupil is fixed in middilation and not responsive to light. The conjunctiva is injected. The patient is clearly in considerable pain and has vomited twice in the department. The emergency department physicians have given intravenous analgesia and anti-emetics. You refer urgently to ophthalmology.

What is appropriate initial treatment?

A. Topical pilocarpine
B. Urgent iridotomy
C. IV acetazolamide
D. Topical latanoprost
E. Topical brimonidine

C. IV acetazolamide

The diagnosis here is acute angle closure glaucoma, an ophthalmological emergency. The correct urgent management is as follows:

- IV analgesia and antiemetics, lie patient supine.
- Topical beta blockers and steroids.
- IV acetazolamide.
- After initial treatment, pilocarpine will induce meiosis and open the angle. This is initially ineffective due to pressure induced ischaemic paralysis of the iris.
- Iridotomy 24-48 hours after intra-ocular pressure is controlled to prevent recurrence.

Question 108 of 299

A 20-year-old female with multiple sclerosis (MS) attends the emergency department following a seizure. She was diagnosed with MS at age 16 and has received different immunomodulatory regimes in the past, including azathioprine. She commenced natalizumab 2 years ago. This is her first seizure however once recovered, she tells you that she has noticed she has been increasingly clumsy over the last 6 months, she drops things easily. In addition, she has had several episodes where her speech has been slurred and people have commented that she sounds tired. On examination, you find that her tone is normal throughout, power in all muscles groups in both upper and lower limbs is normal. On cranial nerve examination, you identify a right homonymous superior quadrantanopia however she does have a full range of eye movements and no ptosis. She has an ataxic gait. Her speech is normal throughout the history.

She is concerned, what is the diagnosis?

A. Progressive multifocal leukoencephalopathy
B. Transient ischaemic attacks
C. Progression of multiple sclerosis
D. Newly diagnosed epilepsy
E. Myasthenia gravis

A. Progressive multifocal leukoencephalopathy

Natalizumab is a humanised monoclonal antibody against the cell adhesion molecule 4-integrin and is licensed as a monotherapy for the treatment of multiple sclerosis in Europe.

Progressive multifocal leukoencephalopathy (PML) is an opportunistic infection caused by the JC virus that only occurs in patients who are immunocompromised and has a risk of 2.1 in every 1,000 using natalizumab. Patients who have taken immunosuppressants before the initiation of natalizumab therapy have the highest risk of developing PML

Question 109 of 299

A 22-year-old, recently married woman presents to the first seizure clinic and is diagnosed with idiopathic generalised epilepsy. She tells you that she is keen to start a family soon.

Which first line anti epileptic drug should be avoided?

A. Levetiracetam
B. Topiramate
C. Lamotrigine
D. Carbamazepine
E. Valproate

E. Valproate

All antiepileptic drugs are associated with a risk of major congenital malformations in the offspring. However, the potential for the mother to develop tonic clonic seizures, which can result in significant adverse health outcomes for the fetus, means that discontinuing antiepileptic drug therapy during pregnancy is still discouraged by most practitioners.The largest randomised trials of newer generation versus older generation antiepileptic drugs (SANAD trials) found that valproate is superior to both lamotrigine and topiramate for the treatment of idiopathic generalised epilepsy. However, valproate is associated with teratogenicity and a risk of low IQ and should therefore be avoided in women of childbearing potential. The risk of teratogenecitiy for the newer generation antiepileptics drugs remains largely unknown, due to small sample sizes and polytherapy exposures. The data for lamotrigine is limited but is believed to be less teratogenic than valproate.

Carbamazepine is associated with a higher risk of developing major congenital malformations. For Levetiracetam, although pregnancy exposure data is limited, the UK epilepsy and pregnancy registry have not identified any major congenital malformations in association with its use.

Question 110 of 299

A 45-year-old male patient presents with an inaugural seizure. During surgery, it is found that he has an aggressive glioma, and on further histological analysis the most aggressive form of glioma is diagnosed.

Given the aggressive nature of this tumour, which of the following is the next step in the standard of care for this tumour?

A. Chemotherapy
B. Radiotherapy
C. Chemo-radiotherapy
D. No further therapy needed
E. Biological agents

C. Chemo-radiotherapy

Glioblastoma multiforme is by far the most common and most malignant/aggressive of the glial tumours. It kills virtually every patient that is affected by it, and until a trial in 2007 was published, the standard of care was surgery and radiotherapy.

Currently, the standard of care is surgery followed by chemo-radiotherapy with temozolomide, an alkylating chemotherapy agent. The key is that the temozolomide and the radiotherapy have a synergistic effect on killing tumour cells.

Question 1 of 189

A house officer is asked to review a patient on her ward as the nurses are concerned about the patients speech. The house officer reports that the patient has unilateral weakness of her right face, arm and leg, dysphasia and a visual field defect. She reports that reflexes are brisk on the right and that the tone of the limbs is increased. There is no disdiadokokinesis on the left, but she is unable to assess on the right arm. The patient's heart rate is 65, blood pressure 198/100 mmHg and respiratory rate 14. Jugular venous pressure is 2cm above the sternal notch. Pupils react to light and are consensual. The patients family are present who report that the symptoms were sudden in onset. The family are unsure about past medical history, but a list of medications includes warfarin, bendroflumethiazide, ramipril, atorvastatin and allopurinol. A CT head reveals a cerebral infarct.

How would this stroke be classified?

A. Partial anterior circulation stroke
B. Total anterior circulation stroke
C. Posterior circulation stroke
D. Lacunar syndrome
E. Thalamic stroke

B. Total anterior circulation stroke

The correct answer is Total anterior circulation stroke. A total anterior circulation stroke (TACS) is characterised by the presence of all three features: motor or sensory deficit affecting at least two out of three areas (face, arm and leg), homonymous hemianopia, and higher cerebral dysfunction (e.g. dysphasia). In this case, the patient presents with unilateral weakness of her right face, arm and leg, dysphasia and a visual field defect which are indicative of a TACS.

A Partial anterior circulation stroke is incorrect because it does not involve all three areas mentioned above. It typically involves at least two out of the three areas but not all. The patient's presentation indicates involvement of all these areas hence suggesting a total rather than partial anterior circulation stroke.

Posterior circulation stroke is also incorrect as it usually results in symptoms such as vertigo, nausea/vomiting, nystagmus and cranial nerve palsy which are not reported in this case. Theposterior circulation supplies the brainstem, cerebellum and occipital lobes so deficits would be expected in these areas rather than the unilateral motor/sensory loss described.

Lacunar syndrome is another incorrect option. Lacunar strokes result from the occlusion of small penetrating arteries that provide blood to the brain's deep structures. They typically present with pure motor or sensory symptoms and do not include cortical signs such as dysphasia or visual field defects.

Lastly, Thalamic stroke is also an incorrect choice. Thalamic strokes can present with various symptoms including sensory loss, weakness, speech difficulties and behavioural changes depending on which part of the thalamus was affected. However, they do not generally cause widespread motor deficits as seen in this patient's presentation.

Question 2 of 189

A 65-year-old man attends neurology clinic for review of his long-standing trigeminal neuralgia. He had first experienced symptoms five years previously and been troubled by his illness ever since despite frequent neurology review. The patient experiences attacks of severe shooting pain affecting the right side of his lower face, each episode usually lasting about one hour. The interval between attacks has steadily reduced over the years so that at the present time the patient experiences four to five episodes per week. The patient describes that his symptoms are profoundly limiting his life and that he rarely leaves his house for fear of an attack.

Four years previously, treatment with carbamazepine had been introduced with an initially good response to symptoms. However, patient proved to be intolerant of carbamazepine due to drowsiness. Subsequent trials of treatment with oxcarbazepine, lamotrigine and baclofen had not given lasting relief.

The patient had recently been diagnosed with depression and initiated on treatment with sertraline. He was also suffered from type 2 diabetes currently managed with diet and metformin 500 mg TDS. The patient lived with his wife and had been unable to work as a school-teacher for the past two years due to his symptoms.

Having previously been reluctant to consider surgical intervention, the patient now felt he would be willing to try any options that could improve his symptoms.

MRI brain with / without contrast: sinuses unremarkable without evidence of inflammation; no space occupying lesion; no extra-cranial mass along course of trigeminal nerves; no evidence of widespread demyelination plaque; no evidence of previous infarction; no abnormal enhancement of the trigeminal nerves.

What is the appropriate surgical intervention for this patient?

A. Stereotactic radiosurgery
B. Balloon compression
C. Microvascular decompression
D. Radiofrequency lesioning
E. Glycerol rhizolysis

C. Microvascular decompression

The patient has long-standing and severe trigeminal neuralgia with life-limiting symptoms despite appropriate medical treatment. Surgical intervention should therefore be considered.

In around 95 % of cases, trigeminal neuralgia is believed to be caused by compression of the trigeminal nerve by adjacent blood vessels, although the exact mechanism of the pathophysiology is incompletely understood. In a minority of cases, trigeminal neuralgia can be due to an alternative diagnosis such as sinusitis, extra-cranial or intra-cranial masses, multiple sclerosis or infarction of the thalamus or brainstem.

The MRI brain undergone by the patient does not provide evidence for any of the rare causes of trigeminal neuralgia with the patient's symptoms therefore very likely due to vascular compression. Using specialist techniques not included in the standard protocol, MRI can detect vascular contact with the trigeminal nerve. However, vascular contact with cranial nerves is also a normal variant in some individuals so these techniques are not routinely used.

Microvascular decompression is therefore the surgical technique of choice and is effective treatment in 95 % of such cases of trigeminal neuralgia. The other possible answers refer to methods of palliative destruction of the trigeminal nerve root, therefore carrying the risk of causing facial numbness. However, they are effective in the minority of trigeminal neuralgia cases not caused by vascular compression.

Question 3 of 189

A 42-year-old woman attends neurology clinic for review of her treatment strategy for multiple sclerosis. The patient had first developed symptoms of multiple sclerosis five years previously. Her first episode had involved sensory loss and motor weakness affecting her left leg. An MRI brain scan conducted at this time had demonstrated multiple lesions suggestive of multiple sclerosis. At this point in time, the patient had decided against receiving disease modifying therapy.

One year after her first episode, the patient suffered an episode of optic neuritis. A repeat MRI scan had demonstrated a progression of the previously observed radiographic lesions. Subsequently, the patient had been initiated on treatment with interferon beta. This treatment was continued for two years during which time the patient suffered no further relapses of multiple sclerosis. However, the patient found the unwanted effects of interferon beta to be progressively harder to tolerate and she eventually decided against continuing with this treatment. A subsequent trial of glatiramer acetate was quickly halted due to the patient experiencing severe symptoms of flushing.

Following the cessation of disease-modifying therapy, the patient had experienced multiple further relapses, including one episode where she had required hospitalisation due to cranial nerve involvement. Further assessment during this period found that the patient met the local criteria for aggressive or highly active multiple sclerosis. Accordingly, later line therapies were discussed with the patient who was keen to proceed.

Aside from her history of multiple sclerosis the patient suffered from no other significant medical problems and took no regular medications. The patient was a science teacher that had been unable to work for the past year due to her multiple relapses.

The later line therapy recommended for the patient is natalizumab.

What is the essential investigation that must be completed prior to initiation of treatment with natalizumab?

A. Transthoracic echocardiogram
B. JCV antibody status
C. Pulmonary function tests
D. Thyroid function tests
E. CMV antibody status

B. JCV antibody status

The patient has highly active multiple sclerosis causing significant morbidity. While she had obtained benefit from the standard initial therapies interferon beta and glatiramer acetate, these treatments were ultimately not tolerated. In this situation, consideration of escalation to secondline disease-modifying therapy is appropriate. Observational studies have reported that escalation from a first-line therapy to a second-line drug reduces relapse rate at one year.

Evidence suggests that alemtuzumab, natalizumab and fingolimod outperformed other disease modifying therapies at preventing relapse at 2 years. In addition, natalizumab also reduced 2-year disability progression rates.

Natalizumab is strongly associated with progressive multifocal leukoencephalopathy, an infection of the central nervous system with the John Cunningham virus (JCV). Therefore, JCV antibody status is an essential part of assessing an individual for treatment with natalizumab. Patient's who are seropositive for JCV are not treated with natalizumab or switched to another drug.

Question 4 of 189

A 45-year-old lady presented to the emergency department with prolonged fever and malaise, She had a new murmur and clinical signs clearly indicating subacute bacterial endocarditis. ECHO confirms vegetations on her aortic valve and blood culture is still pending. She is a known case of myasthenia gravis and has been warned against taking some groups of antibiotics.

Which of the following antibiotics can worsen the symptoms of myasthenia gravis?

A. Gentamicin
B. Augmentin
C. Meropenem
D. Benzylpenicillin
E. Trimethoprim sulphamethoxazole

A. Gentamicin

Answer: Gentamicin.

Aminoglycosides are known to worsen myasthenia gravis. Other medications that have this effect include: ciprofloxacin, verapamil, beta blockers like propranolol, lithium, D-penicillamine and muscle relaxants like Botox.

Question 5 of 189

A 36-year-old female presents to clinic with transient visual loss. She reports three episodes over the last few months where her vision 'turns black' in both eyes despite being alert. This lasts for a few seconds and is then followed by a unilateral throbbing headache associated with nausea and phonophobia. It is worse on exertion and lasts for a couple of days. On examination her visual acuity is 20/20 bilaterally, her visual fields are normal and fundoscopy is unremarkable.

What is the most likely diagnosis?

A. Transient ischaemic attack
B. Amaurosis fugax
C. Anterior ischaemic optic neuropathy
D. Temporal arteritis
E. Migraine with aura

E. Migraine with aura

The headache in this patient is very typical of migraine which can be preceded by an aura lasting between 5 minutes to an hour usually. This patient's visual loss was relatively brief and in the context of unilateral vision loss would certainly raise the possibility of transient ischaemic attack (TIA) However, she reports her vision turned black in both eyes which would be very unlikely with a TIA as there would have needed to be simultaneous involvement of both anterior visual pathways. Since there is isolated bilateral transient visual loss if this was a TIA it would be of the occipital cortex from occlusion of the posterior cerebral artery although this is very unlikely given it does not appear to be a homonymous hemianopia she describes, and there is a characteristic history of migraine which is the most common cause of transient bilateral visual loss in young adults. Amaurosis fugax specifies a type of TIA, which typically causes a unilateral visual disturbance.

Temporal arteritis represents an arteritic form of anterior ischaemic optic neuropathy. Temporal arteritis tends to present later in life and may be associated with other symptoms like scalp tenderness, and jaw claudication. The non-arteritic form of anterior ischaemic optic neuropathy tends to be related to cardiovascular factors, and therefore again presents later in life and more commonly restricted to one eye.

Question 6 of 189

A 54-year-old female attends her GP with sudden onset reduced vision. This is particular to the left eye. The patient has a background of type 2 diabetes mellitus and hypertension. The GP is unable to adequately see the optic fundi on fundoscopy but thinks the left side appears pale.

What is the most likely diagnosis?

A. Retinal vein occlusion
B. Hypertensive retinopathy
C. Giant cell arteritis
D. Stroke
E. Retinal artery occlusion

E. Retinal artery occlusion

Retinal artery occlusion is associated with cardiovascular risk factors as in this case. It causes sudden onset painless visual loss. Fundoscopy may reveal a pale retina and the macula can appear as a 'cherry red spot'.

Retinal vein occlusion may present with distorted and blurred vision rather than visual loss. Additionally, fundoscopy may reveal vascular dilatation with associated haemorrhages.

Hypertensive retinopathy tends to be asymptomatic and is instead generally an indicator of endorgan damage due to long-term effects of hypertension.

Giant cell arteritis may be expected to have preceding symptoms of headache, jaw pain and scalp tenderness before abrupt visual loss.

Question 7 of 189

A 25 year-old woman with ataxia telangiectasia undergoes open biopsy of an enlarged cervical lymph node. Microscopic examination reveals changes consistent with nodular sclerosing Hodgkins disease. Unfortunately, she develops spreading cellulitis around the biopsy site which fails to respond to intravenous antibiotics and ultimately requires surgical debridement.

What is the most common reason for the increased susceptibility to infections in ataxia telangiectasia?

A. Hypogammaglobulinaemia
B. Neutropaenia
C. Hyposplenism
D. Defect in phagocyte
E. NADPH oxidase Complement deficiency

A. Hypogammaglobulinaemia

Ataxia telangiectasia is an autosomal recessive condition resulting in cerebellar ataxia with ocular telangiectasia. Individuals are often immunocompromised, typically due to hypogammaglobulinaemia. Lymphopaenia may also contribute.

There is also an increased risk of malignancy, typically lymphoma or leukaemia. A defect in phagocyte NADPH oxidase is responsible for chronic granulomatous disease.

Question 8 of 189

A 42 year-old man presents with an acute of onset of weakness in the left face, arm, and leg, which has not resolved. On examination there is upper motor neuron facial weakness on the left, with dense weakness of the left arm and leg. There is no evidence of sensory neglect, hemianopia, or dysphasia.

He past medical history includes migraine with visual aura, for which he takes propranolol and sumatriptan. His last migraine attack was a month ago. He has suffered two transient ischaemic attacks in the last year, and now also takes clopidogrel. His father also suffered from migraine with aura and died in his 50s after suffering a series of strokes.

Plain computed tomography shows multiple round lesions in the white matter, which appear the same density as cerebrospinal fluid. Magnetic resonance imaging shows scattered well-circumscribed lesions in the subcortical white matter which appear hypointense on T1 and hyperintense on T2-weighted sequences. On DWI (diffusion-weighted imaging) there is a hyperintense lesion in the right internal capsule, with a corresponding hypointense area on the ADC (apparent diffusion coefficient) map.

What is the most likely underlying diagnosis?

A. Patent foramen ovale
B. Progressive multifocal leukoencephalopathy
C. Migrainous infarction
D. Cerebral amyloid angiopathy
E. CADASIL

E. CADASIL

The clinical and radiological description fits with a lacunar infarction in the right internal capsule resulting in a pure motor hemiparesis. The area of acute infarction is not visible on the CT in the hyperacute phase, but is visible on the diffusion-weighted MR as described. The other white matter lesions are most likely old lacunar infarcts which may have been clinically silent.

The personal history of migraine with aura coupled with the family history make this most likely CADASIL (cerebral autosominal dominant arteriopathy with subcortical infarcts and leukoencephalopathy). The conditions results from mutations in the NOTCH3 gene onchromosome 19. Individuals typically develop migraine with aura around age 30, transient ischaemic attacks and ischaemic strokes in their 40s, and dementia around the 50s. Strokes are typically lacunar.

There is an association between patent foramen ovale, migraine with aura, and stroke. Stroke in this context is embolic, which characteristically produces wedge-shaped infarcts involving cortex and white matter, rather than the small lesions seen in lacunar stroke. Emboli may arise in the venous circulation and cross over into the arterial circulation via the shunt (paradoxical embolism).

Progressive multifocal leukoencephalopthy is a demyelinating disease of the central nervous system caused by reactivation of the JC virus, most typically seen in patients with AIDS and low CD4 counts.

In migranous infarction the neurological deficit develops during an attack of migraine.

Cerebral amyloid angiopathy is an important cause of primary lobar intracerebral haemhorrage in the elderly.

Question 9 of 189

A 43-year-old man is apprehended by the police after a violent assault outside a pub. He is brought to the attention of the duty psychiatrist because he is extremely confused and agitated. He repeatedly says that his wife is having an affair with the Prime Minister. He believes this to be true because of a message encoded in the traffic lights. He also mentions that he has not slept in the last two days.

The psychiatric assessment is interrupted when he has a tonic-clonic seizure, which self-terminates in 3 minutes. He is taken to the local Accident and Emergency department and reviewed by the medical team.

The medical SHO manages to contact his wife, who reports that he is normally fit and well and she last saw him when he left for work five days ago. At that time he appeared normal but complained of a headache, and took some ibuprofen.

During the clerking, he is noted to have repetitive chewing movements of the mouth. Again, the assessment is interrupted by a tonic-clonic seizure which does not terminate after 10mg of intravenous diazepam. After 15 minutes he is loaded with phenytoin. He continues to fit so is intubated and transferred to the Intensive

Care Unit. Termination of seizure activity is only achieved after induction of general anaesthesia with thiopentone.

What is the most likely diagnosis?

A. Schizophrenia
B. Fronto-temporal dementia
C. Acute intermittent porphyria
D. Post-ictal psychosis
E. Autoimmune limbic encephalitis

E. Autoimmune limbic encephalitis

Autoimmune limbic encephalitis presents with acute or sub-acute cognitive dysfunction, behavioural changes, and seizures. A number of specific antibody-related syndromes have been described. The description given here is suggestive of anti-NMDA receptor encephalitis, with a prodromal headache followed by prominent psychiatric features, with orofacial dyskinesias, insomnia, and progression to seizures.

Schizophrenia is a common cause of psychosis but the seizures should point away from a psychiatric disorder towards an organic brain pathology.

Fronto-temporal dementia is a slowly progressive condition.

Acute intermittent porphyria may cause a range of neurological manifestation including psychosis and seizures, but there are no features here that point specifically to this diagnosis.

Post-ictal psychosis is a rare manifestation of the post-ictal state, typically in patients who have had epilepsy for some time. The rapid development of psychosis and seizures in a previously well person should prompt the search for an alternative diagnosis.

Question 10 of 189

A 45 year-old man is referred to the nephrologists for investigation of chronic kidney disease. He has a past medical history of hypertension, type 2 diabetes, and Parkinsons disease, and his medication comprises ramipril, metformin, and bromocriptine.

Review of blood tests shows that the glomerular filtration rate (GFR) has steadily fallen from 85 to 44ml/min/1.73m^2 over the last year.

On questioning, his only symptom is chronic back pain which has been getting worse over the last year. On examination, both kidneys are easily palpable.

Routine investigations are as follows:

Abdominal ultrasound shows bilateral hydronephrosis.

Hb	12.1 g/dl
MCV	94.2 fl
Platelets	264 x10^9/l
WCC	7.1 x10^9/l
Na	137mmol/l
K	4.6 mmol/l
Urea	13.8 mmol/l
Creatinine	157 mol/l
eGFR	44 ml/min/1.73m^2
ALT	24 IU/l
ALP	78 IU/l
Bilirubin	6 mol/l
Albumin	37 g/l
Total protein	64 g/l
Serum protein electrophoresis	pending
Urine dipstick	negative for blood, protein, leucocytes, and nitrites

What is the most likely cause of his chronic kidney disease?

A. Multiple myeloma
B. Diabetic nephropathy
C. Bladder cancer
D. Retroperitoneal fibrosis
E. Autosomal dominant polycystic kidney disease

D. Retroperitoneal fibrosis

Bilateral hydronephrosis suggests that there is an obstruction to the outflow of both kidneys. This typically occurs at the bladder outlet, raising the possibility of a bladder or prostate cancer. The absence of haematuria makes bladder cancer less likely, and the absence of lower urinary tract symptoms makes prostate cancer less likely. However, if another diagnosis were not more forthcoming it would be appropriate to investigate for these conditions.

Bilateral hydronephrosis can more unusually result from obstruction of both ureters. This occurs in retroperitoneal fibrosis, where the ureters are encased in dense fibrous tissue. Back pain is a common symptom. The condition may be idiopathic, or may occur in association with an abdominal aortic aneurysm, or secondary to long-term use of ergot derived dopamine agonists such as bromocriptine, cabergoline, or pergolide.

In the context of back pain and chronic kidney disease, multiple myeloma is a distinct possibility. Serum protein electrophoresis is required to detect the presence of a paraprotein. However, subtracting total protein from albumin (64-37) suggests that the total immunoglobulin level is 27 g/L, and therefore there is unlikely to be a significant level of paraprotein. Equally, there is unlikely to be any urinary Bence Jones protein in the absence of dipstick proteinuria. Finally, one would not expect to see hydronephrosis in myeloma kidney.

The absence of proteinuria makes diabetic nephropathy less likely, and also one would not expect to see evidence of urinary tract obstruction in this condition.

At the age of 45, autosomal dominant polycystic kidney disease can be excluded by the absence of renal cysts on ultrasound.

Question 11 of 189

A 77-year-old female was brought into hospital after waking with left arm weakness predominantly affecting the hand with a left sided facial droop in an upper motor neuron pattern. Her blood pressure on admission was 175/90 mmHg and her heart rate was 80 beats per minute and in sinus rhythm. Her blood glucose level on admission was 7.2 mmol/L.

Her initial CT brain showed some mild bi-temporal atrophic change and some chronic small vessel ischaemia without any acute ischaemic changes and in particular, no haemorrhage.

She was admitted with a suspected diagnosis of minor ischaemic stroke.

Which of the following imaging modalities will confirm the diagnosis?

A. CT cerebral angiogram (CTA)
B. Fluid attenuated inversion recovery (FLAIR)
C. MRI Thin slice non-contrast enhanced CT brain scan
D. Diffusion weighted imaging (DWI) MRI
E. Formal cerebral digital subtraction angiogram (DSA)

D. Diffusion weighted imaging (DWI) MRI

DWI-MRI is the most sensitive and specific imaging modality for diagnosing acute stroke. FLAIR MRI is sensitive for chronic ischaemic changes.

Both CTA and DSA may identify significant intravascular stenoses or thrombosis but are insensitive for small areas of acute ischaemic change.

Thin slice CT scans may identify established ischaemia with more sensitivity than a standard CT scan but is still inferior to DWI MRI.

In general, the purpose of the initial CT scan in accident and emergency for suspected ischaemic stroke patients is to exclude a haemorrhage.

Question 12 of 189

A 37-year-old woman has been diagnosed with relapsing remitting multiple sclerosis and is considering further therapeutic options including natalizumab. She has had two severe relapses despite treatment with glatiramer acetate. She is currently still able to work, but is very fatigued and feels her mobility is limited.

On examination, she has brisk reflexes bilaterally, with mild weakness in her proximal left leg and reduced sensation in the left foot.

She has highly active disease on her brain MRI with multiple acute and sub-acute plaques present. She also has a single lower cervical cord lesion.

Before considering a new therapy, aside from HIV and general immunological status, which of the following investigation results is of greatest importance?

A. Cytomegalovirus status
B. Syphilis serology
C. Hepatitis C serology
D. EBV serology
E. JC virus status

E. JC virus status

Natalizumab is a therapy approved for the management of highly active relapsing-remitting multiple sclerosis (RRMS) and is given as a once-monthly infusion. Natalizumab has been shown to greatly reduce relapse rates in RRMS.

It is a humanised monoclonal antibody against the cell adhesion molecule (CAM) 4-integrin. Natalizumab prevents leukocyte adhesion to endothelial VCAM1, thus preventing the migration of immune cells into the CNS.

An extremely rare, severe and almost universally fatal adverse side effect of Natalizumab is progressive multifocal leukoencephalopathy (PML). Only patients who are seropositive for JC virus are at risk of developing PML with exposure to natalizumab, and therefore checking the JC virus status of patients who are considering natalizumab therapy is mandatory.It is also worth noting that some patients with low titres of JC virus are still commenced on natalizumab under close monitoring and that duration of natalizumab therapy is proportional to the risk of developing PML, i.e..short duration therapy (1-2 years) carries a very low risk of developing PML, even in those with a low positive JC virus titre. Ultimately, patients who are JC virus positive should not receive natalizumab and another therapy should be considered.

Question 13 of 189

A 45-year-old male presents with ascending weakness. He first noticed that he was tripping over more easily, but now has trouble getting out of a chair. He feels otherwise well. Of note, he did have moderate diarrhoea which had completely resolved a week prior to developing this weakness.

On examination, he is haemodynamically stable with a heart rate of 68 beats per minute and a blood pressure of 135/80 mmHg. His respiratory rate is 18 breaths per minute. He has reduced power in ankle plantar and dorsiflexion bilaterally, absent ankle jerks and reduced knee jerks. His plantar responses are downwards.

Acutely, which of the following results will most assist you with a diagnosis?

A. MRI showing inflammation of the lumbo-sacral spinal cord
B. Elevated white cell count on cerebrospinal fluid (CSF) analysis
C. Abnormal nerve conduction tests of the lower limbs
D. Elevated CSF protein
E. Raised erythrocyte sedimentation rate (ESR)

D. Elevated CSF protein

Acutely, an elevated CSF protein level may be the only indication of an inflammatory aetiology, highly suggestive of Guillain-Barre Syndrome (GBS) or Acute Inflammatory Demyelinating Polyradiculoneuropathy in this clinical setting. The CSF white cell count is not elevated in GBS.

In relation to nerve conduction studies, these often provide normal results within the acute setting and the classic demyelination findings may not be detectable for at least a week.

Question 14 of 189

A 65-year-old male with a 60 pack year smoking history has been diagnosed with small cell lung cancer. He is currently undergoing chemotherapy. Over the last few months, he has noticed his vision deteriorating and complains of diplopia. He also feels weaker in his upper limbs although his symptoms do fluctuate.

On examination, he has mild ptosis of the eyelids bilaterally and a complex ophthalmoparesis affecting both eyes. He also has reduced power proximally in the upper limbs.

In this clinical syndrome, in addition to electrophysiological studies, which of the following blood test results is likely to assist with the diagnosis?

A. Presence of anti-muscle specific kinase antibodies
B. Presence of anti-neuronal antibodies
C. Presence of voltage gated potassium channel antibodies
D. Presence of sodium channel antibodies
E. Presence of voltage gated calcium channel antibodies

E. Presence of voltage gated calcium channel antibodies

The paraneoplastic myasthenic syndrome described above associated with small-cell lung cancer is Lambert-Eaton myasthenic syndrome (LEMS).

Antibodies to voltage-gated calcium channels (VGCCs) have been reported in 75-100% of LEMS patients who have small cell lung cancer (SCLC) and in 50-90% of LEMS patients who do not have underlying cancer.

Question 15 of 189

A 75-year-old female on aspirin for known ischaemic heart disease is being investigated for the cause of a recent transient ischaemic attack (TIA) which caused brief left sided weakness. She does not have a history of atrial fibrillation, nor has any been detected on routine telemetry.

Her MRI brain scan does not provide any evidence for acute stroke and her transthoracic echocardiogram does not demonstrate any intra-cardiac thrombus.

A carotid ultrasound study revealed a 75-85% stenosis of the right internal carotid artery.

In addition to ordering a CT carotid angiogram to further investigate the lesion, the next best step in her management would be to?

A. Add clopidogrel
B. Commence best medical management and re-image in 6 months
C. Commence an unfractionated heparin infusion
D. Commence best medical therapy and refer for a carotid endarterectomy in 6-8 weeks
E. Commence best medical therapy and refer for a carotid endarterectomy within 14 days

E. Commence best medical therapy and refer for a carotid endarterectomy within 14 days

Patients with a TIA or small ischaemic stroke with evidence of significant carotid artery stenosis (male patients with a carotid artery stenosis of 50-99% or female patients with a carotid artery stenosis of 70-99%) that corresponds to the same side as the stroke/TIA should be considered for an urgent carotid endarterectomy (CEA) within 14 days.

Question 16 of 189

An 81-year-old lady with a history of congestive cardiac failure was assessed in accident and emergency and clinically diagnosed with an acute ischaemic stroke. Her main deficits were slurred speech and left sided facial droop with some loss of fine motor control in the left hand. On admission, her blood pressure was 185/70mmHg with a heart rate of 95 beats per minute in sinus rhythm.

The initial CT scan of her brain showed some evidence of chronic small vessel ischaemia, but no acute pathology, in particular no haemorrhage was seen.

Which of the following combinations of investigations should be carried out during the acute admission?

A. MRI, transthoracic echocardiogram (TTE), cardiac telemetry and carotid duplex study
B. Transoesophageal echocardiogram (TOE), CT cerebral angiogram and carotid duplex study
C. CT scan, fasting lipids and a video fluoroscopy swallow study
D. Fasting glucose, Electroencephalogram (EEG), MRI and thrombophilia screen
E. Thrombophilia screen, MRI and a TTE

A. MRI, transthoracic echocardiogram (TTE), cardiac telemetry and carotid duplex study

Question 17 of 189

A 42-year-old man presented with a partial left Horner's syndrome (ptosis with miosis, no anhidrosis), ipsilateral neck pain and reduced visual acuity on the same side.

He reported that the first symptom was neck pain and that his vision worsened over the next two hours. On admission, his blood pressure was 145/80 mmHg and heart rate was 80 beats per minute in sinus rhythm. His chest was clear.

His initial non-contrast enhanced CT brain scan showed a very small area of gliosis in the left frontal area possibly related to a distant injury, but no acute pathology.

Which of the following tests is likely to give you a diagnosis?

A. MRI of the brain
B. CT carotid angiogram
C. Chest X-Ray
D. Erythrocyte sedimentation rate (ESR) and temporal artery biopsy

E. CT venogram of the neck and brain

B. CT carotid angiogram

The oculosympathetic pathway leads a course from the posterior hypothalamus, through the brainstem followed by the spinal cord, over the apex of the lung to the superior cervical ganglion, and then finally ascends along with the internal carotid artery to ultimately reach the pupillary dilator muscle and Mueller's muscle of the lid. These post ganglionic fibres within the carotid sheath are vulnerable to the compressive effects of a mural haematoma or pseudoaneurysm resulting from a carotid artery dissection. Such compression can result in a partial Horner's syndrome.

In addition to this, patients with both carotid and vertebral artery dissections often present with neck pain.

Finally, in patients with a carotid dissection retinal ischaemia is often as a result of thrombi produced within the dissected portion of the vessel that then embolize distally towards the brain or in this case, the ophthalmic artery, a branch of the internal carotid artery.The investigation of choice to diagnose a carotid dissection is a CT carotid angiogram, however, other such as MR angiograms of the neck are also adequate.

Question 18 of 189

A 62 year-old man with a history of osteoarthritis, type II diabetes and mild dementia is seen in neurology clinic. He has a 2 month history of weakness in his right arm. He has also noticed that his voice has become softer. He is finding it hard to use door handles and open jars. On two occasions his wife has noticed him stumbling whilst walking.

On examination he has fasciculations over his right deltoid muscle and wasting of the interossei muscles of the right hand. There is 4/5 strength in right shoulder abduction on the Medical Research Council (MRC) scale. Limb reflexes are absent in the right arm but detectable elsewhere. Coordination is normal. Sensation is normal and Romberg's test is negative.

What is the most likely diagnosis?

A. Compressive Cervical myelopathy
B. Diabetic peripheral neuropathy
C. Motor neurone disease
D. Multiple sclerosis
E. Thoracic outlet syndrome

C. Motor neurone disease

Arm weakness can have many causes, but in this case the voice is also affected as are the lower limbs.

Fasciculations imply lower motor neurone involvement and would make a diagnosis of multiple sclerosis unlikely. Cervical myelopathy or diabetic neuropathy would not explain the voice change.

Thoracic outlet syndrome would not affect the patients legs so the most likely diagnosis in this case is motor neurone disease.

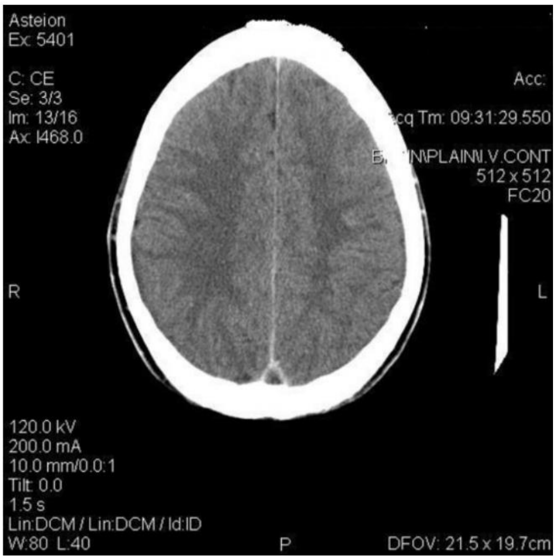
Motor neurone disease is associated with fronto-temporal dementia.

Question 19 of 189

A 19-year-old female is admitted to the Emergency Department after suffering a seizure. Her friends report that she is normally fit and well but had been complaining of a bad headache for the past few hours.

On examination her GCS is 13/15 (M6 V4 E3). No focal neurological deficit is noted. She appears slightly confused and is holding her head in her hands. Her pulse rate is 90/min with a temperature of 37.2º. No neck stiffness is noted.

A CT scan with contrast is requested:

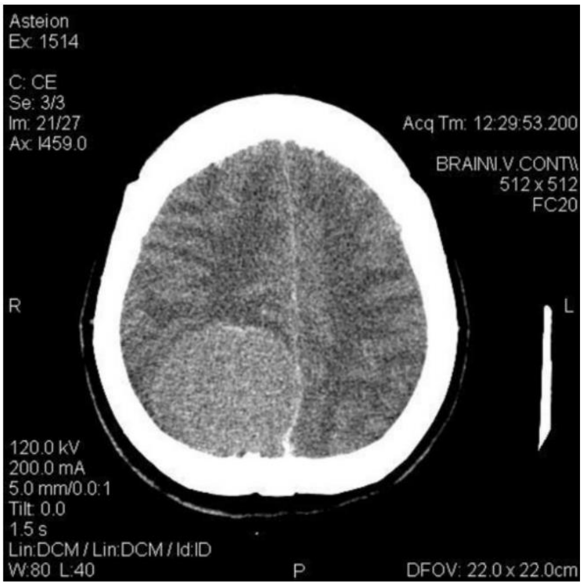


What is the most likely diagnosis?

A. Extradural haemorrhage
B. Superior sagittal sinus thrombosis
C. Herpes simplex encephalitis
D. Subrarachnoid haemorrhage
E. Meningioma

B. Superior sagittal sinus thrombosis

The CT with contrast demonstates a superior sagittal sinus thrombosis showing the typical empty delta sign. Look at the 'bottom' of the scan for the triangular shaped dural sinus. This should normally be white due to it being filled with contrast. The empty delta sign occurs when the thrombus fails to enhance within the dural sinus and is outlined by enhanced collateral channels in the falx. This sign is seen in only about 25%-30% of cases but is highly diagnostic for sagittal sinus thrombosis.

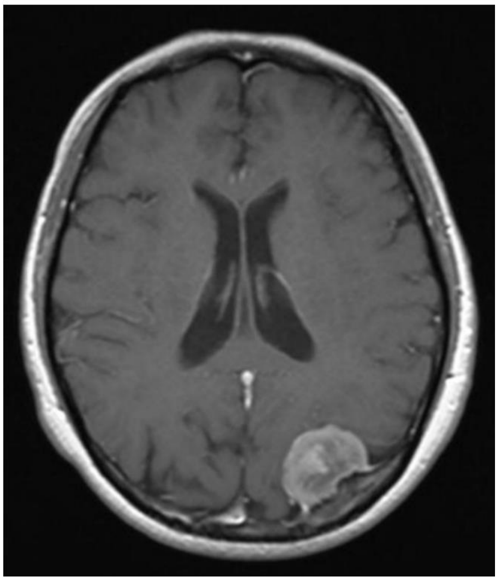


Question 20 of 189

A 54-year-old woman is admitted to the Emergency Department after having a seizure. This occured whilst she was a passenger in a car. There is no history of trauma. By the time of her admission she is alert with a GCS of 15/15. Her partner describes a generalised seizure with a postictal phase lasting around 30 minutes.

On examination her heart rate is 90/min, blood pressure 102/60 mmHg and temperature 37.1ºC. There are no focal neurological signs.

A MRI scan is requested:



What is the most likely diagnosis?

A. Extradural haematoma
B. Gliolastoma multiforme
C. Subdural haematoma
D. Meningioma
E. Oligodendroma

D. Meningioma

The MRI shows the typical well-circumscribed appearance of a meningioma. A dural tail can be where the tumour 'connects' to the dura. It is seen in around 65% of meningiomas.

Question 21 of 189

A 45-year-old woman presents to the Emergency Department due to a headache. This has been getting gradually worse over the past 3 months. Her GP has tried a number of therapies including a triptan, amitriptyline and standard analgesia with limited effect. She is a non-smoker and drinks around 30 units of alcohol per week.

Neurological examination is unremarkable. A CT scan is arranged:

What is the most likely diagnosis?

A. Extradural haematoma
B. Meningioma
C. Glioblastoma multifome
D. Herpes simplex encephalitis
E. Subdural haematoma

B. Meningioma

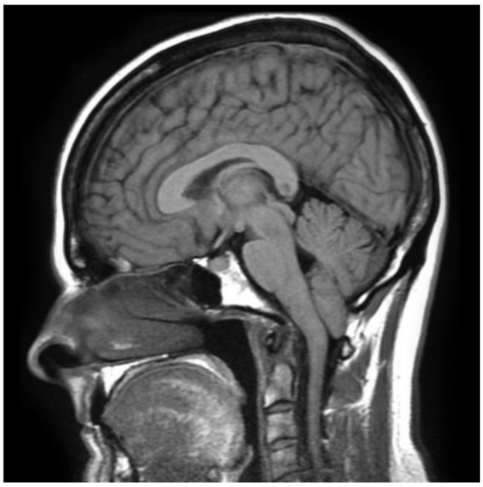
The CT shows a well defined spherical mass in the right posterior falx cerebri consistent with a meningioma. There is mild oedema and mass effect on the right lateral ventricle. The tumour is straddling the inferior surface of the falx.

A glioblastoma multifome would normally have a more heterogenous appearance than shown here.

Question 22 of 189

A 34-year-old woman is referred to the neurology clinic for a 6 month history of progressively worsening headaches. The headaches tend to be occipital in nature and are worsened by coughing.

Neurological examination is unremarkable. Her MRI is shown below:



What finding is shown on the MRI?

A. Brainstem cavernous angioma
B. Pituitary adenoma
C. Arnold-Chiari malformation
D. Arachnoid cyst
E. Spina bifida

C. Arnold-Chiari malformation

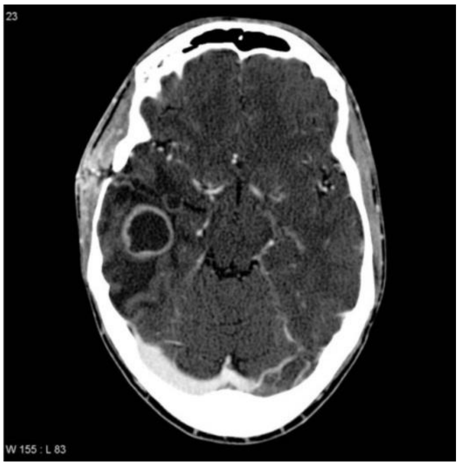
The MRI shows herniation of the cerebellar tonsils through the foramen magnum consistent with a Chiari type I malformation.

Question 23 of 189

A 23-year-old man is admitted to ITU after arriving from India on a medical transfer plane. He had been travelling in India for around 4 weeks before starting to complain of feeling unwell. His girlfriend describes acting out of character and having numerous seizures prior to being admitted to hospital in Patna, east India.

His girlfriend describes him having a scan in India as well as a procedure in theatre which was abandoned.

A CT head with contrast is performed:



What is the most likely diagnosis?

A. Neurocysticercosis
B. Meningioma
C. Glioblastoma multiforme
D. Cerebral abscess
E. Herpes simplex encephalitis

D. Cerebral abscess

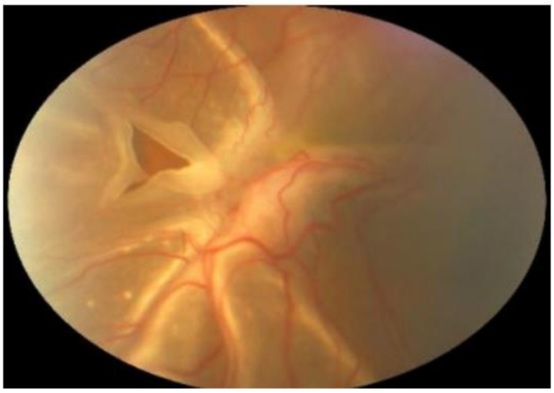
CT demonstrating a cerebral abscess. CT demonstrates a rounded lesions with vast amounts of surrounding vasogenic oedema resulting in mass effect and distortion of the cerebral peduncle. Contrast confirms the presence of a smooth ring of enhancement, characteristic of an abscess.

The scan shows signs of overlying surgical change consistent with a prior attempted drainage via a burr hole.

This patient underwent repeat surgery with successful drainage of the abscess. He made a slow but uneventful recovery.

Question 24 of 189

A woman presents with reduced vision in her right eye. She is unsure when it started but can now only make out hand movements with the right eye. Fundoscopy of the affected eye shows the following



What is the diagnosis?

A. Penetrating foreign body
B. Myelinated optic disc
C. Papilloedema
D. Choroidoretinitis
E. Retinal detachment

E. Retinal detachment

The correct diagnosis is Retinal detachment. This condition presents with symptoms such as sudden or gradual reduction in vision, often described by patients as a 'curtain coming down'. Fundoscopy reveals an elevated retina with folds and a visible retinal tear.

Penetrating foreign body is incorrect because it would typically present with a history of trauma, and the patient would likely experience pain. On examination, there might be visible injury to the cornea or sclera, hyphaema (blood in the anterior chamber), irregular pupil, or low intraocular pressure.

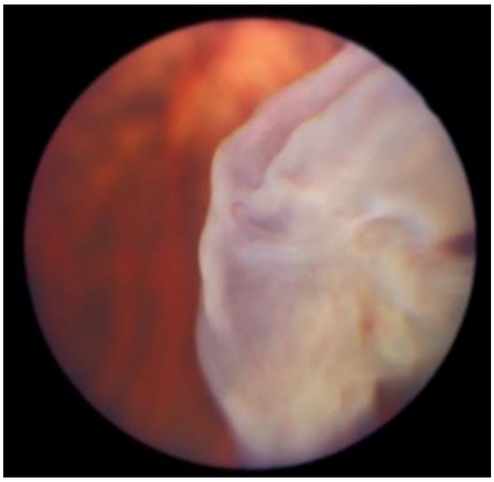
Myelinated optic disc, also known as medullated nerve fibres, is incorrect because it does not typically affect vision. It appears on fundoscopy as whitish patches extending from the optic disc but does not cause elevation or detachment of the retina.

Papilloedema is incorrect because this condition involves swelling of the optic disc due to increased intracranial pressure. Symptoms can include transient visual obscurations and headache. On fundoscopy, there would be blurring of the optic disc margins and elevation of the disc itself rather than the retina.

Finally, Choroidoretinitis, inflammation of the choroid and retina usually due to infection such as by Toxoplasma gondii or Cytomegalovirus, is incorrect because it typically presents with floaters, photopsia (flashes of light), and blurred vision due to inflammatory lesions in the retina. These lesions appear as yellow-white patches during fundoscopy but do not cause retinal detachment unless severe and chronic.

Question 25 of 189

A 71-year-old man presents with sudden painless loss of vision in his left eye: Fundoscopy shows the following



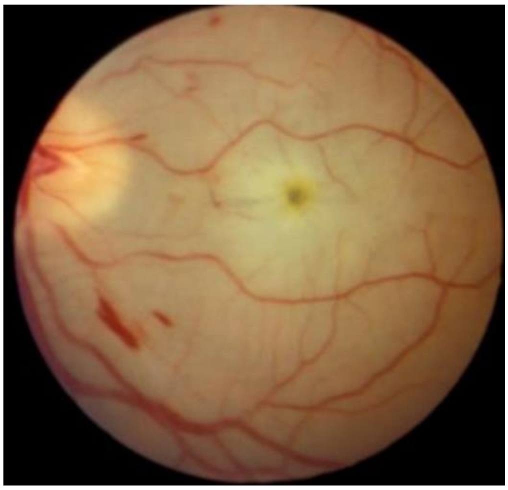
What is the most likely diagnosis?

A. Ciliary body rupture with lens dislocation
B. Vitreous haemorrhage
C. Ischaemic optic neuropathy
D. Central retinal artery occlusion
E. Retinal detachment

E. Retinal detachment

Question 26 of 189

This man presents with unilateral visual loss. On examination he has a relative afferent pupillary defect. Fundoscopy shows the following



What is the most likely diagnosis?

A. Central retinal vein occlusion
B. Subhyaloid haemorrhage
C. Solar retinopathy C
D. entral retinal artery occlusion
E. Optic neuritis

A. Central retinal vein occlusion

The correct answer is **Central retinal artery occlusion.** This condition presents with sudden, painless, unilateral vision loss. On examination, there may be a relative afferent pupillary defect (RAPD), also known as Marcus Gunn pupil, which is an abnormal response to light in the affected eye due to a severe decrease in the number of functioning retinal ganglion cells. Fundoscopy typically reveals a pale retina with a cherry-red spot at the fovea due to obstruction of blood flow in the central retinal artery.

Central retinal vein occlusion is incorrect because it presents differently. Patients often complain of sudden or gradual vision loss. On fundoscopy, there are widespread retinal haemorrhages ('blood and thunder' appearance) and cotton wool spots due to blockage of the central retinal vein leading to increased pressure and subsequent leakage from other vessels.

Subhyaloid haemorrhage is also incorrect. It presents with sudden visual loss or floaters but does not typically cause RAPD. On fundoscopy, you would see a pre-retinal (subhyaloid) haemorrhage which appears as a dark red, boat-shaped mass located between the retina and vitreous humour.

The diagnosis of **Solar retinopathy** is unlikely in this case as it results from direct solar radiation damage to the macula leading to central scotoma (blind spot). The typical fundoscopic finding is yellowish dots at the level of RPE in fovea.

Lastly, **Optic neuritis**, commonly associated with multiple sclerosis, typically presents with painful vision loss over days accompanied by dyschromatopsia (impaired colour vision). However, fundoscopy may initially appear normal or show mild disc swelling rather than the findings described here.

Question 27 of 189

A 19-year-old university student is reviewed in the general medical clinic with bilateral ptosis. It has developed gradually over the last few months and has now progressed to the extent that he has to tilt his head upwards during lectures. He denies diurnal variation in his symptoms.

His past medical history is unremarkable and he takes no regular medications. He tells you that his mother had a pacemaker fitted due to frequent 'blackouts'.

On examination, you note the presence of bilateral ptosis. He also has impaired abduction and adduction of both eyes. Direct fundoscopy shows black spicules in the periphery of both eyes. An ECG is taken and shows sinus rhythm with first-degree heart block.

Given the likely diagnosis, which additional symptom is he most likely to describe?

A. Night blindness
B. Syncope
C. Tremor
D. Flank pain
E. Diplopia

A. Night blindness

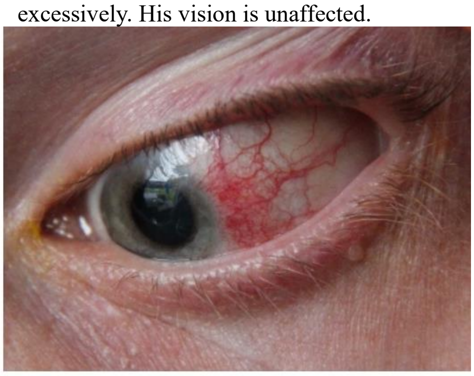
The most likely diagnosis is Kearns-Sayre syndrome, a mitochondrial disorder. Individuals usually present <20 years-of-age with progressive external ophthalmoplegia. Ptosis usually develops first, followed by difficulties with horizontal gaze. Patients don't usually complain of diplopia as the extraocular muscle weakness is symmetrical.

Affected patients also usually suffer from pigmentary retinopathy, which causes atrophy of the retinal pigment with bony spicule formation typical of retinitis pigmentosa. As the changes progress, patients often complain of funnel-vision and night blindness.

Cardiac conduction defects usually develop after the onset of ophthalmoplegia and sudden death may occur as a result. Regular cardiac follow-up is essential. Other abnormalities include cerebellar ataxia, sensorineural deafness, muscle weakness and diabetes mellitus..

Question 28 of 189

A 44-year-old man attends after waking up with a red eye and some slight crusting. He describes mild pain and finds the eye is tearing excessively. His vision is unaffected.



Slit lamp examination shows congestive vessels with a radial distribution pattern. He works as a carpenter and has a background of ulcerative colitis but is otherwise fit and well.

What is the most likely diagnosis?

A. Conjunctivitis
B. Episcleritis
C. Exposure keratopathy
D. Ocular foreign body
E. Scleritis

B. Episcleritis

This is a presentation of episcleritis. **Episcleritis** is usually painless or with mild pain, there are prominent surface vessels that are mobile and blanch with phenylephrine drops.

Episcleritis is correct. It is an inflammatory condition affecting the episclera and is usually self-limiting. It is not classically associated with severe pain and vision is preserved.

Conjunctivitis is incorrect. This usually presents with purulent discharge and extensive crusting around the eyes. The conjunctiva is often inflamed. It commonly initially affects one eye but can spread easily to affect both. In this case, there is no purulent discharge and so conjunctivitis is less likely.

Exposure keratopathy is incorrect. This is when the cornea is exposed for prolonged periods of time. It can present with a gritty or foreign body sensation and the damaged areas of the cornea will stain with fluorescein. In this case, there is no foreign body sensation and no staining on the cornea.

Ocular foreign body is incorrect. This would commonly be associated with discomfort and a foreign body would be seen on slit lamp examination. This is not seen here.

Scleritis is incorrect. This is also an inflammatory condition but is more classically associated with pain, photophobia, and blurring of the vision. Slit lamp examination usually shows new vessel formation and abnormal vessel distribution. These features are not present for the patient in the stem.

Question 29 of 189

A 45-year-old lady was admitted to the Medical Admission Unit with a 16-hour history of weakness. Two days prior to the admission she experienced a sensation of double vision and shortly afterwards had become more unsteady when walking. The weakness developed initially in her arms and was shortly followed by weakness in her legs. Her past medical history included a history of coeliac disease for which she adhered to a strict gluten-free diet, but she was otherwise fit and well.

On examination, she was not distressed and was fully orientated to her surroundings. She had a temperature of 37.6ºC, a heart rate of 88/min, a respiratory rate of 18/min and a blood pressure of 182/82 mmHg. The cardiovascular and respiratory examination was otherwise unremarkable. Examination of the abdomen revealed a mass arising from the symphysis pubis. Examination of her cranial nerves revealed a failure of bilateral external gaze, as well as diplopia on asking the patient to fixate down and in. Her pupils were dilated but reactive. Fundoscopy revealed no abnormalities, and cranial nerve examination was otherwise unremarkable. The tone was reduced in all muscle groups, with a power of grade 3/5 in all muscle groups. Reflexes were absent in all limbs and plantar responses were normal. She was unable to mobilise independently and an ataxia was noted.

Initial investigations revealed the following:

Hb	132 g/l
Platelets	222
WBC	5.8
ESR	16 mm/hr
Na	139 mmol/l
K	3.7 mmol/l
Urea	4.3 mmol/l
Creatinine	77 µmol/l
Bilirubin	11 µmol/l
ALP	101 u/l
ALT	22 u/l
Glucose	6.0 mmol/l
CRP	22 mg/l

CSF

Appearance	Clear
Glucose	4.5 mmol/l
Protein	0.6 g/l
White cells	3 / mm³

CT brain scan: normal appearances, no evidence of haemorrhage, midline shift or space occupying lesion.

What is the single investigation most likely to lead to a diagnosis?

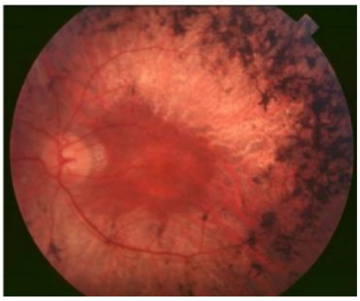
A. Anti Jo1 antibodies
B. Anticholinesterase antibodies
C. Anti GQ1b antibodies
D. Anti GM1 antibodies
E. Antinuclear antibodies

C. Anti GQ1b antibodies

The combination of ophthalmoplegia, ataxia and areflexia is strongly suggestive of Miller-Fisher syndrome, a variant of Guillain-Barre syndrome (GBS). Anti GQ1b antibodies are present in 90% of cases. Anti GM1 antibodies are present in axonal neuropathies including GBS but also other conditions and are not specific to Miller Fisher syndrome. Anti Jo1 antibodies are found in polymyositis whilst anticholinesterase antibodies are found in myasthenia gravis.

Question 30 of 189

A 35-year-old man with a vague history of vision problems is reviewed in the ophthalmology clinic. He has not seen a doctor in years and does not report a record of any medical illness.



Fundoscopic examination revealed the following:

What is the most likely cause of the fundoscopic findings?

A. Hereditary retinal degeneration
B. Hypertensive retinopathy
C. Non-proliferative diabetic retinopathy
D. Proliferative diabetic retinopathy
E. Vitreous haemorrhage

A. Hereditary retinal degeneration

The black bone spicule-shaped pigmentation in the peripheral retina and the mottling of the retinal pigment epithelium seen in this fundoscopic image suggest retinitis pigmentosa.

Retinitis pigmentosa is a hereditary condition characterised by retinal degeneration, affecting predominantly the rods and the peripheral retina. Night blindness is often the initial sign, and tunnel vision is due to the loss of the peripheral retina.

Hypertensive retinopathy is characterised by arteriolar narrowing and AV nipping in the early stages. The flame and blot haemorrhages collected around the fovea can result in a characteristic macular star pattern. Papilloedema is seen in advanced disease.

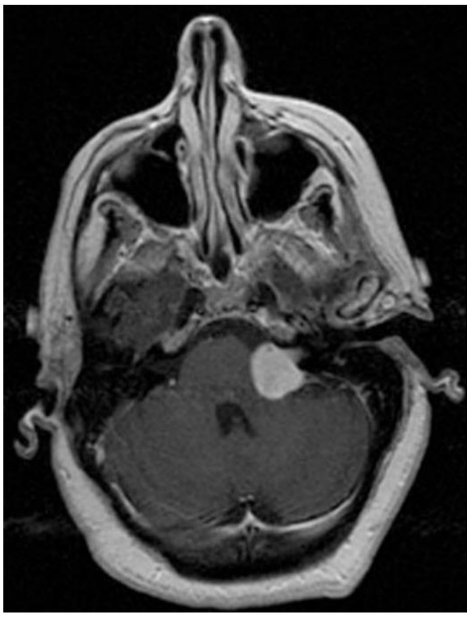
Non-proliferative diabetic retinopathy is characterised by microaneurysms, blot haemorrhages, hard exudates, cotton wool spots and venous beading or looping with no evidence of retinal neovascularisation or fibrous tissue.

Proliferative diabetic retinopathy is characterised by retinal neovascularisation predominantly around the optic disc or can be seen as neovascularisation elsewhere (NVE). Occasionally fibrous tissue may be seen anterior to the retinal layer.

Vitreous haemorrhage is seen as a red haze of haemorrhage into the vitreous. Dot blot haemorrhages, neovascularisation or boat-shaped retinal tears may also be seen.

Question 31 of 189

A 55-year-old man presents with six weeks history of tinnitus. He has noted some changes in hearing lately. There is no record of any medical illness, and he does not take any regular medications. He is a chronic smoker and has 40 pack year history. His MRI brain showed:



What is the next best step in his management?

- A. Computed tomography (CT) of thorax
- B. Intratympanic steroid therapy
- C. Systemic steroid therapy
- D. Urgent audiogram
- E. Urgent referral to ENT

E. Urgent referral to ENT

The MRI brain shows an intracanalicular solid nodular mass with an intense contrast enhancement in the right internal auditory meatus (IAM), which is suggestive of vestibular schwannoma.

Urgent referral to ENT is the correct answer. The patient's presentation and the MRI findings are in keeping with vestibular schwannoma, and the patient requires an urgent referral to ENT for further management.

Computed Tomography of thorax is incorrect. While a metastatic lesion of the cerebellopontine angle is a likely differential given the smoking history, the patient may benefit from a chest radiograph and/or a CT of the thorax to assess for lung neoplasms. However, the presentation and the MRI findings are characteristic of vestibular schwannoma, and the patient needs an urgent referral to ENT.

Intratympanic steroid therapy is incorrect. Intratympanic steroid injection treats vestibulocochlear symptoms of inner ear disease, such as Meniere's disease or idiopathic sudden sensorineural hearing loss. However, this patient's MRI suggests vestibular schwannoma as the likely cause for his hearing loss and requires urgent referral to ENT.

Systemic steroid therapy is incorrect. While systemic steroid therapy may be considered in treating sensorineural hearing loss associated with vestibular schwannoma, there is no adequate evidence to support its routine use in patients with vestibular schwannoma. The patient should be referred to ENT.

Urgent audiogram is incorrect. Patients with sensorineural hearing loss, tinnitus and vertigo should have an audiogram and MRI brain to assess for vestibular schwannoma. However, this patient's MRI brain is diagnostic of vestibular schwannoma and requires urgent ENT referral.

Question 32 of 189

An 82-year-old man is referred to neurology clinic with slowness. He presented with his wife who reported that he has been becoming progressively slower in his movements and his facial expressions have become more limited. His symptoms have progressed rapidly following the onset of dizziness a few weeks ago. He has a past medical history of type two diabetes, hypertension, high cholesterol, previous hemicolectomy for diverticulitis. His current medications include ramipril, atorvastatin, paracetamol, amlodipine, metformin, prochlorperazine and gliclazide.

On examination, he has a coarse bilateral tremor at rest, and rigidity in both arms. He has a slow gait as well.

What is the most likely diagnosis?

- A. Parkinson's disease
- B. Drug-induced parkinsonism
- C. Multi-system atrophy
- D. Vascular parkinsonism
- E. Wilson's disease

B. Drug-induced parkinsonism

The answer is drug-induced parkinsonism. There are several hints to why this is more likely. A bilateral tremor of rapid onset makes drug-induced parkinsonism a more probable cause when compared to Parkinson's disease. The recent dizziness suggests that prochlorperazine was only recently introduced, making it more suspicious that it may the cause of the current symptoms. Vascular parkinsonism would have a more step-wise progression with definitive days of significant deterioration, which are not described here. Multi-system atrophy generally has greater autonomic involvement, as well more cerebellar dysfunction. Wilson's disease typically occurs in younger patients, and is also assoaciated with liver dysfunction.

Question 33 of 189

A 28-year-old woman presents with a gradually worsening headache, nausea and vomiting.

The headache started about 3 days ago coming on over about 3-4 hours and is worse on coughing and lying flat. She has been complaining of some nausea and vomiting which she had attributed to some reheated curry she had the night before.

She is usually fit and well but did suffer a spontaneous below-knee deep vein thrombosis (DVT) 3 years ago which was treated with warfarin for 6 months. She has been taking over-the-counter paracetamol and ibuprofen for the headache and currently has a Mirena intrauterine coil in-situ for contraception. She has no known drug allergies.

She currently works as a sales associate. She smokes 10 cigarettes a day and drink 21 units of alcohol a week.

On examination she looks unwell, pale and nauseated. Her blood pressure was 140/98 mmHg, heart rate 100 bpm, oxygen saturations of 98% on air. Her temperature was 36.1 degrees Celsius.

Heart sounds were normal, and her calves soft and non-tender with no evidence of thromboembolism.

Her chest was clear and her abdominal examination was unremarkable.

On neurological examination there was no cranial nerve abnormalities noted. She had normal tone, power, reflexes and sensation in both her upper and lower limbs. Coordination was intact. Bloods taken by the Emergency Department were as follows:

Na+	136 mmol/L
K+	3.9 mmol/L
Urea	4.8 mmol/L
Creatinine	76 µmol/L
Hb	12.5 g/dL
WBC	11.0 x 10^9/L
Platelets	350 x 10^9/L
INR	0.9
aPTT	30 seconds
LFTs	Normal

CT head was performed and reported as normal. Following this a lumbar puncture was performed:

CSF colour	Clear
Opening pressure	250 mmH20
White cell count	5.0 x 10^6/L (all lymphocytes)
Red cell count	15 x 10^6/L
Protein	0.3 g/L
Glucose	4.4mmol/L (Serum glucose 5.3mmol/L)

What is the most likely diagnosis?

A. Intracranial venous thrombosis
B. Subarachnoid haemorrhage
C. Migraine
D. Intracerebral malignancy
E. Subdural empyema

A. Intracranial venous thrombosis

This lady has had a previous venous thromboembolism and presents with a gradually worsening headache with multiple red flags. The history and examination is rather non-specific but it is the CSF opening pressure in the context of previous venous thrombus that most points towards the diagnosis of an intracranial venous thrombosis. This can present with a gradual or severe headache, signs of raised intracranial pressure and cranial nerve palsies.

Subarachnoid haemorrhage is unlikely in the context of the gradual onset of symptoms. This patient has no previous history of migraine and the investigations are not consistent with this.

She is apyrexial and there is nothing in her presentation to indicate an intracerebral abscess, furthermore the CT is normal.

A normal CT also rules out intracerebral malignancy.

Question 34 of 189

You are the medical registrar on call. While reviewing another patient, you notice an 82-year-old female inpatient on the same ward, currently treated for community-acquired pneumonia sitting in a chair. You notice a persistent movement of her head, in a nodding motion, associated with a tremor of both hands, worse in the left than the right. Reading through her notes, she has previously been treated for epilepsy and was started on oral phenytoin by her GP 4 months ago. On examination, the hand tremor appears to worsen when her arms are outstretched. She performs finger-nose dysmetria testing without difficulty with no speech. She demonstrates no cogwheeling. The patient appears unconcerned by the symptoms 'I have learned to live with it for years doctors!' she tells you.

What is the most likely diagnosis?

A. Tremor-predominant
B. Parkinson's disease
C. Phenytoin induced cerebellar tremor
D. Essential tremor
E. Physiological tremor
F. Orthostatic tremor

D. Essential tremor

The patient's examination is one of isolated tremor with no other cerebellar or neurological features. The clinical features of worsening on posture, head nodding and lack of cerebellar symptoms suggest a diagnosis of essential tremor. She demonstrates no signs of Parkinson's disease, a tremor that does not alleviate with movement and the history describes onset beyond the start of phenytoin. Orthostatic tremor onsets in the trunk and legs when the patient stands. The described features are perhaps too severe for a physiological tremor, which is typically low in amplitude and barely visible. It is unlikely the patient would have received a beta agonist for a community-acquired pneumonia in the absence of obstructive airways disease, hence a druginduced tremor is also unlikely.

Question 35 of 189

A 48-year-old male presents with a sudden onset occipital headache onset 2 and half hours ago, associated with slurred speech, vomiting and unsteadiness in all movements. In the 90 minutes after admission to the emergency department, it was noted that the patient became increasingly drowsy, deteriorating from a GCS of 15 on admission to E3 V4 M5. On examination, pupils are equal, his speech is dysarthric and bilateral plantars are downgoing. You are unable to elicit more formal power, tone or sensation examination in the patient. An initial CT head is unremarkable but a subsequent MRI head with diffusion weighting sequences demonstrates restricted diffusion in bilateral cerebellar hemispheres with significant swelling around the cerebellum, brainstem and aqueduct. GCS currently 14/15 at 10 hours post-event. You have initiated aspirin 300mg and inserted a nasogastric tube.

What is the appropriate management?

A. Add clopidogrel 300mg
B. Start warfarinisation
C. Thrombolysis

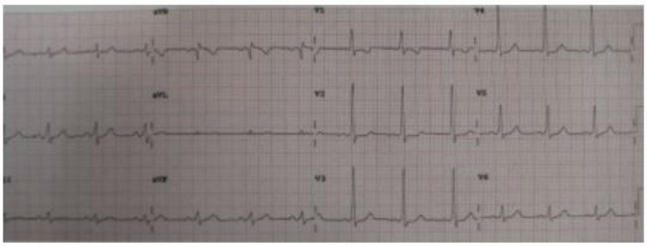
D. Discuss with neurosurgery for decompressive posterior craniectomy
E. Hourly neuro observations with empirical anti-seizure medications

D. Discuss with neurosurgery for decompressive posterior craniectomy

This patient is critically unwell. He has presented with a posterior circulation infarct significant oedema around a very tight part of the neuro-anatomy. Although pupils are currently equal and reactive, the drop in GCS is a major concern. It may not be possible to illicit upper motor neurone signs in the hyperacute phase after a stroke. The most concerning complication at this stage is a progression towards obstructive hydrocephalus from CSF flow blockage at the aqueduct or 3rd or 4th ventricle. Prophylactic posterior craniectomy must be considered in such a young patient, discussion with neurosurgery is essential.

Question 36 of 189

A 14-year-old boy presents his third generalised seizure over the past 72 hours, despite recently being started on sodium valproate by a neurologist for recurrent seizures 6 weeks ago, with worsening vision at night and hearing loss bilaterally. The patient has a number of myoclonic jerks as you arrive. On examination, his heart sounds are unremarkable but you notice a tachycardia at 140 and regular. The ECG is shown below:



The patient is uncooperative to further neurological examination but you notice sluggishly reactive pupils of equal size. His mother reports that he has been educated in a special needs school for the past 5 years but had been attending the local primary school until aged 9, when he dropped further behind than his peers.

Which investigation produces the underlying diagnosis?

A. MRI
B. Lumbar puncture
C. Electroencephalogram (EEG)
D. Cardiac electrophysiology studies
E. Muscle biopsy

E. Muscle biopsy

The patient is a young male presenting with cognitive impairment developing after a period of normal development, seizures, myoclonic jerks, Wolff-Parkinson-White syndrome and worsening vision (consistent with optic atrophy). The unifying diagnosis is that of myoclonic epilepsy with ragged red fibres (MERRF), which is a mitochondrial DNA disorder diagnosed by ragged red fibres on muscle biopsy.

Question 37 of 189

A 61-year-old male presents with his sixth episode of binocular visual phenomenon over the past 14 months. He describes these episodes as 'lights' and 'white dots' in his field of vision. He denies any limb or facial weakness or sensory loss. He denies having a headache. He is an active smoker, with a 60 pack year smoking history and has known hypertension on ramipril 5mg OD. Your neurological exam is unremarkable; CT head demonstrates no acute infarct or haemorrhage. MRI head is unremarkable.

What is the most likely diagnosis?

A. Posterior circulation transient ischaemic attacks (TIAs)
B. Posterior circulation strokes
C. Acephalgic migraine
D. Multiple sclerosis
E. Occipital space occupying lesion

C. Acephalgic migraine

The patient gives a strong history of positive visual symptoms such as flashing lights and dots, with no negative such as weakness or sensory loss. This would be far more suggestive of a migrainous syndrome or primary retinal pathology than cerebrovascular insufficiency, where negative symptoms would be prominent. In addition, a diagnosis of TIA or stroke should be suspicious in any patient with repeated stereotyped, similar symptoms over a long period of time. It is unlikely any patient will present with recurrent multiple TIAs without stroke over a 14 month period.

Acephalgic migraine is a frequently missed diagnosis of exclusion, where patients experience auras without a headache. Up to 38% of migraineurs experience migraine attacks without and with a headache, while 4% of migraineurs exclusively experience migraines without headaches .

Question 38 of 189

A 58-year-old female is brought into hospital by her husband with recurrent falls associated with cognitive decline noted by her husband over the past 8 weeks. The patient's husband reports a possible cough and cold about 3 months ago, during which she took 2 days off work as a personal assistant to a FTSE 100 company CEO but otherwise has no significant past medical history, is a non-smoker and drinks minimal alcohol.

On examination, she is alert and orientated to time and place but appears easily startled every time you start a sentence. You note significant bilateral finger-nose and heel-shin dysmetria, mild postural tremor and mild speech slurring. The remainder of her neurological examination was unremarkable.

Her blood tests are as follows:

Hb	124 g/l
Platelets	282
WBC	4.8
Na	143 mmol/l
K	4.7 mmol/l
Urea	6.7 mmol/l
Creatinine	86 µmol/l
CRP	3 mg/l

TSH	8.0 mu/l
Free T4	12.0 pmol/l
Anti-TPO	negative
HIV	negative
Glucose	6.5 mmol/l

A lumbar puncture was performed with results as follows:

WCC	<1 /mm³
RBC	28 /mm³
Protein	0.71 g/l
Glucose	3.4 mmol/l
Culture	no organisms grown
Viral PCR	awaited
Cytology	awaited
14-3-3	positive

An EEG demonstrated brief periodic spikes but these were not correlated with any seizure activity clinically. An MRI head demonstrated no parenchymal abnormalities except mildly increased signal in the cortical sulci.

What is the most likely diagnosis?

A. Creutzfeldt-Jakob disease
B. Viral encephalitis
C. Acute disseminated encephalomyelitis (ADEM)
D. Progressive multifocal leucoencephalopathy (PML)
E. Hashimoto's encephalopathy

A. Creutzfeldt-Jakob disease

This middle aged female demonstrates a number of worrying features suggestive of Creutzfeld Jakob disease (CJD): rapid cognitive decline, myoclonus, extrapyramidal signs (ataxia), startle response, positive 14-3-3 on CSF and periodic spiking on EEG. The MRI findings likely represent 'cortical ribboning', with high signal on the cortical sulci surfaces, which along with increased signal in putamen and caudate head, is suggestive of CJD.

She is also in the expected age group for CJD, typically presenting between 57 and 62 years old.

A number of features in the history and investigations are red herrings: it is unusual for a viral encephalitis, related to a probable viral respiratory tract infection, to be indolent for 8 weeks and continuing to progress in a non-immunosuppressed patient. The MRI findings and time course are also not suggestive of ADEM, which produces many focal areas of central demyelination within 1 to 3 weeks of a viral illness.

PML results in central demyelinating and is caused by activation of JC papovavirus in immunosuppressed patients, typically HIV positive individuals, and hence unexpected in this patient. The raised TSH likely represents subclinical hypothyroidism in this patient: while Hashimoto's encephalopathy can produce personality change, aggressive and disorientation, myoclonus and ataxia, 14-3-3 is typically negative in CSF with positive anti-TPO antibodies. Adiagnosis of CJD is thus most likely diagnosis, with the patient progressing along a typically fatal clinical course.

Question 39 of 189

A 21-year-old Caucasian female presents to eye casualty with an episode of sudden onset blurred vision in her left eye. She reports noticing it about 10 days ago and reports progressive deterioration associated with pain on eye movement until her presentation today. There is a vague history of a possible 'cough and cold' about 3 weeks ago but she has reported no other recent illnesses. She has no past medical history, is on the oral contraceptive pill and has no other medications. There is no smoking, alcohol or recreational drug history.

On examination, you note visual acuity and colour vision on Ishihara plates to be 6/6 and 17/17 plates in her right eye, 6/24 and 0/17 plates in her left eye. A left relative afferent papillary defect was demonstrated, fundoscopy was unremarkable. Examination of her other cranial nerves and limbs were unremarkable. Her blood tests were unremarkable and she has declined a lumbar puncture. An MRI head and whole spine demonstrates no abnormalities except for a swollen left optic nerve is visualised.

What is the most appropriate treatment?

A. No treatment, reassure patient vision will resolve
B. Oral prednisolone
C. Intravenous methylprednisolone
D. Intravenous immunoglobulin
E. Interferon beta

Not answered in the book

Question 40 of 189

A 32-year-old female patient presents with 48 hours of new onset vertiginous symptoms and speech slurring. She has no past medical history and is a non-smoker. On examination, you notice a left partial ptosis and miosis. Finger-nose coordination is impaired with her left arm and she also has reduced sensation to your cold tuning fork on her left face, right arm and right leg.

What is the most likely diagnosis?

A. Bilateral cerebellar infarcts
B. Right middle cerebral artery ischaemic infarct
C. Left middle cerebral artery ischaemic infarct
D. Lateral medullar brainstem infarct
E. Left basal ganglia haemorrhage

D. Lateral medullar brainstem infarct

This patient describes almost all the features of lateral medullary syndrome, classically known as Wallenberg's syndrome, consisting of ipsilateral cerebellar ataxia (lesion at the inferior cerebellar peduncle), ipsilateral loss of facial pain and temperature (ipsilateral trigeminal nucleus), vertigo and nystagmus (vestibular nucleus) and Horners syndrome (first order sympathetic chain neurones). In such a young patient without cardiovascular risk factors, vertebral artery dissection would be the most likely cause of the posterior circulation infarct, likely secondary to sudden neck twisting or extension from recent trauma, classically following a whiplash injury.

Question 41 of 189

A 16-year-old male is referred to your clinic after reporting feeling increasingly tired at school over the past 2 months. His parents are similarly concerned that their son seems to be relying on large volumes of Lucozade to keep himself alert during this stressful period of secondary school examinations. He reports no headaches. The patient was born following a normal pregnancy and development until he was 12 years old, when he underwent chemotherapy and radiotherapy treatment for an optic chiasm glioma, forcing him to miss one academic year of school. On completion of his treatment, he returned to school and has been achieving good grades. There is no other past medical or family history.

On examination, the patient is short for his age (at 2nd centile) and you note an absence of facial hair or other secondary sexual characteristics. He is thin with a BMI of 13.7 kg/m^2. Chest, cardiovascular and abdominal examinations are unremarkable. There are no skin lesions. Neurological examination including cranial nerve is unremarkable. His initial blood tests are as follows:

Hb	142 g/l
MCV	89 fl
Platelets	410 * 10 /l
WBC	7.4 * 10 /l
Na	139 mmol/l
K	4.6 mmol/l
Urea	5.1 mmol/l
Creatinine	44µmol/l
FSH	low
ACTH	low
TSH	0.13 mu/

What is the most likely diagnosis?

A. Cranial radiation injury
B. Glioma recurrence in optic chiasm
C. Klinefelter's syndrome
D. Turner's syndrome
E. Haemochromatosis

A. Cranial radiation injury

A combination of clinical features and pituitary function tests reveal panhypopituitarism. Delayed puberty does not appear to be gonadal but instead central in cause. While haemochromatosis may be a cause of panhypopituitarism, other features such as skin pigmentation, liver cirrhosis, arthralgia and type 1 diabetes mellitus would be expected. Turner's syndrome appears in females only due to an XO karyotype. Klinefelter's can cause delayed puberty secondary to a failure of Leydig cell development but would be associated typically with clinical features such as hypothyroidism, diabetes mellitus, with increased pituitary hormone release in response to gonadal dysfunction.

The location of the optic chiasm adjacent to the hypothalamic-pituitary structures and previous medical treatment should raise suspicion of cranial radiation injury, common in childhood survivors of intracranial cancers treated with radiotherapy. There is little to suggest recurrence of the tumour, which would typically present with proptosis and visual disturbance.

Question 42 of 189

An 18-year-old man presents to your follow-up clinic after the first episode of generalised tonic-clonic seizures, witnessed by his mother lasting for 4 minutes, involving all 4 limbs, before spontaneously terminating. He was initially referred to a first fit clinic and underwent an EEG and MRI, neither of which demonstrated any significant abnormalities. He has now returned to discuss his results and further treatment. The patient and his family realise that there are many ways to manage seizures and are happy to take your recommendation.

What is the most appropriate management?

A. No treatment
B. Sodium valproate
C. Lamotrigine
D. Levetiracetam
E. Carbamazepine

A. No treatment

Treatment is not usually recommended after a single seizure, as this may have been a provoked seizure instead of the first presentation of epilepsy. One prospective, but methodologically flawed, study demonstrated a 14% risk of seizure recurrent after a single, unprovoked seizure at 1 year . However, the risks significant increase after a patient has had two seizures, rising to 73% recurrent rate at 4 years . It is therefore accepted that antiepileptic treatments should only be commenced after 2 seizures, except if the risks of a further seizure is unacceptable to the patient, an underlying structural defect is proven to be the likely cause of the seizure or if the EEG demonstrated epileptiform activity.

Question 43 of 189

A 16-year-old boy is brought into the emergency department by his mother after an episode of unresponsiveness while walking, lasting transiently for seconds before fully resolving. He has no recollection of these episodes. He was diagnosed with absence seizures aged 11 and was previously prescribed anti-epileptics. These had been gradually weaned off two years ago by his neurologists following a 3-year seizure-free period.

Over the past 48 hours, he has now had 4 of his typical absences. You decide to prescribe an antiepileptic.

Which antiepileptic should be avoided?

A. Carbamazepine
B. Sodium valproate
C. Lamotrigine
D. Levetiracetam
E. No contraindications

A. Carbamazepine

Classically, carbamazepine can aggravate juvenile myoclonic epilepsy and absence seizures . In general, carbamazepine should always be avoided in patients where the type of seizure is uncertain.

It is otherwise a useful AED in the treatment of partial or secondary generalised seizures.

Question 44 of 189

A 72-year-old male is brought into the emergency department by ambulance after a third episode of limb jerking in 72 hours was witnessed by his wife. As you arrive, the patient becomes increasingly unresponsive and you witness small amplitude jerking of all four limbs. While the resus nurse obtains midazolam two minutes into his seizure, you note his eyes are closed tight and it is not possible to forcefully open them in order to check papillary reflexes. The amplitude of his limb jerking also become increasingly large. You note blood on his tongue and urinary incontinence. He does not suffer head or limb injury. His seizure spontaneously terminate before any medication is administered about 6 minutes after its onset. He becomes increasingly responsive about 2 minutes after the end of limb jerking and appears tearful. He is extremely apologetic for the fuss he has caused.

What is the most likely diagnosis?

A. Partial seizure
B. Absence seizure
C. Secondary generalised seizure
D. Pseudoseizure
E. Myoclonic seizure

D. Pseudoseizure

A number of features in this description of the seizure indicate the possibility of pseudoseizure. The patient's eyes are closed to resistance and he has a very short post-ictal state. He also appears to become alert and orientated in speech very quickly afterwards. Patients with pseudoseizures often have accompanying underlying psychiatric concerns and can be tearful around the time of the seizure. In addition, prolonged jerking of limbs, of increasing amplitude (crescendo features) should make you suspicious. A number of features should also be noted: firstly, patients of any age can present with pseudoseizures. Secondly, features such as tongue biting and urinary incontinence are not absolute features of an organic seizure, they are often present in pseudoseizures.

Question 45 of 189

You receive a call from a GP in the community. A 65-year-old female patient who was diagnosed with generalised myasthenia gravis six years ago and last reviewed in neurology outpatients 6 weeks ago reports no improvements in her neck weakness, voice weakness and fatigue. In the neurology clinic, her dose of pyridostigmine was increased from 90mg QDS to 120mg QDS. She does not appear acutely unwell but complains that her life is significantly affected by her symptoms. She has no other past medical history. On examination by her GP, she has no respiratory distress and able to swallow salivary secretions normally.

What is your advice?

A. Increase pyridostigmine to 150mg QDS
B. Start prednisolone 60mg in community, neurology to follow up in 8 weeks as outpatient
C. Start azathioprine in community, neurology to follow up in 8 weeks as outpatient
D. Admit to hospital, start oral prednisolone in hospital
E. Admit to hospital, start intravenous immunoglobulin in hospital

D. Admit to hospital, start oral prednisolone in hospital

The patient remains symptomatic despite being increased onto the maximum dose of pyridostigmine. The second decision is whether the patient is acutely, severely unwell, in which case rapid therapies such as IV Ig or plasmapheresis must be initiated, as per myasthenic crisis. In this case, she has no signs of respiratory distress or dysphagia, her symptoms are more of a case of not improved instead of rapid deterioration. Therefore, the patient does not require rapid therapy. However, she does require immunotherapy initiation. As a rule of thumb, patients who remain symptomatic despite 60mg QDS of pyridostigmine should be considered for immunotherapy.

The modality of immunotherapy must be individualised to the patient, as each drug has their own contraindications. In younger patients with no concerns regarding blood sugars, oral prednisolone is reasonable. Those with liver disease should avoid azathioprine, renal disease patients should avoid ciclosporin while those with haematological abnormalities should avoid mycophenolate and azathioprine.

However, starting of prednisolone, particularly at high doses, have been classically described to cause a paradoxical reaction in half of all patients, resulting in a transient deterioration before improvements are observed. Up to 10% of patients have been reported to have sufficiently severe respiratory failure requiring mechanical ventilation take place in a monitored hospital setting.

Question 46 of 189

A 42-year-old male presents with double vision and weakness in his fingers, which he noticed when he repeatedly dropped his pen whilst trying to write at work. His symptoms appear to have onset over the past few days. He has no past medical history other than a recent episode of diarrhoea and vomiting about two weeks ago. He reports no limb weakness and no sensory loss. He denies any back pain or palpitations. On examination, there is a 3/5 weakness in finger flexion, finger extension and wrist extension in both hands, with no fatigability. No reflexes present in the lower or upper limbs. There is no ptosis or nystagmus but reduced eye movements in all directions. His finger-nose test demonstrates reduced coordination bilaterally and the patient has too little confidence to walk.

Which investigation is most likely to be diagnostic?

A. Anti-GM1 antibody
B. Anti-GQ1b antibody
C. MRI brain and whole spine
D. Anti-MUsK (muscle specific kinase) antibody
E. Anti-acetylcholine receptor antibody

B. Anti-GQ1b antibody

The patient has presented with a classic triad of ophthalmoplegia, ataxia and areflexia, which should suggest Miller-Fisher syndrome, an acute onset demyelinating peripheral neuropathy recognised as a variant of Guillain-Barre Syndrome. Anti-GQ1b antibody is present in between 85-90% of patients with Miller-Fisher syndrome .

Key differentials of MFS include myasthenia gravis, brainstem infarcts and brainstem encephalitis. In this case, the absence of fatigability and nystagmus rule against neuromuscular junction disorders and brainstem events respectively.

Question 47 of 189

A 50-year-old Caucasian female on holiday from Australia presents to the walk-in urgent care centre with four-day history of left-sided temporal headache, which is persistent and of gradual onset. In addition, she complains of double vision as well. She is known to have migraines but is relatively well-controlled. She is normally independent with no significant family history. She takes no regular medications except the oral contraceptive pill. On examination, you note a left sided loss of the afferent papillary reflex. She also has a loss of vertical gaze and is unable to adduct her left eye. She has a reduced sensation to light touch on the left forehead and cheek, not crossing the midline.

What is the most likely diagnosis?

A. Left space-occupying lesions
B. Multiple sclerosis
C. Right MCA territory ischaemic infarct
D. Cavernous sinus thrombosis
E. Migraine with aura

D. Cavernous sinus thrombosis

The patient describes a clear history of loss of her 3rd cranial nerve, with the possible involvement of the left 4th, with V1 and V2 branches of the left trigeminal nerve. The only structure involving all these cranial nerves (and classically with the sympathetic postganglionic neuron) is the cavernous sinus. The patient has a number of risk factors for venous thrombotic disease, including recent long-haul flight and the oral contraceptive pill. The most likely causes are dehydration and VTE disease risk factors, but a carotid artery aneurysm should also be considered.

Question 48 of 189

A 78-year-old male presents to the hyperacute stroke unit with a sudden onset left sided hemiparesis and which is subsequently demonstrated to represent an acute ischaemic infarct in the right middle cerebral artery territory with no haemorrhagic transformation. He was not thrombolysed due to presentation being outside the time window. As part of his stroke investigations, echocardiogram demonstrates no mural thrombus or regional wall abnormalities and an ejection fraction of 70%. The 24 hour tape recorded no arrhythmias. Carotid Dopplers demonstrate 40% stenosis in the right internal carotid artery, 55% stenosis in the left internal carotid artery. Blood pressure measured 125/75mmHg. He takes simvastatin 40mg nocte and has no known drug allergies.

What would be the optimal treatment?

A. Refer to vascular surgery for right carotid endarectomy
B. Refer to vascular surgery for left carotid endarectomy
C. 300mg oral aspirin for 14 days, then clopidogrel 75mg
D. Subcutaneous low molecular weight heparin, then warfarin loading
E. Perindopril 4mg OD

C. 300mg oral aspirin for 14 days, then clopidogrel 75mg

Carotid endarectomy (CEA) is not indicated for either internal carotid artery in this case. The present NICE guideline recommends surgical intervention if the symptomatic side demonstrates greater than 70% stenosis and to be discussed for CEA if 50-69% stenosis . The management of asymptomatic stenosis is controversial: minimal but significant benefit is proven only for patients with greater than 60% stenosis and if the benefits outweigh the risk of stroke or death post CEA, which currently stands at 3%. NICE recommends consideration for asymptomatic CEA if stenosis is greater than 60% only. In this case, neither internal carotid stenosis fits the criteria for surgical intervention. Instead, best medical management with antiplatelet therapy is the treatment of choice. The use of anticoagulation beyond cardioembolic disease is controversial and not routinely used. The patients blood pressure is within the target range. The patient is already on a statin.

Beware when interpreting carotid stenosis measurements: two major trials of CEA for carotid endarectomy measured the degree of stenosis differently: ECST compared the stenotic diameter to the probable original diameter at the carotid bulb, the clearest benefit was for patients with greater than 80% stenosis . NASCET compared residual diameter to the lumen directly distal to the stenosis, the greatest benefit of CEA was for patients with greater than 70% stenosis . Simplistically, any patient with greater than 70% to 99% symptomatic stenosis would benefit significantly from CEA, patients with 50 to 69% stenosis would also benefit from CEA but to a lesser degree and only if performed within 2 weeks of the last episode.

Question 49 of 189

A 64-year-old gentleman presented to the neurology clinic for review. He had a history of motor neuron disease which had been progressing over the past five years. He had significant weakness in all of his limbs and was dependent on a wheelchair. He took baclofen for relief of muscle spasms, and amitriptyline to reduce salivation but was otherwise not on any medication. He mentions that in addition to his weakness continuing to progress he has been suffering from headaches in the morning. These were generalised and described as throbbing in nature. He also reported not feeling refreshed after sleep.

On examination, there is generalised muscle wasting, with fasciculations in all limbs. Reflexes were absent, other than bilateral extensor plantar responses. Eye movements and fundoscopy are normal. Palatal movements were normal, and there was no significant dysphagia on swallow test.

Which intervention is most likely to confer the greatest survival benefit in this patient?

A. Riluzole
B. Feeding via percutaneous endoscopic gastrostomy
C. Non-invasive ventilation
D. Home oxygen therapy
E. Prophylactic antibiotics

C. Non-invasive ventilation

The morning headaches and lack of feeling refreshed after sleep are suggestive of carbon dioxide retention due to respiratory impairment in patients with motor neuron disease. Non-invasive ventilation improves quality of life and confers a survival benefit in patients with respiratory impairment in motor neuron disease.

Riluzole also offers a modest survival benefit in motor neuron disease (it is the only drug known to do so), prolonging life expectancy by on average two to four months. Given there is no evidence of dysphagia feeding via a percutaneous endoscopic gastrostomy is not indicated in this patient. Home oxygen therapy and prophylactic antibiotics are not routinely used in motor neuron disease.

Question 50 of 189

A 42-year-old female presented with a 6-month history of weakness affecting her upper limbs. She found that this was worse at the end of the day, particularly if she had been doing a lot of lifting. She also reported occasional difficulty in swallowing at the end of the day, with drinks causing her to cough. She had been prescribed pyridostigmine, but she stopped taking this as she had not noticed any benefit.

On examination, there was a mild partial ptosis, and a slight facial droop bilaterally. Extra-ocular movements were normal. Power in shoulder abduction was grade 4/5 bilaterally, but was reduced to 3/5 after repeatedly abducting and adducting the shoulder 20 times. Tone and sensation were normal.

Initial investigation results are shown below:

- Nerve conduction studies: Decremental response to repetitive nerve stimulation
- Single-fibre electromyography: Increased jitter
- Anti-Jo antibodies: Negative
- Anti-Mi2 antibodies: Negative
- Anti-acetylcholine receptor antibodies: Negative

Which of the following immunological tests is most likely to contribute to the diagnosis?

A. Anti-muscle-specific tyrosine kinase(MuSK) antibodies
B. Anti-N-methyl D-aspartate (NMDA) receptor antibodies
C. Anti-glutamic acid decarboxylase (GAD) antibodies
D. Anti-acetylcholine receptor antibody
E. Anti-Hu antibodies

A. Anti-muscle-specific tyrosine kinase(MuSK) antibodies

This patient presents with weakness affecting the upper limbs, eyelids, facial muscles and pharynx, which demonstrates fatiguability. This is a characteristic description of myasthenia gravis, and this is supported by the electrophysiological tests. The majority of patients with this condition are positive for anti-acetylcholine receptor antibodies, which are present in about 85%. In the remaining patients, about 40% are positive for anti-muscle-specific tyrosine kinase antibodies. In anti-acetylcholine receptor negative cases, response to acetylcholinesterase inhibitors (such aspyridostigmine) tends to be worse.

Anti-aquaporin antibodies are associated with neuromyelitis optica, anti-N-methyl D-aspartate receptor antibodies are associated with autoimmune encephalitis, anti-glutamic acid decarboxylase antibodies are associated with stiff man syndrome, and anti-Hu antibodies are associated with a paraneoplastic sensory neuropathy and encephalitis.

Question 51 of 189

You are called to a general medical ward during the night to review 48 year female who has become acutely agitated and confused. She came into hospital two days ago with a suspected urinary tract infection and has been making uneventful progress. She has a past history of traumatic spinal injury at level C5 three years ago with resulting spastic quadraparesis. On your arrival her blood pressure is 220/105 mmHg, heart rate is 55/min, and she is flushed and diaphoretic.

Of the following, what additional feature might you expect to find?

A. Palpable bladder
B. Ejection systolic murmur in the pulmonary area
C. Flushing of the skin in the trunk and legs R
D. elative afferent pupillary defect
E. Complete heart block

A. Palpable bladder

This question is describing a case of autonomic dysreflexia. Of the options given, palpation of a distended bladder is likely and also diagnostic. Pulmonary murmurs can be a feature of the carcinoid syndrome. A relative afferent pupillary defect is seen with optic nerve pathology such as optic neuritis. Flushing of skin above the cord lesion is common due to parasympathetic feedback causing vasodilation but of course anything below the cord lesion remains vasoconstricted.

Question 52 of 189

A 53-year-old accountant presents with two weeks of a constant headache with two seizures in the last 24 hours. His family say that he has changed in character and seems more aggressive. They also say that he appears weaker, is eating less and has 2 lbs of weight over the past week.

He is known to be HIV positive and had an anterior resection for rectal carcinoma 3 years ago and a recent endoscopy shows gastric polyps. He is currently well but has suffered from multiple episodes of sinusitis and is taking a four-week course of oral antibiotics.

On examination, he has reduced power in the left upper limb across all muscular groups but preserved sensation. Reflexes are reduced on the left side. In the left lower limb, power is mildly reduced and there is an upgoing plantar with normal sensation. He has a Glasgow Coma Score of 13 and he is drowsy. Pupils are equal and reactive but there is papilloedema. His observations show a heart rate of 96 beats per minute, blood pressure 110/98 mmHg, temperature 38.0'C and respiratory rate 18 per minute with saturations of 98%.

Na	137 mmol/l
K	4.3 mmol/l
Urea	3.5 mmol/l
Creatinine	40 µmol/l
Hb	100 g/l
Platelets	378* 10 /l
WBC	12.3* 10 /l
Blood cultures	pending
CT head	requested

What is the likely diagnosis?

A. Tertiary syphilis
B. Lymphoma
C. Toxoplasmosis
D. Cerebral abscess
E. Glioma

D. Cerebral abscess

Features of raised intracranial pressure and fever points towards a cerebral abscess

There is evidence of raised intracranial pressure that is gradual in onset. Subarachnoid presents with a severe headache which has not occurred here. Tertiary syphilis is rare and there are no specific signs pointing towards this diagnosis. Toxoplasmosis is possible given he is HIV positive but there are no additional features pointing towards this. Of cerebral abscess and glioma, the presence of fever makes the former likely and the abscess is likely secondary bacterial spread from sinusitis. A glioma is possible given the weight loss and previous malignancy but would usually present over a longer period of time.

Question 53 of 189

A 23-year-old man with known Von Hippel Lindau syndrome sees his GP to routinely review his mood. He has been well previously but has known retinal haemangiomas. He is taking venlafaxine and has improved in his mood which was damaged from a recent relationship break down. He does continue to get panic attacks where he develops palpitations and feels clammy and is using mindfulness to control these.

On examination, he is well and alert. His blood pressure is 170/110mmHg. He has 6/6 visual acuity bilaterally and there are no haemorrhages or cotton wool spots on fundoscopy. His abdomen is soft and there are no palpable masses.

Na	134 mmol/l
K	4.1 mmol/l
HCO3	25mmol/l
Urea	6.7 mmol/l
Creatinine	87 µmol/l
TSH	3.2 mU/l (range 0-4)
ECG	large QRS complexes consistent with left ventricular hypertrophy

What is the likely cause of his presentation?

A. Venlafaxine
B. Phaeochromocytoma
C. Renal artery stenosis
D. Depression/anxiety
E. Liddle's syndrome

B. Phaeochromocytoma

Hypertension in Von Hippel Lindau -> phaeochromocytoma

This gentleman has presented with hypertension and episodes of sweating and palpitations. He is usually well and is on venlafaxine which can cause hypertension but not the other symptoms. Renal artery stenosis would usually present with deranged urea and electrolytes and is rare in this age group. Liddle's syndrome presents with a hypernatraemia, hypokalaemia metabolic alkalosis which is not seen here. Depression/anxiety can cause BP fluctuations but is a diagnosis of exclusion. Phaeochromocytoma would explain these symptoms and also is more common in Von Hippel Lindau so is the answer.

Question 54 of 189

A 70-year-old man with hypertension and previous myocardial infarction 3 years ago presents to his GP with a 2 day history of a painful vesicular rash over his left chest and back. It is well demarcated and confined to the T6 dermatology. His GP diagnoses shingles and prescribes pain relief. His rash resolves gradually over the next 2 weeks.

Four weeks later the man develops chest pain on the left side which is stabbing in shooting in nature. He attends the Emergency Department. On examination his blood pressure is heart rate is 50/min and blood pressure is 154/96 mmHg. His oxygen saturations on room air 98%.

His investigations results were as follows: Chest x-ray: No abnormalities.

ECG: 50/min. PR interval 230 milliseconds. QRS interval 110 milliseconds. 5 millimetre Q waves in the inferior leads. Normal T wave and ST segments.

His d-dimer and 12 hour troponin are both normal.

Which analgesic agent is contraindicated?

A. Amitriptyline
B. Duloxetine
C. Gabapentin
D. Oxycodone
E. Pregabalin

A. Amitriptyline

This gentleman has post-herpetic neuralgia causing his chest pain.

He has a history of heart attack and his ECG shows evidence of inferior myocardial infarction in the territory of the right coronary artery. This has resulted in a first degree heart block as evidenced by the long PR interval.

NICE Guidance on Neuropathic Pain (CG173) advocates a choice of amitriptyline, duloxetine, 61% 18% 5% 12% 4%gabapentin or pregabalin. According to the BNF amitriptyline is contraindicated in arrhythmias particularly heart block, as in this case. It is also contraindicated in the recovery period following myocardial infarction, though this gentleman is outside of this.

Question 55 of 189

A 25-year-old F1 driver complains of neck pain following a race. Three days later he presents with left sided facial numbness as well as numbness across the right upper and lower limb. Positive findings on examination are the loss of pain and temperature sensation on the left side of the face in all trigeminal distributed areas. There is also the loss of pain and temperature sensation in the right upper and lower limbs. The left pupil is much smaller than the right and there is a partial ptosis on the left. Eye movements are normal. Power, coordination, and reflexes are all normal.

What is the gold standard brain-imaging method used in order to establish the primary diagnosis?

A. Plain MRI brain
B. Carotid artery doppler ultrasound scan
C. Plain CT scan brain
D. CT angiogram head and neck
E. Electroencephalogram (EEG)

D. CT angiogram head and neck

This gentleman has a vertebral artery dissection following neck hyperflexion during the F1 car race. It is not uncommon for young people to get these sorts of strokes, particularly young sporting people such as in the sad case of the cricketer Phil Hughes.

The vertebral artery has a tributary (the posterior inferior cerebellar artery -PICA) to the medulla and in this case, the F1 driver has therefore developed a left sided lateral medullary syndrome (aka Wallenberg syndrome). Below are the features of this syndrome:

Cerebellar features: ipsilateral limb ataxia, vertigo, nystagmus to the side of the lesion. These are due to taking out the ipsilateral spinocerebellar tracts within the medulla.

Brain stem features:

dizziness and vomiting (taken out the vestibular and vagal nuclei) dysphagia and dysarthria (again this represents the vagal nuclei being taken out) ipsilateral Horner's syndrome (the sympathetic tract has been taken out on that side) ipsilateral facial pain and temperature sensory loss (ipsilateral trigeminal tract taken out) ipsilateral pharyngeal and laryngeal paralysis from cranial glossopharyngeal and vagus palsiesIMPORTANTLY, contralateral pain and temperature sensory loss (spinothalamic tract taken out. Remember, this crossed over well before the medulla, down at the level of the cord itself).

With all brainstem strokes, the side of the lesion is the side of the cranial nerve sign. Ie...loss of sensation on the left side of the face, for example, but with a loss of sensation of the right side of the body...still tells you that the lesion is on the left because that is the side of the cranial nerve sign. In a left-sided 'ventral pontine syndrome' (Millard Gubler), for example, you get ipsilateral facial weakness (the left facial nerve has gone -ie the cranial nerve sign) with contralateral body hemiparesis (the right corticospinal tract has been taken out).

The management of vertebral artery dissection is actually anticoagulation (once bleed excluded by scans). This is intended to prevent further thromboembolic complications (here, for example, our patient has had a subsequent brainstem stroke). Note that there are actually no randomised controlled trials to support the use of anticoagulation here, but its what people do. Antiplatelet therapy is a reasonable option to consider in patients who are suspected of suffering from a vertebral artery dissection while awaiting definitive investigations. So here, if a plain CT excluded bleed, then you clearly have a stroke and aspirin would be a reasonable choice whilst you wait for you CT angiogram. Note plain CT would not establish the diagnosis. In order to visualise the dissected vessel, you need an angiogram.

Question 56 of 189

A 29-year-old man has developed a gradual onset of bilateral leg weakness over the last 24 months such that he now needs to walk with crutches. He is also intermittently incontinent of urine. He will also often fall over in the dark because he feels his balance is worse then. He has never noticed any other symptoms prior to these. He has no significant past medical conditions. He works as an accountant. He went travelling for some years in his early 20's to the Carribean, Japan and Africa. He admits occasional intravenous drug use whilst travelling and getting a tattoo. He also has casual sexual contact with sex workers occasionally whilst travelling and does not recall using barrier contraception. On examination, you find hyperreflexia bilaterally in the legs with upgoing plantar responses. He has loss of vibration and joint position sense in the legs. Legs are 3/5 power in the knee and plantar extenders and 4/5 in the flexors. He has a stomping gait. Routine blood tests plus HIV screening come back unremarkable. MRI brain and whole spine show areas of demyelination in the lumbar spine.

Which test would you do to confirm the most likely suspected cause of his symptoms?

A. Serm JC virus levels
B. Serum and CSF HTLV-1 antibody levels
C. Syphilis serology
D. Hepatitis B serology
E. Paired serum and cerebrospinal fluid (CSF) oligoclonal band levels

B. Serum and CSF HTLV-1 antibody levels

The diagnosis is tropical spastic paraparesis (TSP).

This is otherwise called HTLV-1 Associated Myelopathy (HAM), describing how this demyelinating condition is caused by infection with HTLV-1. HTLV-I is a retrovirus endemic in southern Japan, equatorial Africa and South America. Transmission occurs through sexual or other intimate contacts, intrauterine, breastfeeding, sharing of needles by drug users, or blood transfusion from infected persons. This patient has many of these risk factors from his time travelling.

The incubation period between infection and symptomatic disease may be a few months up to decades and actually, the majority of infected individuals remain lifelong asymptomatic carriers. It is not clear what determines whether or not one becomes symptomatic but the key is suspected to be bigger viral loads resulting in progression to symptoms.

The presentation is with progressive upper motor neurone symptoms and signs particularly confined to the lower limbs (see below for differentials of such a presentation.

The key to knowing which of these differentials is the likely cause here is understanding the risk factors and ordering the correct tests (e.g. not missing out an HIV test); as the signs and symptoms are fairly similar in all of these upper motor neurone conditions.

Question 57 of 189

A 35-year-old lady with no significant past medical history presents to you with lethargy and fatigue over several years, often finding she does not sleep well at night. She complains of a sensation of discomfort in her lower extremities at rest, particularly worse when she is trying to fall asleep. She also describes an abnormal crawling and itching sensation below the knees and often walking will relieve her symptoms. There is no history of pain or nigh time snoring. Clinical examination and routine blood investigations are unremarkable. She tells you that over the years her general practitioner has ordered several tests on her that have all been normal, including brain imaging, thyroid function, and monitoring of her haemoglobin levels. She is not on any medications. She is now struggling to do her job as a teacher and is now working only part time because of the symptoms.

Considering the likely underlying diagnosis, which of the following would you use to try and alleviate her symptoms?

A. Lithium
B. Quinine
C. Pramipexole
D. Amitriptyline
E. Acetazolamide

C. Pramipexole

The diagnosis is restless leg syndrome (RLS). This is a neurologic movement disorder of the limbs that is often associated with a sleep complaint. This will often end up presenting in neurology clinics and it is important to know how to help such patients. RLS can lead to significant physical and emotional disability.

Specific DSM-5 criteria for RLS are as follows:

- an urge to move the legs that is usually accompanied by or occurs in response to uncomfortable and unpleasant sensations in the legs, characterized by all of the following: (1) the urge to move the legs begins or worsens during periods of rest or inactivity; (2) the urge is partially or totally relieved by movement; and (3) the urge to move legs is worse in the evening or at night than during the day or occurs only in the evening or at night.
- Symptoms occur at least 3 times per week and have persisted for at least 3 months.
- Symptoms cause significant distress or impairment in social, occupational, educational, academic, behavioural or other areas of functioning.
- The symptoms cannot be attributed to another mental disorder or medical condition (e.g., leg oedema, arthritis, leg cramps) or behavioural condition (e.g. positional discomfort, habitual foot tapping)
- The disturbance cannot be explained by the effects of a drug of abuse or medication

85% of patients with RLS have periodic movements of sleep, usually involving the legs (periodic leg movements of sleep [PLMS]). This is characterised by involuntary, forceful dorsiflexion of the foot lasting 0.5-5 seconds and occurring every 20-40 seconds throughout sleep. It is, therefore, worth taking a collateral history from a partner if available.

Investigations should include excluding iron deficiency (which may potentiate RLS) and you should order sleep studies to characterise the extent of sleep disturbance.

First line drug treatments are dopaminergic agents (eg, pramipexole, ropinirole, bromocriptine, levodopa-carbidopa, and rotigotine) or gabapentin/pregabalin.

Nonpharmacologic approaches include exercise, avoidance of caffeine, alcohol, and nicotine. Also important to try and stop medications that exacerbate RLS if possible, eg selective serotonin reuptake inhibitors (SSRIs), serotonin-norepinephrine reuptake inhibitors (SNRIs), diphenhydramine, and dopamine antagonists.

Question 58 of 189

A 75-year-old woman presents with a 24-hour history of gradual onset, severe 10/10, frontal headache associated with double vision. The history is otherwise unremarkable. On examination, she has a partial ptosis of the right eye and a pupil resting outwards and downwards with a dilated sluggishly reacting pupil. There is a failure of intorsion of the right eye. Neurological and physical examination are otherwise unremarkable. She has normal routine blood tests and a normal plain CT head.

What is the most important next investigation?

A. Lumbar puncture
B. CT angiogram cerebral vessels.
C. Carotid artery biopsy
D. Indomethacin trial
E. Single fibre EMG of the ocular muscles

B. CT angiogram cerebral vessels.

Presentation with a painful third nerve palsy is a neurosurgical emergency that normally presents to the medics as it may indicate an expanding posterior communicating artery aneurysm. Urgent CT angiogram of the cerebral vessels is required for diagnosis. Management is with surgical clipping. If this does not happen quickly, there is a risk of death from rupture or there may be permanent ptosis and eye movement deficit.

It is important to be able to differentiate between surgical causes of third nerve palsy (eg the above, or a tumour), and medical causes, eg diabetes or vasculitis. The surgical causes indicate an emergency whilst the medical causes are likely chronic conditions requiring optimisation of medical management. The parasympathetic fibres of this nerve are on the periphery of the nerve and the sympathetic fibres are in its centre. Therefore, a fixed dilated or sluggish pupil indicates that the parasympathetic fibres have been wiped out (eg by external compression from an aneurysm or tumour), leaving unopposed sympathetic tract flow within this nerve (dilating the pupil). The blood supply to the nerve is within its internal portion, meaning medical causes like diabetes or vasculitis wipe out the sympathetic portion of the nerve, leaving unopposed parasympathetic flow and consequently a constricted pupil.

The trochlear nerve sits right next to the oculomotor nerve, meaning if a structural cause is big enough, it will take the trochlear nerve out too. The trochlear nerve is responsible for intorsion of the eye (ie internal rotation in the plane of the face). So when you test for a 3rd nerve palsy, youshould also test for a 4th nerve palsy by asking the patient to follow your finger as you draw a half-circle from out to in (anti-clockwise): ie your 9'oclock to 3 o'clock. If the eye fails to follow, this is a failure of intorsion.

Question 59 of 189

A 75-year-old man presents with slowly progressive dyspnoea. There is no cough or chest pain. He is a non-smoker with a background of Parkinson's disease, rheumatoid arthritis, type 2 diabetes mellitus and atrial fibrillation. Peak flow investigations demonstrate a reduced Forced Vital Capacity (FVC), a Forced Expiratory Volume (FEV1):FVC ratio of 90%, and a low transfer factor for carbon monoxide (TLCO). High-resolution CT confirms ground-glass changes.

Which of his following medications is the most likely contributor to his lung-changes?

A. Azathioprine
B. Levodopa
C. Digoxin
D. Cabergoline
E. Exantide

D. Cabergoline

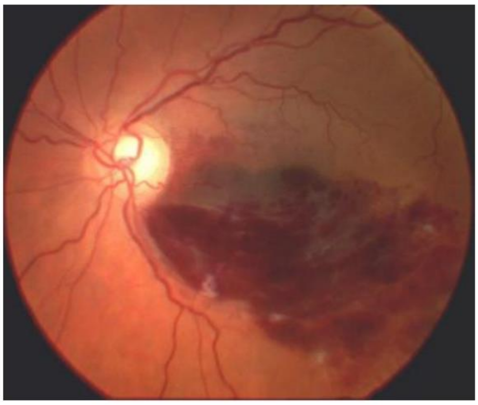
He has pulmonary fibrosis, probably related to rheumatoid arthritis and medication side-effects. Cabergoline and other ergot-derived dopamine-receptor agonists like bromocriptine and pergolide have more or less been phased out of use in Parkinson's disease now because of sideeffects including pulmonary, retroperitoneal, and pericardial fibrotic reactions. We tend to start now with the non-ergot-derived dopamine-receptor agonists including pramipexole, ropinirole, and rotigotine. Most patients eventually require levodopa. Monoamine-oxidase-B inhibitors such as rasagiline or selegiline are also used (alone or in combination with levodopa for end-of-dose fluctuations). Watch out for postural hypotension as a side effect anti-parkinsonian medication, particularly early on in the introduction of dopamine-receptor agonists. An apomorphine pump (given subcutaneously) can be very useful in the management of refractory PD, as can be deep brain stimulation (DBS).

Question 60 of 189

A 62-year-old woman presents to the emergency eye unit with a 1-day history of sudden loss of vision in her left eye. She denies pain or previous concerns with her vision. Her past medical history includes hypertension and type 2 diabetes for which she takes ramipril and metformin, respectively.

On examination, there is reduced visual acuity in her left eye and she is only able to make out hand movements. The visual acuity in her right arm is unchanged from 6/9.

Fundoscopic examination of her left eye is shown below:



What is the most likely diagnosis?

A. Diabetic retinopathy
B. Hypertensive retinopathy
C. Retinal artery occlusion
D. Retinal vein occlusion
E. Retinitis pigmentosa

D. Retinal vein occlusion

This patient has a retinal vein occlusion characterised by a sudden onset of painless loss of vision. Risk factors for retinal vein occlusion include hypertension and type 2 diabetes and are more common in the middle-aged. The fundoscopic examination shows dilated tortuous veins and intraretinal haemorrhage secondary to occlusion of the retinal vein. Other features that can be seen include cotton wool spots, dot and blot haemorrhages and macular oedema.

Hypertensive retinopathy is incorrect. Hypertensive retinopathy is typically associated with arteriolar narrowing, cotton wool spots, retinal haemorrhages and hard exudates. These are not seen in this fundoscopic examination, making the diagnosis unlikely.

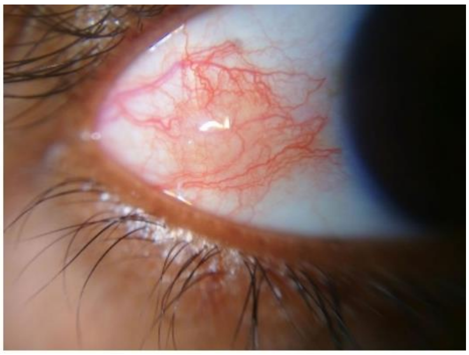
Diabetic retinopathy is incorrect. Diabetic retinopathy is a reasonable differential diagnosis given the past medical history of type 2 diabetes. However, it is unlikely to cause sudden onset visual loss. Rather, diabetic retinopathy causes gradual visual loss and can be staged from preproliferative, proliferative and maculopathy. Fundoscopic features include cotton wool spots, microaneurysms ane neovascularisation which, in turn, can lead to retinal detachment and vitreous haemorrhage.

Retinal artery occlusion is incorrect. Retinal artery occlusion is another common cause of sudden onset painless vision loss, frequently from embolic phenomenon. However, fundoscopic findings of retinal artery occlusion include a pale retina with a classic 'cherry-red spot' (the unaffected vascular choroid at the centre of the macula). Cotton wool spots may also be seen.

Retinitis pigmentosa is incorrect. Retinitis pigmentosa is an inherited disorder of the retina that is characterised by degeneration of the rod cells (responsible for night vision). In retinitis pigmentosa, there is bone-spicule deposition within the periphery of the retina. This is not seen here, nor is there a history of night blindness or reduced peripheral vision.

Question 61 of 189

A 27-year-old man presents to the general practitioner with a 1-day history of red eyes. He reports noticing them whilst brushing his teeth in the morning. He complains that his eyes are mildly sore but manageable without the need for analgesia. There is no history of itch and he otherwise feels well in himself. His past medical history includes ankylosing spondylitis.



On examination, his visual acuity is 6/6 bilaterally. His eyes are examined, as shown below:

Given the diagnosis, what is the recommended management for this patient?

A. Acetazolamide
B. Reassurance and NSAIDs
C. Topical aciclovir
D. Topical chloramphenicol
E. Topical corticosteroids

B. Reassurance and NSAIDs

This patient has a diagnosis of episcleritis. Episcleritis is the acute inflammation of the episcleral tissues and presents as an acute-onset red eye that may be mildly irritable but should not be painful or associated with vision loss. Examination reveals dilated episcleral vessels on the surface. Most cases are idiopathic, but risk factors include inflammatory bowel disease, seronegativespondyloarthropathies and vasculitides. The condition is self-limiting and no treatment is usually necessary. However, the use of NSAIDs is sometimes recommended for the active inflammation and treatment of any discomfort, and artificial tears may also be used for the relief of symptoms.

Acetazolamide is incorrect. Acetazolamide is a carbonic anhydrase inhibitor used in the treatment of acute angle closure glaucoma. Features of acute angle closure glaucoma include a unilateral painful red eye associated with photophobia, headache and vomiting. The pupil may be partially fixed and dilated with a hazy cornea. This patient's presentation is not in keeping with this diagnosis.

Topical aciclovir is incorrect. Topical aciclovir would be indicated for an acute herpetic infection of the eye. However, the mild symptoms that this patient is presenting with make an infective cause of his symptoms unlikely.

Topical chloramphenicol is incorrect. This would be the treatment of choice for a patient with bacterial conjunctivitis. Features of bacterial conjunctivitis include an itchy, watering and gritty red eye associated with blurred vision and purulent discharge with or without crusting of the eyelashes. This is not seen in this case, making conjunctivitis unlikely.

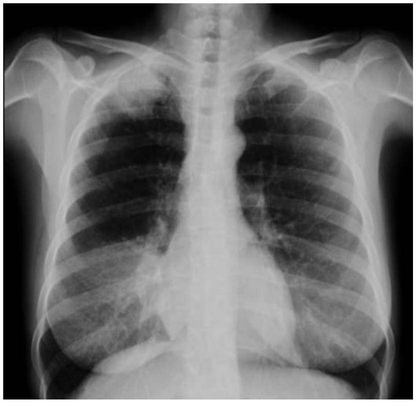
Topical corticosteroids is incorrect. This is used in the management of anterior uveitis. Patients with inflammatory bowel disease, such as this patient, are at an increased risk of anterior uveitis. However, symptoms typically include an acute red and painful eye with patients often complaining of photophobia. These symptoms are not given in this presentation. Furthermore, this patient's eye demonstrates injection of the episcleral vessels which is uncharacteristic of anterior uveitis.

Question 62 of 189

A 56-year-old woman is referred to the respiratory clinic with a 3-month history of shortness of breath and a dry cough. She was admitted to the hospital for community-acquired pneumonia 1 year ago but had recovered well with complete resolution of symptoms. Her past medical history includes hypertension and hyperlipidaemia for which she takes amlodipine, ramipril and simvastatin. She drinks 4-6 units of alcohol per week and smokes 25 cigarettes/per day.

On examination, her observations are within normal limits. There are vesicular breath sounds bilaterally.

A chest x-ray is requested, as shown below:



Given the diagnosis, what additional clinical finding is most likely to be present?

A. Erythema nodosum
B. Haematuria
C. Miosis
D. Pericardial friction rub
E. Purpura

C. Miosis

This patient has a rounded opacity in the apical region of the right lung. It appears white and has a well-circumscribed outline. This is most likely in keeping with a Pancoast tumour. The history of progressive breathlessness, cough and smoking history makes malignancy a likely diagnosis. Given the location of the tumour, it may not always be detected on auscultation. A Pancoast tumour is usually a non-small cell lung carcinoma that presents with various symptoms, including malignancy (e.g. malaise, weight loss, night sweats) and compression from the tumour (e.g. thoracic outlet syndrome, recurrent laryngeal nerve compression and Horner's syndrome). Horner's syndrome results from compression of the sympathetic ganglion and the lack of sympathetic innervation to the face giving rise to the characteristic features of ptosis, miosis and anhidrosis.

Erythema nodosum is an extra-pulmonary manifestation of sarcoidosis. Sarcoidosis presents with shortness of breath, cough and fatigue. However, chest x-ray findings differ and range from being normal to having hilar lymphadenopathy and pulmonary infiltrates which are not seen here. Erythema nodosum is also an extra-pulmonary feature of tuberculosis. The history of prolonged dyspnoea and dry cough tuberculosis. However, the patient's significant smoking history and lack of risk factors for tuberculosis make malignancy more likely.

Haematuria is a clinical feature associated with vasculitic diseases (e.g. Goodpasture's syndrome and granulomatosis with polyangiitis). Both can cause pulmonary haemorrhage that presents with dyspnoea, haemoptysis, and chest x-ray findings of pulmonary infiltrates. This is not in keeping with the current presentation.

Pericardial friction rub is occasionally heard in patients with a pericardial effusion secondary to pericarditis. Trauma, viral and bacterial infections and autoimmune diseases (e.g. rheumatoid arthritis and systemic lupus erythematosus) are all causes of pericardial effusion. However, neither of these diagnoses accounts for the apical opacity.

Similarly to haematuria, purpura is a clinical feature of vasculitic diseases or a significant infection causing septicaemia. The progressive onset of symptoms and normal observations makes septicaemia unlikely.

Question 63 of 189

A 27 year old lady with relapsing-remitting multiple sclerosis (MS) is referred to your clinic. She was diagnosed at age 18 and has been managed on interferon β 1a, with only four relapses in her total history. She has made good recovery from all four relapses and the only finding on neurological examination is of mild dysdiadochokinesis of the left arm. She scores functionally very well on an expanded disability status scale (EDSS) and has no disability as a result.

She is planning her first pregnancy and wanted ad0vice on how her MS may be affected. She has already been advised on stopping interferon β 1a in preparation for pregnancy. Aside from the impact of stopping interferon β 1a, she wants to know how pregnancy itself will affect her MS.

Which of the following is most accurate?

A. Relapse rates may decrease during pregnancy, increase at 3-6 months post-partum then return to pre-pregnancy rates.
B. There is an increased risk of developing visual disorders associated with MS.
C. There is a 30% chance that her RRMS will evolve into progressive MS.
D. There is a 15% chance that her relapse rate will decrease for the five years following pregnancy.
E. Relapse rates may increase during pregnancy, then return to pre-pregnancy rates at 9 months post-partum.

A. Relapse rates may decrease during pregnancy, increase at 3-6 months post-partum then return to pre-pregnancy rates.

A knowledge of the issues surrounding pregnancy in MS is important, since females of a childbearing age are a high risk group for developing the disease.

NICE guidance states that 'relapse rates may reduce during pregnancy and may increase 3 to 6 months after childbirth before returning to pre-pregnancy rates'. It also states that pregnancy does not increase the risk of progression of disease; the net effect of protection during pregnancy and subsequent increased relapse rate carries no increased risk of exacerbation.

With regard to Interferon β 1a in pregnancy, most neurologists would advise stopping when pregnancy is considered, due to inconsistent and conflicting data in the area. The BNF advises against use in pregnancy 'unless potential benefit outweighs risk'.

If a person with MS is thinking about pregnancy, they must have the opportunity to talk with a healthcare professional with knowledge of MS about:

- Fertility
- the risk of the child developing MS
- use of vitamin D before conception and during pregnancy medication use in pregnancy
- pain relief during delivery (including epidurals)
- care of the child
- breastfeeding.

In this scenario the use of the expanded disability status scale (EDSS) was referred to. This provides an index of clinical disability and may be used to quantify disease progression. It has widespread use in clinical trials.

Question 64 of 189

A 29-year-old man presents to the stroke ward following a right-sided middle cerebral artery territory infarct. You are reviewing him four weeks after as an outpatient. Whilst taking a history from him and his father you find out that he had a normal delivery and early development and initially performed well in school. However, from about the age of 12 he started developing focal impaired awareness seizures with secondary generalisation that were difficult to control with medications and started to struggle in school.

Since adulthood, he has suffered from frequent migraines that have again been difficult to treat with medications. He also suffers from recurrent vomiting although a thorough gastroenterological assessment including OGD with biopsies could not find any cause for the vomiting.

When aged 26 whilst out walking, he suddenly became very dizzy and unable to walk properly. These symptoms resolved in a day and he never sought medical attention. Since that incident, he has been left with a tremor that now makes his work as a painter almost impossible to do. He has also noticed that he is becoming more fatigued by walking. He goes to the gym regularly and is able to lift less and less weights as time has progressed. His mother died suddenly aged 37

Medications included: carbemazepine, clopidogrel and atorvastatin.
Examination

- Height 4ft 11inches (150 cm)
- Generalised muscle weakness with some atrophy
- Left upper limb power 3/5, right upper limb power 5/5
- Left lower limb power 5/5, right lower limb power 5/5
- Lateral nystagmus 5 beats
- Dysdiadochokinesia
- Moderate intention tremor
- MMSE 25/30

Hb	14.7 g/dl
Platelets	256
WBC	8.6
Na	140 mmol/l
K	4.5 mmol/l
Urea	4.6 mmol/l
Creatinine	54 µmol/l
Bilirubin	6 µmol/l
ALP	89 u/l
ALT	21 u/l
Total cholesterol	5.7 mmol/l
Albumin	40 g/l

CT Brain: right sided MCA territory infarct. Generalised atrophy
ABGs on air

pH	7.38
CO2	3.7 kPa
O2	15.6 kPa
Bicarb	49 mmol/l
Lactate	8.4 mmol/l

What is the most likely diagnosis?

A. Familial hypercholesterolaemia
B. MELAS syndrome
C. Carbemazepine poisoning
D. CADASIL
E. Pyruvate carboxylase deficiency

B. MELAS syndrome

The answer is B MELAS (Mitochondrial Encephalopathy with Lactic Acidosis and Stroke).

MELAS is a rare mitochondrially inherited condition characterised by short stature, recurrent migraines, vomiting, muscle weakness, seizures and then development of multiple strokes at an early age (normally before 40 for diagnosis to be considered) along with progressive dementia. There is also a raised lactic acid level in the blood and skeletal muscle biopsy will show ragged red fibres.

1% 68% 1% 28% 3%Regarding the vignette, it is possible that his mother had the same condition which is typical of mitochondrial inheritance. It also sounds like he may have had a posterior stroke that was not treated as he had sudden onset of cerebellar symptoms and is left with an intention tremor and has nystagmus. Cerebellar infarcts do not always show up on CT and MRI is the best modality for viewing the posterior fossa. Although is MMSE score is normal, for a young man a score of 25/30 is a potential worry in the clinical context.

Although CADASIL would also present with migraines and stroke at an early age, lactic acidosis is not a feature and subcortical infarct would be more typical. Although this gentleman has a high cholesterol it is not high enough for a diagnosis of familial hypercholesterolaemia and would not explain the muscle weakness, vomiting and lactic acidosis.

Carbamazepine poisoning could explain the cerebellar symptoms but not the other symptoms. Pyruvate carboxylase deficiency would cause muscle weakness, vomiting and lactic acidosis but would not explain the early onset of stroke

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A 58 year old man presents to Accident and Emergency with his wife complaining of memory loss. His first noticed a problem when he had difficulty making breakfast in the morning. When you question him, he has no memory of the events of today and patchy memory of the events of the past week although his wife informs you that he had no problem with his memory yesterday. He appears extremely anxious about his memory loss and repeatedly asks you 'Have I got dementia?'. He is able to remember his name and date of birth and recognise and name his wife. However, when using the toilet, he becomes lost and is unable to find his way back to the cubicle.

On examination his HS 1 + 2 + 0, he has corneal arcus, chest is clear. There is reduced power 4/5 to all arm movements in the right arm with normal reflexes, tone and sensation. His wife tells you that this has been present for several years following a stroke. There is no facial droop, there is no dysarthric speech or any other peripheral focal neurology and there are no cerebellar signs. Gait is normal.

He is able to count back from 20-1 and can name objects you present to him without difficulty. However he scores 0/3 on the delayed recall section of the MMSE. He is alert and fully responsive throughout the consultation. Past medical history is of previous left sided middle cerebral territory infarct and one previous TIA one year ago. Hypercholesteroalemia, hypertension. He has a 20 pack year history of smoking and used to drink 4-6 units of alcohol per day for 20 years but stopped this 25 years ago and his wife corroborates this information. His medications include Clopidogrel, Atorvastatin, Ramipril and Bendroflumethiazide.

CT Brain was performed 3 hours after waking up and is consistent with an old left sided infarct. No other abnormality detected.

What is the most appropriate treatment for this diagnosis?

A. Intravenous thiamine and Vitamin B
B. Oral thiamine and vitamin B

C. Thrombolysis
D. Donepezil
E. Supportive care only

E. Supportive care only

The answer is E. This man has a diagnosis of transient global amnesia. TGA is characterised by a sudden onset of global loss of recent memory and impaired new learning with no other cognitive defects. These patients are often very aware of their memory loss making them extremely anxious. Because of the dense anterograde amnesia, it is also characterised by repetitive questioning. This is sometimes called the broken record syndrome Loss of personal identity effectively rules out TGA Patients are alert and responsive during the episode

TGA is probably due to a transient malfunctioning of the limbo-hippocampal system which is involved in the formation of new memories and retrieval of recent memories. It normally presents in the 5th decade of life in those with pre-existing vascular risk factors, meaning there is probably a cerebro-vascular component but the cause is not known. TGA resolves spontaneously without need for treatment except for re-assurance and subsequent control of vascular risk factors.

Options A and B are inappropriate because the diagnosis is not Wernicke-Korsakoff syndrome. The presentation is too acute and too long after his heavy drinking of alcohol to be a likely diagnosis. Although there is similar defects in memory, there are normally defects in attention, concentration, frontal lobe functioning and is accompanied by symptoms such as apathy and withdrawal.

Option C is inappropriate as thrombolysis is not a treatment for TGA. In addition, thrombolysis would not usually be given to someone who has woken up with symptoms suggestive of a a stroke, even if the time window is less than 4.5 hours.

Option D is inappropriate as the diagnosis is not dementia. The onset of symptoms are too acute for this diagnosis to be considered.

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You review an 80-year old retired teacher in your clinic in your clinic. She has been complaining of numbness on her arms and difficulty walking. On examination you find she has reduced sensation lateral aspect of her arms and forearms in the anatomical position. She has no wasting on the muscles of her hands. She has reduced biceps and supinator reflexes and reduced power bilaterally.

When she gets up to go you notice she has a scissoring gait which is wide based. You ask her to close her eyes and she is unable to stand still without support.

She lives on her own and usually is independent, although recently she has been struggling to cope. She has a background of hypertension and high cholesterol.

What is the most appropriate investigation to diagnose this lady?

A. Cervical myelography
B. Cervical X-Ray
C. Nerve conduction studies
D. Nerve biopsy
E. MRI of the cervical cord

E. MRI of the cervical cord

This lady has lower motor neuron deficit and sensory loss in her upper limbs at the level of C5-C7. She has spastic paraparesis, Romberg +ve indicating loss of proprioception in her lower limbs.

This presentation is suspicious of cervical myelopathy. The main segments which are affected of the cervical cord in this condition are C5-C7, as is the case in this lady. The dermatomal distribution of her sensory loss is confined to C5-C7 therefore supporting this hypothesis.

Cervical myelopathy is caused by narrowing of the cervical spinal canal, most commonly caused by stenosis.

The aetiology includes:

- Cervical cord tumours
- ossification of the posterior longitudinal ligament
- Trauma
- Cervical degenerative changes (spondylosis)
- Cervical degenerative disc disease

The investigation of choice and one which would enable you to asses spinal and foraminal stenosis would be cervical myelography or MRI of the cervical cord. Cervical myelography entails the use of contrast and can be nephrotoxic alas MRI is the preferred modality to image and diagnose cervical spondylosis.

Question 67 of 189

A 57 year-old smoker presents to you with difficulty walking. He used to be able to mobilise unaided but recently he has required the assistance of his wife. He reports being more clumsy than usual. He drinks 3 cans of lager every month. He takes simvastatin for high cholesterol and has been taking aspirin for 10 years since his GP started it. He has lost a lot of weight in recent months, which he can't quantify.

On examination, you find that he is cachectic. He has an intention tremor and exhibits dysdiadochokinesia. His gait is broad-based. He has bilateral nystagmus on horizontal gaze. On auscultating his chest you find he has a degree of bronchial breathing on the right middle lobe. His observations are unremarkable.

His blood tests are as follows:

Hb	100 g/l
MCV	79.9 fL
Platelets	239 * 10 /l
WBC	6.6 * 10 /l
Na	134 mmol/l
K	4.3 mmol/l
Urea	6 mmol/l
Creatinine	88 µmol/l
Anti-Hu antibody	POSITIVE
Bilirubin	6 µmol/l
ALP	62 u/l
Albumin	29 g/l

CT head: No acute intracranial pathology.

What is the most appropriate investigation?

A. Chest X-ray
B. Vitamin B12 levels
C. ECG
D. Visual evoked potentials
E. Muscle biopsy

E. Muscle biopsy

This gentleman presents with signs and symptoms of the cerebellar syndrome.

He is cachectic, has a history of smoking and some signs of his chest which would raise concern given his history. Another vital clue in the question is the presence of anti-hu antibodies, which are associated with paraneoplastic cerebellar syndrome secondary to small cell lung cancer. Given the history and examination findings it is paramount to perform an X-Ray of his chest, and should that be inconclusive he will require a high-resolution CT scan.

An intracranial lesion, such as a tumour or a vascular event is also unlikely, given the normal CT head result and bilateral presentation.

Vitamin B12 deficiency is associated with subacute combined degeneration of the cord. This would present with upper and motor neuron deficits and a sensory neuropathy. It would not present with isolated cerebellar signs.

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A 40 year-old man presents to the general medical clinic with a one year history of generalised weakness. He mentions his father suffered from the same symptoms, although from a later age.

His past medical history includes type two diabetes mellitus (diet controlled) and eczema.

On examination he has male pattern balding and an expressionless face. You note a diminished left red reflex, with a normal cranial nerve examination. He has symmetrically reduced reflexes throughout, with mild reduced power distally in both upper and lower limbs.

Hb	140 g/l
Platelets	200 * 10 /l
WBC	8 * 10 /l
CK	70IU/L (reference range 60- 174 IU/L)

What is the most likely diagnosis?

A. Hyperthyroidism
B. Inclusion body myositis
C. Cushing's disease
D. Becker's muscular dystrophy
E. Myotonic dystrophy

E. Myotonic dystrophy

Myotonic dystrophy

Myotonic dystrophy is a genetic condition causing by a trinucleotide repeat expansion on chromosome 19. It demonstrates anticipation (earlier onset with successive generations).

Clinical features of the condition include:

- Frontotemporal balding Ptosis
- Distal weakness
- Depressed tendon reflexes
- Myotonic (slow relaxation of muscles)

Complications include arrhythmias, cataracts, testicular atrophy and diabetes mellitus.

Cushing's disease and hyperthyroidism cause a proximal myopathy, and the patient has no symptoms of either.

Becker's muscular dystrophy causes a proximal myopathy, but the average age of presentation and diagnosis is much younger (average is 11 years old), with several disability at 40.

Inclusion body myositis can cause both a proximal and distal myopathy. This patient's creatinine kinase is normal, which makes the diagnosis less likely and would not explain the genetic component and facial features.

Question 69 of 189

A 73 year old gentleman is admitted to the medical assessment unit with a fall. A collateral history from his wife reveals that he has been having frequent falls (nearly 2-3 times a week) for the last 4 months. This has resulted in multiple admissions where a chest infection is thought to be the main cause for his deterioration. His wife also states he is having problems swallowing, chokes on his food frequently and has not been himself recently with frequent bouts of aggression. On examination you note the patient has an expressionless face, and kyphosed posture and mild tremor bilaterally. He has cogwheeling of both upper limbs. Examination of his legs is unremarkable. On inspection of his gait, it appears parkinsonian in character, but you stop him only after a few steps as he seems unsteady. On cranial nerve examination you note he has difficulty following your finger downwards but the remainder of the cranial nerve examination is normal.

Given these features, what is the most likely underlying diagnosis?

A. Multiple system atrophy
B. Idiopathic Parkinsons disease
C. Progressive supranuclear palsy
D. Vascular Parkinsonism
E. Lewy body dementia

B. Idiopathic Parkinsons disease

There is no doubt that this patient has an element of Parkinsons disease, the clues to the underlying condition include:

- The SYMMETRICAL tremor compared with the ASYMMETRICAL tremor of idiopathic Parkinsons.
- Swallowing difficulties suggesting repeated admissions for aspiration pneumonia.
- Changes in personality.
- Truncal instability and rigidity > limbs.

- Inability of look downwards (Vertical gaze palsy).

Parkinsons Plus syndromes:

- Progressive supranuclear palsy (PSP, also known as Steele-Richardson-Olszewski syndrome) Characterised by early postural instability, frequent falls, truncal rigidity, asymmetrical onset, speech and swallowing problems, vertical gaze palsy.
- Multiple system atrophy - (MSA, also known as Shy-Drager syndrome). Autonomic features specifically postural blood pressure drop, bladder/bowel dysfunction, cerebellar and/or pyramidal signs.
- Lewy body dementia - Visual hallucinations accompanying dementia.
- Vascular parkinsons - Parkinsonism worse in the legs than the arms.
- Cortico-basal degeneration (CBD) - Asterognosis (inability to recognise objects by touch), akinetic rigidity involving one limb. Some patients may experience the 'alien limb phenomenon'.

NB: Knowledge of this differential is also vital for PACES examinations.

Question 70 of 189

A 32 year old man presented to the Emergency Department with a severe headache. The headache had started suddenly that evening while the patient was at home with his girlfriend watching television. The patient described his headache as an intense throbbing pain felt all across his head. The pain had started very quickly with the patient reporting a severity of 10 / 10 within 30 seconds. The pain had eased slightly following some morphine given by the ambulance crew. The patient denied any symptoms of neck stiffness or photophobia. There were no visual symptoms and no weakness in the patient's limbs or abnormal sensation.

The patient had no previous past medical history and did not normally suffer from headaches. He took no regular medications. The patient worked as a retail assistant and lived with his girlfriend. He rarely drank alcohol but did smoke cannabis on several occasions each month. The patient's girlfriend reported that the patient had smoked cannabis that evening prior to suffering his headache.

On examination, the patient was in visible discomfort due to his headache although had no meningitic signs. Glasgow Coma Score was 15/15 and the patient was afebrile. Cranial and peripheral nerve examination was unremarkable.

Further analgesia was given to the patient and investigations requested as detailed below.

CT brain: no evidence extra-dural or sub-dural bleeding; no intracerebral haemorrhage or subarachnoid blood; no evidence of cerebral infarction; normal ventricular system; no radiological contra-indication to lumbar puncture

Cerebrospinal fluid results:

Red cell count	1 / mm^3
White cell count	2 / mm^3
Pprotein	0.75 g / L
Glucose	60 % of serum value
Microscopy	No organisms seen
Red cell breakdown products	Negative
Opening pressure	19 cmCSF

Following initial investigation as above, the patient continued to experience severe recurrent headaches similar to that experienced at presentation and was observed to have a short,generalised seizure on the admission ward prompting further investigation. CT angiography: diffuse arterial beading identified; results otherwise as for previous CT head

What is the cause for the patient's headache?

A. Atypical migraine
B. Cervical artery dissection
C. Reversible cerebrovascular vascoconstriction syndrome
D. Posterior reversible leucoencephalopathy syndrome
E. Pituitary apoplexy

C. Reversible cerebrovascular vascoconstriction syndrome

The patient presents with a typical 'thunderclap' headache, with pain reaching a severe intensity within one minute of onset. Reversible cerebrovascular vasoconstriction syndrome (RCVS) is a cause of thunderclap headache with normal CT brain and normal or near-normal lumbar puncture results. RCVS may also be associated with focal neurological signs and seizures. Diagnosis is made by the finding of diffuse arterial beading on angiography (CT, MRI or catheter). One-half of cases occur during the postpartum period or after exposure to serotoninergic agents, adrenergic substances or cannabis. There are no evidence-based treatments for RCVS although bed-rest and nimodipine are usually prescribed. RCVS is not always benign, with one-in-four patients suffering sub-arachnoid haemorrhage or ischaemic / haemorrhagic stroke

The other answers listed in the stem are all causes of thunderclap headache with normal CT brain and lumbar puncture (i.e. sub-arachnoid haemorrhage excluded). None of these conditions area associated with diffuse arterial beading and so are less likely differential than RCVS.

Question 71 of 189

A 68-year-old man is admitted with weakness. He states that it came on at 09:45 (2 hours and 15 minutes ago). He states that his right arm and leg are weak. He also complains of word finding difficulties. He has a past medical history of hypertension and hypercholesterolaemia. On examination he has grade 4 weakness of his right arm and leg, and there is an obvious right sided facial droop. He has a right homonymous hemianopia. He can follow complex 3 stage commands, however he struggles to name simple objects.

You suspect an acute stroke and are considering suitability for thrombolysis. You perform blood tests and arrange an urgent CT scan.

What scoring system will you utilise to further assess this patient?

A. CHA,,DS,,-VASc
B. ABCD2
C. HAS-BLED

D. Total Health Risk In Vascular Events (THRIVE)
E. National Institutes of Health Stroke Scale (NIHSS)

E. National Institutes of Health Stroke Scale (NIHSS)

The NIHSS score is useful in stroke for assessing patients suitability for thrombolysis Important for me Less important

The most immediate concern is to assess the suitability for thrombolysis. Thrombolysis can result in extremely good outcomes for patients, however it is also has significant risks and it is therefore essential to balance benefits versus potential risks. The National Institutes of Health Stroke Scale (NIHSS) is an extremely useful tool for assessing the severity of the stroke. Some hospitals use an NIHSS of less than 5 (mild stroke) to exclude patients from thrombolysis. The patient in this case is likely suffering from a total anterior circulation stroke due to the presence of hemiparesis, homonymous hemianopia and higher function involvement (e.g. expressive dysphasia). He will therefore likely have a high NIHSS score.

The CHA,,DS,,-VASc score is used to predict the risk of stroke in patients with atrial fibrillation. The ABCD2 score is used in transient ischaemic attacks to predict the likelihood of a stroke in theshort term period following the TIA. The ABCD2 score ranges from 0 to 7. Recent studies have shown that the ABCD3-I scoring system is superior in the assessment of TIAs. The ABCD3-I incorporates recurrent TIA events and imaging. The minimum score is 0 and the maximum is 13. The broader range of scores allows for potentially greater refinement of risk prediction.

The HAS-BLED score is used to predict the risk of bleeding from anticoagulation in atrial fibrillation.

The Total Health Risk In Vascular Events (THRIVE) score uses NIHSS score, age, and chronic disease to predict long-term neurologic outcomes in stroke patients.

Question 72 of 189

A 54-year-old woman is seen in the neurology clinic. She initially presented to her general practitioner with paraesthesia and a burning sensation down her right side. She reports a similar episode two years ago, during which she had a four-week period of paraesthesia in the left lower limb which self resolved. In between these episodes, she experienced total resolution of her symptoms.

An MRI of the brain and spinal cord was performed and concluded as below:

MRI brain and spinal cord shows Several areas of T1 weighted hypointensities and T2 weighted hyperintensities consistent with multiple sclerosis

Which of the features in this woman represents a poor prognostic factor?

A. Female sex
B. Long interval between relapses
C. Older age of onset
D. Relapsing-remitting disease
E. Sensory symptoms only

C. Older age of onset

Older age of onset is associated with a poor prognosis in multiple sclerosis

As stated in the question, this woman has been diagnosed with multiple sclerosis (MS). Of the features listed, only older age is associated with a poor prognosis. The onset of symptoms in patients, who are in their 20s or 30s, is associated with a good prognosis.

MS is less common in males; however, when diagnosed, the prognosis is worse. Female sex is therefore associated with a better prognosis and is, therefore, an incorrect answer here.

This woman has had two years separating her two attacks which is a good prognostic indicator rather than a poor one. A long interval between relapses is, therefore, an incorrect answer.This woman exhibits a relapsing-remitting pattern of disease with distinct episodes of symptoms with improvement in between. A relapsing-remitting disease has a significantly better prognosis than a progressive disease course (continuous symptoms with gradual worsening), and therefore this is not the correct answer.

Motor symptoms are associated with a poorer prognosis. This woman has only experienced sensory symptoms, which is a good prognostic factor in this case.

Question 73 of 189

A 72-year-old male diagnosed with dementia after a one-year history of progressive cognitive decline, particularly in planning, cognitive flexibility, attention, visual memory and visuospatial manipulation on detailed neuropsychological assessments. His mini-mental state examination scores 15/30. His past medical history includes Parkinson's disease, for which he was started on Madopar 6 years ago. His son attends clinic with his father and both are very keen to start some treatment.

What is the most available treatment?

A. No treatment
B. Rivastigmine
C. Donepezil
D. Increase Madopar dose
E. Deep brain stimulation

B. Rivastigmine

The patient describes a syndrome of executive and visuospatial dysfunction on a background of PD diagnosis more than 12 months before diagnosis of cognitive impairment. This represents PD dementia (instead of Lewy Body dementia). The only licensed proven treatment for PD dementia demonstrated in a randomised clinical trial is rivastigmine. Donepezil is also useful for PD dementia but is not currently licensed in the UK. Neither DBS nor increasing PD medications improve cognitive impairment.

Question 74 of 189

A 54-year-old man presents to the emergency department with an intractable headache, worse on bending down and in the morning. It has been present for one month. He has no past medical history and does not take any regular medications.

A CT head is organised, which demonstrated a large right-sided mass lesion that appears to be causing displacement of the uncus of the temporal lobe under the tentorium cerebelli.

What is the earliest expected abnormal clinical sign to be detected given the clinical presentation?

A. Cheyne-Stokes breathing
B. Contralateral hemiparesis
C. Contralateral mydriasis
D. Ipsilateral hemiparesis
E. Ipsilateral mydriasis

E. Ipsilateral mydriasis

Uncal herniation results in pressure on the midbrain resulting in ipsilateral CN III palsy (mydriasis is the earliest sign) and contralateral hemiparesis

Ipsilateral mydriasis is correct. The CT demonstrates uncal herniation as a consequence of an intracranial mass lesion. The ipsilateral dilated pupil is caused by compression of the ipsilateral oculomotor nerve.

Contralateral mydriasis is incorrect. This is a rarer clinical sign. It may develop from contralateral oculomotor nerve compression from midline shift.

Contralateral hemiparesis is incorrect. This is an expected consequence of progressive uncal herniation but typically occurs subsequent to ipsilateral mydriasis.

Ipsilateral hemiparesis is incorrect. This is rarer than contralateral hemiparesis. However, it can occur as a consequence of the 'Kernohan phenomenon'. This is a radiological description of extensive midline shift causing mass effect. The result is denting of the contralateral cerebral crus by the tentorium cerebelli. . The corticospinal tracts are damaged and a hemiparesis contralateral to the crus cerebri results. This is ipsilateral to the mass lesion and is a false localizing sign. It is a rarer occurrence and would occur subsequent to mydriasis.

Cheyne-Stokes breathing is incorrect. This signifies impending death and is a terminal sign of progressive cerebral herniation.

Question 75 of 189

A 65-year-old female is referred to the neurology clinic with complaints of tingling and loss of sensation. Five years previously she experienced an episode of numbness and tingling affecting her right upper limb. Most recently, there has been a loss of sensation affecting her left lower limb. There has been a full recovery between episodes. She has no past medical history and does not take any regular medications.

The examination at the time of review is unremarkable. An MRI of her brain demonstrates lesions in the periventricular white matter.

Given the likely diagnosis, what clinical feature of her presentation portends the worst prognosis?

A. Age of onset
B. Complete recovery between relapses
C. Relapsing-remitting disease
D. Sensory symptoms only
E. 5 year interval between first two relapses

A. Age of onset

Older age of onset is associated with a poor prognosis in multiple sclerosis

Age of onset is correct. This patient has a diagnosis of multiple sclerosis on the basis of sensory neurological symptoms separated in time and space along with classic imaging findings of periventricular white matter lesions. Older age of onset is associated with a worse prognosis in multiple sclerosis.

Relapsing-remitting disease is incorrect. This is associated with a better prognosis than other forms of the disease e.g. primary progressive MS.

Sensory symptoms only are incorrect. This is typically associated with a good prognosis.

5-year interval between the first two relapses is incorrect. A short interval between the first relapses is associated with a worse prognosis. A 5-year interval would be associated with a betterprognosis.

Complete recovery between relapse is incorrect. If the patient did not recover fully between relapses then this would be associated with a worse prognosis.

Question 76 of 189

A 56-year-old gentleman is brought by his wife with unusual behaviour. This afternoon, she found him doing gardening in the back garden and he was repeatedly cutting at the same shrubs. He was conscious but asked her repeatedly when dinner was ready. He returned to the house where he had no recall of gardening and continued to ask the same questions repeatedly.

In the department, he seems well. He is a little confused over what has happened but is orientated in time and place. On examination, there is no ophthalmoplegia but there were two beats of nystagmus seen. He has 5/5 power in all limbs with normal tone and no myoclonus or fasciculations. His gait is normal.

His wife reports that there have been no memory problems or change in personality prior to today. He seems to be back to normal at present. He denies any excessive alcohol use and his father did have Alzheimer's disease which was diagnosed at 60 years of age.

Na	138 mmol/l
K	4.2 mmol/l
Urea	5.6 mmol/l
Creatinine	78 µmol/l
Bilirubin	27 µmol/l
ALP	105 u/l
ALT	30 u/l
Albumin	34 g/l
Hb	110 g/l
Platelets	394 * 10 /l

WBC	5.2 * 10 /l
MCV	96 fL (normal 80-100)
B12	250 pg/ml (normal 180-1000)
Folate	5 ng/ml (normal >4)
CT head	age related involutional changes, old lacunar infarcts seen on left side, no intracranial haemorrhage

What is the likely diagnosis?

A. Transient ischaemic attack
B. Creutzfeldt-Jacob Disease
C. Alzheimer's Disease
D. Wernicke's encephalopathy
E. Transient global amnesia

E. Transient global amnesia

Transient global amnesia - acute onset of anterograde amnesia, patent may keep asking the same questions, resolves within 24 hours

This gentleman presents with an acute episode of amnesia that has resolved in a number of hours. He has no neurological abnormality on examination and has returned to normal. Alzheimer's disease and Wernicke's encephalopathy would not present this acutely. Creutzfeldt-Jacob Disease has a rapidly progressive dementia and would not resolve after some hours. A TIA is possible but usually presents with stroke-like symptoms and isolated amnesia would be hard to reconcile neurologically. Transient global amnesia is a poorly understood phenomenon that may be related to migraines in pathophysiology where the individual has a profound amnesia for hours to days and is not able to form new memories. They resolve and are well following this but it can occur again.

Question 77 of 189

A 32 year old man presents to your haematology clinic with a 4 year history of fatigue, weight loss and a feeling of fullness in the stomach. He has been off sick from work for the past 3 months and has recently been divorced. He also complains of low mood and has attempted suicide in the past. He has a family history of NHL. He has been investigated in the past with CT scans, blood films and lymph node biopsies on several occasions with the most recent set of investigations occurring 6 months ago.

Despite your reassurances that he does not have a lymphoma, he is still convinced that he does and that he is dying. You suggest a referral to a psychiatrist and he becomes very angry.

What is the most likely underlying diagnosis?

A. Atypical depression
B. Specific phobia
C. Hypochondriasis
D. Conversion disorder
E. Somatoform disorder

C. Hypochondriasis

Unexplained symptoms

Somatisation = Symptoms

hypoChondria = Cancer

The answer is hypochondriasis. For a diagnosis is hypochondriasis to be made the patient needs to have a persistent belief that he or she has a serious illness which may be related to misperception of bodily symptoms. Physical causes need to have been ruled out and the patient is not reassured despite overwhelming evidence to the contrary. In addition there needs to be an impact on psychosocial functioning. In this case the patient fits all of these criteria.

Patients with hypochondriasis are likely to have concurrent depression, although depression is not the primary diagnosis here. Hypochondriasis is more common in those who have lost a close family member to a serious illness.In conversion disorder and somatoform psychological trauma is manifested as physical symptoms. In these disorders, it is a preoccupation with the symptoms which is the problem, rather than fear of a diagnosis. Whilst this patient has a fear of having lymphoma, hypochondriasis is a more appropriate diagnosis.

Question 78 of 189

A 63-year-old man presented with a three-week history of double vision and fatigue. Over this period he had noticed that when he swallowed liquids these often came back out of his nose. He reported a decreased exercise tolerance over the past six months due to fatigue and shortness of breath.

On examination he was thin. He had bilateral ptosis and diplopia on looking in multiple directions. His voice was soft and he appeared peripherally cyanosed. On auscultation his chest was clear and heart sounds were normal.

Observations:

- Heart rate: 90 beats per minute
- SaO2: 92% on room air
- Respiratory rate: 22 breaths per minute
- Temperature: 37.1 degrees Celsius
- Blood pressure: 110/68 mmHg

Arterial blood gases breathing air:

PO2	7.80 kPa (11.3 12.6)
PCO2	9.52 kPa (4.7 6.0)
pH	7.31 (7.35 7.45)
bicarbonate	32.4 mmol/L (21 29)
base excess	10 mmol/L (+/- 2)

Which urgent investigation should be performed next?

A. Chest X-ray
B. Forced vital capacity
C. Computed Tomography Pulmonary Angiogram
D. Electrocardiogram
E. Bedside echocardiogram

B. Forced vital capacity

This patient has myasthenia gravis as suggested by his fatigue, ptosis, complex ophthalmoplegia and bulbar weakness. He has an element of neuromuscular weakness which is resulting in type 2 respiratory failure. The most important step after adequately resuscitating this patient is to perform a bedside Forced Vital Capacity measurement. If this is < 1.5 then it may be necessary to involve the Intensive Treatment Unit team and consider ventilatory support.

Question 79 of 189

A 59-year-old woman is referred by her GP to neurology clinic for assessment. The patient describes intermittent episodes of pain affecting the left side of her face. She recalls the first attack vividly as a sudden onset of severe 'electric-shock' pain coming on suddenly around one year previously as she had cleaned her teeth with her electric toothbrush. The pain was felt around the cheek extending down to the jaw-line. The first attack had lasted several hours and she had ultimately attended her dentist and received a filling to a left molar tooth. While symptoms from that episode had resolved, she had then suffered from similar episodes at increasing frequency, approximately every fortnight over the last couple of months. These attacks had on occasion been unprovoked but were sometimes induced by stimulation of the affected area or cold winds. The patient was tearful as she recounted the negative impact her symptoms had had one her lifestyle. There was no history of headaches, sinus symptoms or seizures.

The patient had a fairly unremarkable past-medical history, notable only for hypothyroidism and long-standing struggles to avoid overweight. Her only regular medication was thyroxine 125 micrograms daily and the patient reported no allergies. There was no family history of neurological disease. The patient worked as a law clerk and did not smoke or drink significant alcohol.

Examination demonstrated an unremarkable cranial nerve and peripheral nerve examinations. In particular, colour vision, visual acuity and hearing where normal.

What is the correct first line management of the patients symptoms?

A. Gabapentin
B. Carbamazepine
C. Pregabalin
D. Baclofen
E. Lamotrigine

B. Carbamazepine

The patient reports a classical history of trigeminal neuralgia. Notable features are the memorable first occurrence, attacks precipitated by touch or vibration and the history of inappropriate dental treatment. Time between attacks often gradually reduces leaving patients quality of life significantly impaired.The patient has no other symptoms or neurological signs that might suggest an alternative diagnosis such as multiple sclerosis, sinusitis or a space-occupying lesion. In the presence of such features, an MRI brain is the most appropriate investigation.

Carbamazepine is the only drug licensed specifically for trigeminal neuralgia in the UK. In 70 % of patients it provides initial 100 % pain relief. However, caution must be used due to side-effects and drug interactions. Oxcarbazepine is a derivative of carbamazepine with similar efficacy but improved tolerability and side-effect profile.

In the evidence of allergy or intolerance to the above drugs then use of baclofen or lamotrigine should be considered, however the evidence base for both is thin. Gabapentin in combination with ropivacaine injections did show benefit in one randomised controlled trial but this result has not been replicated in subsequent investigations. Pregabalin had reported effectiveness in one prospective study of 53 patients.

Question 80 of 189

A 78-year-old man is admitted following being found on the floor at home. He has no recollection of how he got to floor or how long he had been there. He reports feeling generally unwell and having a cough for a number of days. There is no medical history of note and he takes no regular medications. He lives alone and appears unkempt. Examination reveals bronchial breathing throughout his left mid zone. Neurologically, he has new onset weakness of left sided shoulder abduction and adduction, alongside mild weakness in left elbow flexion. Additionally, reduced sensation in the lateral aspect of his upper arm is noted. A CT head is undertaken.

CT head report

Age related involutional change. No evidence of intracranial haemorrhage or recent ischaemic event.

What is the most likely diagnosis?

A. Stroke
B. Rotator cuff tear
C. Brachial plexus injury
D. Brown-Sequard syndrome
E. Botulism

C. Brachial plexus injury

Given the pattern of neurological signs in his left limb, this would not fit with a stroke. Global weakness and sensory loss would be expected in the affected limb.

A rotator cuff tear is possible given the likely trauma. However, sensory loss would not be expected.

Brown-Sequard syndrome is caused by damage to half of the spinal cord. It produces ipsilateral paralysis and proprioception loss, and contralateral temperature and pain loss.

Botulism classically presents with cranial nerve deficits, followed by a descending weakness and autonomic dysfunction.

A brachial plexus injury may be sustained following trauma, as in this case. The level of injury will 5% 7% 79% 5% 4%correlate with the signs. In this patient, it is at C5, causing the specific distribution of weakness and sensory loss.

Question 81 of 189

A 23 year-old student with epilepsy presents with generalised tonic-clonic status epilepticus. He is already taking phenytoin. Despite intravenous administration of diazepam and then phenobarbital, he is still fitting after 30 minutes.

What is the best course of action?

A. Check phenytoin levels and reload if necessary
B. Send urine for toxicology
C. Organise brain imaging
D. Obtain an electroencephalogram (EEG)
E. Induction of general anaesthesia with thiopentone

E. Induction of general anaesthesia with thiopentone

The correct answer in this scenario is the induction of general anaesthesia with thiopentone. In a case of refractory status epilepticus, where seizures have not responded to first and second-line treatments such as benzodiazepines (e.g., diazepam) and anti-epileptic drugs (e.g., phenobarbital), it is important to escalate treatment rapidly. Induction of general anaesthesia with thiopentone can help achieve rapid control of seizures, prevent further neuronal injury, and reduce the risk of long-term complications.

Regarding the other options:

Check phenytoin levels and reload if necessary: While checking phenytoin levels may be useful in determining whether the patient's ongoing seizures are due to subtherapeutic drug levels, it would not be appropriate to delay more definitive treatment in this case. The urgency of treating status epilepticus outweighs waiting for lab results, which could take hours.

Send urine for toxicology: Although identifying an underlying cause for the seizure is important, sending urine for toxicology would not provide immediate information needed to manage this acute situation. Management should prioritize stopping the seizure activity first before investigating potential causes.

Organise brain imaging: Brain imaging can provide valuable information about structural abnormalities that may be contributing to the patient's seizures; however, it is not an immediatepriority during an ongoing status epilepticus episode. Once seizure activity has been controlled and stabilised, brain imaging may be considered as part of a comprehensive evaluation.

Obtain an electroencephalogram (EEG): An EEG can help confirm the diagnosis of status epilepticus and monitor response to treatment. However, obtaining an EEG should not delay urgent interventions like induction of general anaesthesia with thiopentone. Ideally, continuous EEG monitoring should be initiated after initiating anaesthesia to assess seizure control and guide further management.

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A 28 year-old woman presents with a two month history of double vision, which is worse at the end of each day. On examination, there is bilateral ptosis which is fatiguable. There is a complex ophthalmoplegia which does not conform to the pattern of one or more cranial nerves. Examination of the limbs is unremarkable.

Which of the following would be most suggestive of a diagnosis of myasthenia gravis?

A. Weakness confined to extraocular muscles
B. A decremental response to repetitive nerve stimulation
C. An incremental response to repetitive nerve stimulation
D. Thymic enlargement seen on chest imaging
E. Internuclear ophthalmoplegia

B. A decremental response to repetitive nerve stimulation

In myasthenia gravis affected muscles show a decremental response to repetitive nerve stimulation (there is a progressive decline in the amplitude of the compound muscle action potential with repeated stimulation).

An incremental response to repetitive nerve stimulation is seen in the Lambert-Eaton myasthenic syndrome.

Weakness confined to extraocular muscles is certainly compatible with myasthenia gravis but is not diagnostic, and may be seen in thyroid eye disease, brainstem syndromes, and mitochondrial diseases. Ocular myasthenia is the mode of presentation of around half of patients with myasthenia gravis. A proportion may go on to develop generalised disease.

Thymic hyperplasia and thymoma are commonly seen in association with myasthenia gravis, as well as other autoimmune disease. Thymectomy is indicated in all patients with thymoma, due to the risk of malignant transformation. Thymectomy in the case of thymic hyperplasia is more controversial, and is generally reserved for younger patients with generalised disease and antiAChR antibodies.

True internuclear ophthalmoplegia occurs in lesions of the medial longitudinal fasciculus in the brainstem, and as such is not a feature of neuromuscular junction disorders such as myasthenia gravis. However, the involvement of extraocular muscles (one or both medial recti) may on occasion mimic internuclear ophthalmoplegia.

Question 83 of 189

As the medical registrar on-call you are fast-bleeped to see a patient in the resuscitation room of the Emergency Department.

A 27 year-old lady presented with severe breathing difficulties and hypoxia, and had become increasingly drowsy whilst in the department. Arterial blood gases performed by the emergency physicians showed:

- pH 7.142
- pCO_2 12.5 kPa
- pO_2 9.19 kPa
- HCO_3 25.3 mmol/l

Due to the rapidity of her decline, the emergency physicians tell you that only a very brief history was possible before she required intubation. She described a productive cough over the last few days, and mentioned that she was taking tablets for a neurological condition.

On examination, she is intubated and maintained on sedation with propofol. You notice a wellhealed midline sternotomy scar. On auscultation of the chest there are coarse crackles audible in the left mid and lower zones.

She is transferred to the Intensive Care Unit for continued mechanical ventilation and is commenced on empirical broad-spectrum antibiotics.

Which one of the following additional interventions will most hasten her recovery?

A. Botulinum anti-toxin
B. Edrophonium
C. Plasma exchange
D. 3,4-diaminopyridine
E. Steroids

C. Plasma exchange

This lady has myasthenia gravis and is taking pyridostigmine tablets for symptomatic relief. Shehas had a thymectomy in the past.

The acute presentation is of myasthenic crisis, with dramatically deteriorating weakness progressing to respiratory failure. Crisis is typically precipitated by infection (in this case pneumonia), or non-compliance with medication.

The initial priority is ventilatory support. Falling oxygen saturations and PaO2 are late indicators of ventilatory compromise. A rising PaCO2 is a more sensitive marker. Although inappropriate in this case, forced vital capacity should be measured and generally if below 1.5L ventilatory support should be considered.

In considering antibiotic cover, gentamicin should be avoided as it may interfere with neuromuscular transmission. Similarly, the anaesthetists should be warned that the patient will be extremely sensitive to small doses of non-depolarising muscle relaxants such as atracurium and vecuronium, which are commonly used for rapid sequence induction in the emergency setting.

Plasmapheresis and intravenous immunoglobulin are used to provide a rapid disease-modifying effect in myasthenic crisis. In plasma exchange, circulating anti-acetylcholine receptor antibodies are removed, relieving neuromuscular blockade and improving weakness. The major side effects are worsening of sepsis, due to removal of protective antibodies, and coagulopathy due to removal of clotting factors and destruction of platelets.

Botulinum anti-toxin is a specific treatment for botulism.

Edrophonium (Tensilon) is a short-acting acetylcholinesterase inhibitor which is used in the diagnosis of myasthenia gravis.

3,4-diaminopyridine is a specific treatment for the Lambert-Eaton myasthenic syndrome.

Steroids and other immunopressive agents may be used for the treatment of myasthenia gravis but in this case plasma exchange is preferred as a rapid response is required.

Question 84 of 189

A 46 year-old man is admitted to hospital after he is found wandering the streets, unable to remember who he his or how he got there. After a collateral history is obtained, he is referred to the neurologists for further investigation.

Over the last two months he has become increasingly forgetful and clumsy, and his behaviour has become progressively more bizarre. His speech has become confused, and he struggles with daily tasks. Others have commented that he has become very 'twitchy', and he sometimes drops things out of his hands without meaning to.

He was previously fit and well and the main carer for his father who suffers from motor neuron disease. His sister who previously helped him care for their father was recently killed in a car accident.

His only past medical history is that at age 22 he underwent extensive surgical resection of a large parafalcine meningioma which was discovered after investigation of chronic headache. Since the operation he has been free of headache.

On examination he is completely disoriented in time, place, and person. His gait is broad-based and uncoordinated, and during the examination you notice several shock-like jerks of his arms. Tone, power, reflexes, and sensation are otherwise normal, and cranial nerve examination is unremarkable.

What is the most likely diagnosis?

A. Viral encephalitis
B. Creutzfeld-Jakob disease
C. Frontotemporal dementia
D. Alzheimer's disease
E. Dissociative fugue

B. Creutzfeld-Jakob disease

The history of rapidly progressive dementia, ataxia, and myoclonus is highly suggestive of Creutzfeld-Jakob disease (CJD). The previous neurosurgery may have required the use of a dural graft and if so this is the likely method of transmission of the prion protein (iatrogenic CJD).Viral encephalitis is unlikely over a two month period.

Frontotemporal dementia causes a dementia with prominent personality change and behavioural disturbance, which may progress rapidly over months to years. There is an association with motor neuron disease.

Alzheimers disease would not typically occur at such an early age, or cause such a rapid deterioration. Ataxia would be unusual, and although myoclonus can occur in advanced dementia, this would not be typical early in the disease.

Dissociative fugue is a psychiatric condition characterised by disintegration of personality and memory, typically precipitated by a stressful event, and accompanied by wandering.

Question 85 of 189

A 35-year-old female presents to the Emergency Department with three days of increasing weakness in the left arm and reduced visual acuity in the left eye. She was diagnosed with relapsing-remitting multiple sclerosis two years earlier. She takes fingolimod as maintenance therapy and denies any compliance issues.

On examination, she has weakness in wrist extension and finger abduction in the left hand and visual acuity in the left eye was measured at 6/24 with an associated reduction in colour saturation.

Her blood tests were unremarkable and in particular, her white cell count was normal. Her MRI scan does show two new enhancing lesions in the right pericallosal region.

How should this be managed acutely?

A. Commence high dose oral prednisone and wean over a month
B. Commence intravenous dexamethasone
C. Commence natalizumab infusion
D. Commence high dose methylprednisolone for 3-5 days
E. Biopsy the enhancing lesions

D. Commence high dose methylprednisolone for 3-5 days

Given that this patient has relapsed on maintenance therapy, and that she has multi-focal signs affecting vision and motor function, she should be admitted to hospital and commenced on highdose intravenous methylprednisolone.

Acute relapses can be treated with either high dose oral prednisolone or intravenous therapy. The choice depends on multiple factors including the severity of symptoms, the requirement for hospital admission for monitoring, and co-morbidities such as diabetes and depression.

After this patient has recovered from this acute relapse a review of her maintenance therapy should occur, and this may be escalated through her neurologist based on a number of criteria.

Question 86 of 189

A 67-year-old male presents to the hospital with 2 hours of acute onset right sided weakness and speech difficulties.

A CT cerebral angiogram shows proximal left middle cerebral artery (LMCA) thrombosis.

Seven days prior to this presentation he underwent a laparotomy for bowel obstruction secondary to an incarcerated umbilical hernia.

In the absence of IV thrombolysis which of the following emergency therapies may benefit the patient?

A. Intra-arterial clot retrieval
B. Prasugrel
C. Mannitol
D. Intravenous heparin infusion
E. Rosuvastatin

A. Intra-arterial clot retrieval

Patient's presenting with a confirmed acute proximal MCA or distal internal carotid artery or basilar artery occlusion may benefit from intra-arterial clot retrieval. Ideally, patients should undergo the procedure within 6 hours of symptom onset.

Recent abdominal surgery is an exclusion criterion for intravenous thrombolysis which is why the patient in this question may benefit from intra-arterial clot retrieval.

Three recently published randomised trials established the safety and efficacy of intra-arterial clot retrieval in addition to intravenous thrombolysis for eligible patients presenting with acute stroke. Patients who are ineligible for IV thrombolysis who present within the appropriate time-frame with large vessel occlusion may also benefit from intra-arterial clot retrieval.

All other answers have not been proven to benefit the patient in the emergency setting.

A statin based therapy should however be initiated in all ischaemic stroke patients as part of their secondary prevention regimen.

Question 87 of 189

A 75-year-old man is being reviewed after transient expressive dysphasia and clumsiness in the right hand. His symptoms completely resolved after 20 minutes.

He does not have a background diagnosis of ischaemic heart disease or diabetes but is an exsmoker with a 50 pack year history. His blood pressure was 160/70 mmHg and he was in sinus rhythm. He had no detectable neurological signs on examination.

His MRI brain scan showed a very small area of restricted diffusion in the left frontotemporal region consistent with ischaemia.

He has a total fasting cholesterol of 4 mmol/L (normal) with an LDL level of 2.5 mmol/L (normal).

With regards to cholesterol, in addition to dietary advice and exercise what else may benefit this patient?

A. Commence on a fibrate
B. Commence a statin
C. Commence a fibrate and a statin
D. Commence fish oil tablets
E. Assess for diabetes and only if present commence a statin

B. Commence a statin

In addition to diet and exercise modifications, all patients who are diagnosed with stroke or TIA should be commenced on statin therapy irrespective of the cholesterol level.

Question 88 of 189

A 63-year-old man presents to the emergency department with a 5-minute episode of slurred speech earlier in the day. His wife noticed that his face was drooping to one side as well. He had no arm weakness and is now completely back to normal. He is normally well and on no regular medication and is not allergic to any medication. He works as a plumber and smokes 10 cigarettes per day for the last 40 years and drinks alcohol socially. On further questioning he mentions that he had a similar episode also lasting 5 minutes four days ago whilst at work. On examination, his blood pressure is 135/70 mmHg and his heart rate is 58/min. He has no focal neurology and his cardiovascular and respiratory examinations are unremarkable. He has been given 300mg of Aspirin by the paramedics.

His blood tests are as follows:

Hb	138 g/l
Platelets	283 * 10 /l
WBC	8.1 * 10 /l
INR	1.1
PT	13 seconds
Na	142 mmol/l
K	4.4 mmol/l
Urea	6.4 mmol/l
Creatinine	89 µmol/l
CRP	5 mg/l
Total cholesterol	3.8 mmol/l
HDL	1.3 mmol/

His ECG shows normal sinus rhythm and rate of 65/min.

What is the most appropriate management for this patient?

A. Outpatient TIA clinic appointment
B. Outpatient CT head and carotid dopplers within the next week
C. Admit for urgent (< 24 hours) CT head & carotid dopplers
D. Thrombolysis
E. Long term clopidogrel

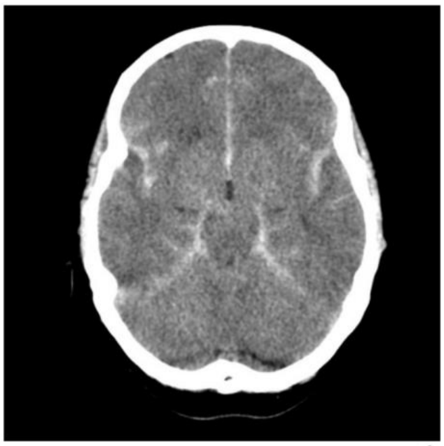
C. Admit for urgent (< 24 hours) CT head & carotid dopplers

This gentleman has had crescendo TIAs (2 in a 7 day period). This necessitates treatment as high risk i.e. admission and urgent (<24 hours) CT head and carotid dopplers. His ABCD score is 3 (1 for age and 2 for symptoms). This would normally put him in the low-risk category (see below) but the presence of crescendo episodes the ABCD score is irrelevant. Thrombolysis is not appropriate as the neurology has resolved. OP TIA clinic and imaging and dopplers within the week are not appropriate given the crescendo episodes.

Question 89 of 189

A 79-year-old woman is admitted to the Emergency Department via an ambulance after being found in an unresponsive, drowsy state. Her daughter phoned her earlier in the day routinely. At this point she complained of a headache. Upon arriving at her mother's house she found her confused and lying on the floor.

On examination her GCS is 9/15 (M4V3E2), pulse 96/min and blood pressure 140/78 mmHg. A CT head is arranged:



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What is the most likely diagnosis?

A. Epidural haematoma
B. Subdural haematoma
C. Subarachnoid haemorrhage
D. Sagittal sinus thrombosis
E. Cavernous sinus thrombosis

C. Subarachnoid haemorrhage

The CT shows diffuse subarachnoid haemorrhage in all basal cisterns, bilateral sylvian fissures and the inter-hemispheric fissure. This case demonstrates the typical distribution that takes the blood into the subarachnoid space in a subarachnoid hemorrhage.

Question 90 of 189

An 83-year-old female presents to the emergency department. The patient describes a vague history of arm and leg weakness and loss of vision though is unsure of a particular limb or visual field distribution. She states that these symptoms seemed to last around 6 hours. There are no abnormalities noted in the upper and lower neurological examination nor cranial nerve examination in the emergency department. The patient has a history of regular migraine of around 2 a week for the last 20 years, type two diabetes mellitus treated with metformin and hypertension which was diagnosed 8 years ago currently treated with amlodipine.

Her observations are.

- Blood pressure - 145/90 mmHg
- Heart rate - 83 beats per minute
- Temperature - 37.5C
- Respiratory rate – 15
- Oxygen saturation - 98% on air

You suspect the patient may have had a transient ischaemic attack.

Which radiological investigation is the most appropriate to aid diagnosis at this stage?

A. MRI head with fluid attenuation inversion recovery (FLAIR)
B. CT head with contrast
C. Carotid dopplers
D. MRI head
E. MRI head with diffusion weighted imaging

E. MRI head with diffusion weighted imaging

This patient is an example of someone who has had a suspected transient ischaemic attack (TIA) who requires urgent brain imaging.The national institute of clinical excellence (NICE) state that patients with a suspected TIA who are at a high risk of stroke (ABCD2 score of 4 or above) where the pathology or vascular territory is uncertain require urgent brain imaging, preferably diffusion weighted MRI.

The patient in this question has an ABCD2 score of at least 5, the vague clinical history also make the vascular territory uncertain. Therefore diffusion weighted MRI is most suitable. She may require a Doppler ultrasound of her carotid arteries in the future, though this would preferably happen after the MRI.

Question 91 of 189

A 72-year-old lady was admitted with a cough productive of green phlegm, shortness of breath and a low-grade fever. Past medical history included Parkinson's disease for which she was on cocareldopa and hypertension. Chest x-ray showed a right basal consolidation and she was treated for pneumonia with oral antibiotics. As a result of the pneumonia she had a poor appetite and the patient had been refusing to take her medication.

She was given intravenous fluids and encouraged to take her oral antibiotics. Over the next 1-2 days, the nurses noted that she had started to have fever spikes of greater than 38ºC, had developed a tremor and was becoming increasingly rigid, agitated and confused. Her blood pressure had also been extremely variable.

What test would help you confirm the diagnosis?

A. Repeat chest x-ray
B. Creatine kinase
C. Blood cultures
D. CT head
E. Urine sample

B. Creatine kinase

This patient has developed neuroleptic malignant syndrome (NMS) as a result of not taking her Parkinson's medication. NMS is a life-threatening neurological disorder most often caused by an adverse reaction to antipsychotic drugs (e.g. olanzapine, risperidone, aripiprazole, amisulpride, quetiapine, chlorpromazine, haloperidol and clozapine). However, a wide range of drugs can also cause NMS. These include dopaminergic medication i.e. levodopa and this often occurs when these medications are stopped abruptly, anti-dopaminergic medication such as metoclopramide and even drugs without anti-dopaminergic effects such as lithium can also cause NMS. NMS is caused by a sudden, reduction in dopamine activity, either from blockade of dopamine receptors or withdrawal of dopaminergic agents.

NMS should be suspected if a patient has muscle rigidity, muscle cramps, fever, autonomic instability and alterations in their mental status (confusion, agitation, coma). Symptoms can last anywhere from eight hours to forty days. These features are coupled with an elevated plasma creatine kinase (CK). A raised white cell count may occur and patients can develop rhabdomyolysis.Metabolic acidosis and hypertensive crisis have also been reported.

Treatment is needed urgently as death can occur if untreated. Culprit medication should be stopped. Hyperthermia can be treated with fan therapy, cold blankets and ice packs. Dantrolene is the treatment of choice, however, amantadine and bromocriptine are also used. Patients may require supportive care in an intensive care unit to help support capable of ventilation and circulation.

Question 92 of 189

A 56-year-old female was admitted to the Emergency Department with a 90-minute history of new onset weakness in her left arm and leg. She stated that she was out shopping when the weakness suddenly came on. Since the onset of the weakness she has noticed some regained strength but still felt definite weakness. She had a past medical history comprising new atrial fibrillation for which she was undergoing investigation, hypertension, hypercholesterolaemia and asthma. She also had a perforated gastric ulcer 16 years ago. She was prescribed amlodipine 5mg OD, bisoprolol 2.5mg OD, atorvastatin 20mg OD, Clenil modulite 200mcg BD and aspirin 75mg OD. She declined formal anticoagulation regarding her atrial fibrillation. When questioned specifically, she denied the presence of any visual loss, headache, vomiting, loss of consciousness or cardiac symptoms. She smoked 20 cigarettes per day and did not consume alcohol.

On examination she was alert and walking with a hemiplegic gait. Her blood pressure was 168/74 mmHg, heart rate 78bpm, respiratory rate of 18/min, temperature of 37.2 C and oxygen saturations of 99% on air. Other than an irregularly irregular pulse, examination of the cardiovascular, respiratory and gastrointestinal systems were unremarkable.

Examination of the central neurological system revealed normal cranial nerves 2-12, with equal and reactive pupils, normal fundoscopy and a GCS of 15. Examination of the peripheral nervous system revealed the presence of power 3/5 in all muscles of the left upper and lower limbs, with decreased tone and absent deep reflexes. Power was otherwise 5/5 in all other muscle groups, with downgoing plantar reflexes. Sensation and coordination testing was unremarkable.

Initial investigations revealed the following results:

Hb	179 g/l
MCV	99 fl
Platelets	452 * 10 /l
WBC	12.2 * 10 /l

ECG: atrial fibrillation 74 bpm no acute changes

CT head scan: no evidence of intracranial haemorrhage, mass shift or space occupying lesions

What is the next best management step?

A. Commence intravenous heparin
B. Commence thrombolysis therapy
C. Arrange urgent haematology consult
D. Commence aspirin 300mg and admit to stroke unit
E. Commence immediate amlodipine 5mg OD

B. Commence thrombolysis therapy

This lady has suffered an ischaemic stroke and fulfils the criteria for urgent thrombolysis. There are no absolute contraindications to thrombolysis and this will maximize the probability of regaining full function of the affected limbs. The other management options may serve a role but in the hyperacute setting of the above options initiating thrombolysis is the most appropriate management option.

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A 69-year-old retired accountant was seen in the neurology clinic having been referred by his GP. He presented with a three-month history of malaise and weakness. The weakness initially affected his legs, and at times he felt that his legs would not support him standing up. Over the course of the last month, however, he also noticed weakness in his hands, especially his left hand. This was particularly noticeable when he rode his motorbike when he felt that his fingers were not strong enough to apply the brakes. In addition, his wife remarked that his speech had changed in tone though he did not have any difficulties with understanding or expressing speech. His past medical history comprised hypertension, hypercholesterolaemia, depression and hypothyroidism. His medication regimen included amlodipine 5mg OD, atorvastatin 20mg ON, citalopram 20mg OD and levothyroxine 125mcg OD. He also remarked that he had been referred by his GP to the fast track gastroscopy clinic and was pending an appointment for new-onset difficulty in swallowing food over the last month. He did not smoke and did not consume alcohol. Upon a systems review, he denied the presence of other symptoms though he did admit to losing 3kg of weight since the onset of his symptoms.

On examination, he appeared cachectic with obvious dysarthria. His blood pressure was 128/78 mmHg and his heart rate was 78bpm. Examination of his cranial nerves revealed the presence of tongue fasciculations with tremulous lips and an absent jaw jerk reflex but was otherwise unremarkable. Examination of his peripheral nervous system revealed small muscle intrinsic wasting of both hands, as well as wasting of his thigh muscles. The tone was reduced in his lower limbs but increased in his upper limbs. He had obvious weakness of all muscle groups, with a grade 4/5 weakness in his lower limbs and proximal upper limb and a grade 3/5 weakness in his distal upper limbs. Fasciculations were present in his thighs and forearms. Both upper and lower limb reflexes were brisk with downgoing plantars. Examination of sensation was normal. Examination of the cardiovascular, respiratory and gastrointestinal systems was unremarkable.

Investigations conducted by the GP prior to referral are as follows:

Hb	111 g/l
Platelets	99 * 10 /l
WBC	12.2 * 10 /l
ESR	18 mm/hr
CRP	6 mg/l
ANA	positive
Rheumatoid factor	negative

Which one of the following interventions is most likely to be of benefit?

A. Mitoxantrone
B. Riluzole
C. Rivastigmine
D. Methotrexate
E. Azathioprine

B. Riluzole

This patient has motor neurone disease (MND), with clinical evidence of amyotrophic lateral sclerosis and bulbar palsy. Of the above options only riluzole, an anti-glutamate, has been shown to be of any benefit and is suitable for the amyotrophic lateral sclerosis variant of MND. The presence of ANA antibodies is a red herring; it is thought to be present in up to 15% of healthy individuals and is not associated with MND.

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A 60-year-old male with a history of hypercholesterolaemia is brought to the emergency department with a 3-hour history of right sided body weakness and an inability to talk. The weakness was gradual in onset and has been progressing thereafter.

The patient has a history of peptic ulcer disease secondary to H.Pylori infection. He was treated with omeprazole, clarithromycin and amoxicillin-clavulanic acid. A recent upper GI endoscopy done 2 months ago was normal.

His medication includes omeprazole 20mg daily, atorvastatin 40mg daily and he takes paracetamol occasionally for joint pains.

On examination, he has a blood pressure of 140/80mmHg. He is aphasic, the tone on the right side is increased with the muscle power being 2/5 in the right upper limb proximally and distally while it is 1/5 in the lower limb both proximally and distally. There is no evidence of meningeal irritation and his pupils are normal.

Lab investigations reveal

Hb	150 g/l
Platelets	450 * 10 /l
WBC	13.0 * 10 /l
Na	138 mmol/l
K	4.4 mmol/l
Urea	6.9 mmol/l
Creatinine	118 µmol/l

ECG: Sinus tachycardia with occasional ventricular ectopics

CT scan brain: no evidence of cerebral haemorrhage, suspected left middle cerebral artery infarction.

What is the most appropriate management option for this patient?

A. Aspirin, clopidogrel, IV fluids and bowel and bladder care
B. Thrombolysis with alteplase
C. Modified release dipyridamole
D. Anticoagulation with LMWH
E. Anticoagulation with apixaban

B. Thrombolysis with alteplase

The patient has developed an ischaemic stroke and the question assesses the most appropriate management options. Evidence supports the role of thrombolysis in patients with a proven ischaemic stroke of less than 4.5 hours duration.

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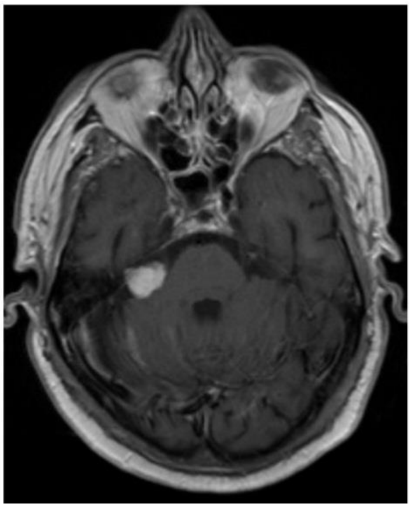
A 75-year-old woman presents to the clinic with a six-month history of lower limb weakness and difficulty rising from a chair. The weakness is more pronounced in the morning and shows temporary improvement after exercise. She has been diagnosed with ovarian carcinoma for which she is receiving palliative care. On examination, her deep tendon reflexes are absent, but it is observed that they reappear after brief periods of exercise following her admission to an inpatient facility.

Which antibodies are most likely to be positive in this patient?

A. Anti Hu
B. Aquaporin 4
C. NMDA receptors
D. Voltage-gated calcium channels
E. Voltage-gated sodium channels



D. Voltage-gated calcium channels

Lambert-Eaton syndrome - antibodies against the voltage-gated Ca2+ channels

Voltage-gated calcium channels is the correct answer. The clinical presentation of this patient suggests Lambert-Eaton myasthenic syndrome (LEMS), which is often associated with malignancies, most commonly small cell lung cancer and, in very rare instances, ovarian cancer as highlighted by a case report. The pathogenesis of LEMS involves autoantibody production against presynaptic voltage-gated calcium channels within the peripheral nervous system. This autoimmune response leads to reduced acetylcholine release and subsequent muscle weakness. A characteristic feature of LEMS is post-tetanic facilitation, an improvement in reflexes following exercise, which serves as a strong diagnostic indicator for the syndrome. The antibodies in question specifically target voltage-gated calcium channels, thereby disrupting normal calcium influx and diminishing acetylcholine secretion at nerve terminals.

The role of voltage-gated sodium channels is not relevant in this scenario since it is the calcium channels that are targeted by autoantibodies in cases of LEMS.

Although the patient's history of ovarian cancer might raise suspicion for anti-NMDA receptorencephalitis, this condition usually presents with acute psychiatric symptoms and behavioural changes that are inconsistent with the clinical presentation observed here; therefore, NMDA receptors are an incorrect choice. Moreover, post-exercise facilitation of deep tendon reflexes is not a feature of NMDA encephalitis.

Anti-Hu antibodies serve as markers for various paraneoplastic neurological syndromes such as limbic encephalopathy and sensory neuronopathy. These conditions do not correspond with the clinical findings discussed here. Anti-Hu antibodies are predominantly linked to small-cell lung cancer, with approximately 85% of positive cases being associated with this type of malignancy.

Lastly, neuromyelitis optica (NMO) is characterised by aquaporin 4 antibodies; however, there are no clinical signs or symptoms indicative of NMO present in this patient's case. Therefore, selecting aquaporin 4 as an answer would be incorrect.

Question 96 of 189

A 60-year-old schoolteacher is referred to the neurology clinic by his General Practitioner to assess his symptoms further. He has an MRI brain before review in the neurology clinic. The MRI brain showed

What is the most likely diagnosis?

A. Cavernous hemangioma
B. Facial nerve schwannoma
C. Infratentorial meningioma
D. Trigeminal schwannoma
E. Vestibular schwannoma

E. Vestibular schwannoma

The MRI brain shows an intracanalicular solid nodular mass with an intense contrast enhancement in the left internal auditory meatus (IAM) suggestive of vestibular schwannoma. Occasionally, cystic degeneration or haemorrhagic areas may also be seen, but calcification is typically not present.

Vestibular schwannoma is the correct answer. Vestibular schwannomas, or acoustic neuromas, are relatively common tumours arising from the vestibulocochlear nerve (CN VIII) and represent ~80% of cerebellopontine angle (CPA) masses. The classical presentation for vestibular schwannoma includes tinnitus, sensorineural hearing loss, vertigo and absent corneal reflex. Cranial nerves V and VII may also be affected.

Cavernous hemangiomas, also commonly known as cerebral cavernous venous malformations or cavernomas, are common cerebral vascular malformations, usually with characteristic appearances on MRI. MRI often demonstrates a characteristic 'popcorn' or 'berry' appearance with a rim of signal loss due to hemosiderin. The patients usually remain asymptomatic but may present with headaches and focal neurological deficits from intracranial haemorrhage.

Facial nerve schwannoma, or facial nerve neuroma, is a rare schwannoma that arises from the facial nerve. They are generally uncommon and make up less than 1% of all temporal bone tumours when involving the temporal bone.

Infratentorial meningiomas are rare even though meningiomas, in general, are the second most common cause of a cerebellopontine mass. They typically appear as extra-axial masses with a broad dural base and are usually homogeneous and well-circumscribed. Patients often present with headaches, paresis and changes in mental status.

Trigeminal schwannomas are uncommon slow-growing encapsulated tumours of Schwann cells. They are the second most common intracranial schwannoma, far less common than vestibular schwannoma, and have a predominantly benign growth.

Question 97 of 189

A 74-year-old patient with known Parkinson's disease presents for review in the geriatric clinic. He has been suffering from sudden onsets of attacks where he is barely able to move. This has been a problem for the last few months. His symptoms are normally well controlled and attacks occur suddenly, lasting for 30 minutes and then resolving without any intervention. He takes levodopa and carbidopa for his Parkinson's disease which has been on a stable dose for the last three years. On examination, he has a mild unilateral tremor and slight bradykinesia which is worse on the right side. His eye movements are normal and he has no postural symptoms when standing.

What is the likely explanation of his symptoms?

A. Dyskinesia
B. Vascular Parkinsonism
C. Multi-system atrophy
D. Progressive supranuclear palsy
E. On-off effect

E. On-off effect

The correct answer is on-off effect. Over time, levodopa becomes less effective. Patients can experience after a few years of treatment that their symptoms are well-controlled parts of the day and other times their mobility is heavily affected. This is the most likely explanation given the fact that these symptoms started over two years after treatment and the medication regime will need to be adjusted. A Parkinson-plus syndrome is unlikely as these would not have originally responded to levodopa, and would be associated with other features. For example, progressive supranuclear palsy is associated with gaze palsy whilst multi-system atrophy is associated with postural hypotension. Dyskinesia is continuous writhing movements which occur as a side effect of levodopa.

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A 35-year-old man is brought into the Emergency Department after 3 successive tonic-clonic seizures. He was given 10mg of rectal diazepam and has since stabilised.

A brief history from his girlfriend states that he was usually fit and well apparent from recurrent sinusitis. Over the past month, however, he had been complaining of increasing headaches and had received a course of antibiotics from his GP.

He takes no regular medication but his partner states that they do occasionally take ecstasy whilst on nights out.

On examination, he is drowsy with a Glasgow Coma Scale (GCS) of 12. His temperature is 38.8 degrees Celsius, his pulse is 57 bpm and regular, blood pressure 150/90 mmHg, oxygen saturations 97% on 15L oxygen via a non-rebreathe mask.

Cardiovascular examination reveals normal heart sounds, capillary refill time of 3 seconds. His calves were soft and non-tender. His chest was clear with no signs of consolidation. Abdominal examination was unremarkable.

Neurological examination was difficult in view of the patients low GCS but no focal abnormality could be detected. On attempt to passively flex his neck he became agitated and obviously uncomfortable. Pupils were equal and reactive to light. Fundoscopy demonstrated bilateral oedematous optic discs.

Bloods were as follows:

Na+	130 mmol/L
K+	3.9 mmol/L
Urea	5 mmol/L
Creatinine	80 µmol/L
Hb	160 g/L
WBC	25.0x10^9/L
Neutrophils	91%
LFTs	Normal
CRP	90 mg/L

Based on the findings above, what is the most likely diagnosis?

A. Intracerebral abscess
B. Sepsis secondary to sinusitis
C. Ecstasy overdose
D. Meningitis
E. Cerebral lymphoma

A. Intracerebral abscess

This is a 30-year-old man who presented with a history of headaches, seizures and a fever. The history of headaches and a course of antibiotics could suggest partially treated sinusitis. This has likely extended posteriorly resulting in an intracranial abscess as indicated by the inflammatory markers, signs of raised intracranial pressure and seizures.

Meningitis can sometimes present with seizures if this has progressed to meningoencephalitis but the history of recurrent sinusitis and headaches with raised intracranial pressure are more suggestive of a space occupying abscess.

Although ecstasy overdose can cause pyrexia and seizures, it is not in keeping with the chronic progression of this patient's symptoms.

Seizures have been associated with severe sepsis but it is not in keeping with all of the signs and symptoms demonstrated here.

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A 79-year-old is brought into hospital with muscle cramps, fevers and passing dark urine. He is known to have previously had Parkinson's disease and takes Sinemet 125 five times a day. His daughter, who normally picks up his medicine for him from the pharmacy has been called away on business for the past 5 days. As a result, the patient's supplies ran out and he has not taken his PD meds for 3 days. His blood pressure is fluctuant, from 77/52 mmHg to 150/88mm Hg. On examination, his temperature is 39.2 degrees, his heart sounds are quiet but present and chest auscultation is unremarkable. You note rigid muscles in four all limbs, no obvious superficial evidence of head injury and new confusion, with abbreviated mental test 0/10. You start intravenous fluids, intravenous broad spectrum antibiotics, catheterise the patient and insert a nasogastric tube to administer his regular medications.

What is the underlying diagnosis?

A. Urosepsis
B. Restrictive pericarditis
C. Idiopathic Parkinson's disease progression
D. L-dopa dyskinesia
E. Neuroleptic malignant syndrome

E. Neuroleptic malignant syndrome

The history gives a clear history of lack of access to regular Parkinsonian medications associated with pyrexia and muscle cramps. This would point towards a diagnosis of Parkinsonismhyperpyrexia syndrome, a form of neuroleptic malignant syndrome observed when PD medications are withdrawn or changed. A key feature to be noted here is the labile blood pressure, suggesting autonomic instability, suggesting the need for careful intensive care monitoring and supportive therapy. Treatment is by replacing the original dose of withdrawn medication, either orally or via a nasogastric tube. If this is not possible intravenous L-dopa is possible while dopamine agonists can be given parenterally: rotigotine can be applied as a transdermal patch while apomorphine is available as an intravenous or subcutaneous infusion.

Question 100 of 189

A 58-year-old female with known myasthenia gravis complains of increasing tiredness and reducing exercise tolerance over the past 3 days. During your examination, she has a weak voice is unable to finish her sentences and appears slightly slumped in the chair she is sitting in.

What is the most important investigation at this acute stage?

A. Forced vital capacity (FVC)
B. 12 lead ECG
C. Forced expiratory volume in 1 second (FEV1)
D. Blood tests including full blood count, U+Es, LFTs, CRP
E. Chest x-ray

A. Forced vital capacity (FVC)

The patient is approaching a myasthenic crisis. One of the most acute dangers to be aware of is impending respiratory distress, caused by a failure of respiratory muscles. Other symptoms can include dysphonia, dysphagia and generalised weakness. Forced vital capacity is the most accurate assessment of inspiratory and expiratory mechanisms. FEV1 is better at demonstrating an obstructive than respiratory muscle strength. Although blood tests and chest radiography may be useful in identifying an underlying infective cause for the deterioration, the acute issue remains the patients possible progressive respiratory deterioration, which may warrant elective intubation.

Question 101 of 189

A 22-year-old male is referred to clinic by his GP. His initial complaint had been that his friends have been increasingly remarking on how his eyelids look increasing 'droopy' over the past 9 months. He denies any diplopia or muscle weakness. He has no past medical history, is a nonsmoker and non-drinker. On examination, you note bilateral inability to abduct or adduct his eyes. Vertical gaze is also inconsistently impaired. His upper and lower limb neurological investigation was unremarkable except very mild finger-nose dysmetria, his bloods tests show no abnormalities. His ECG demonstrates sinus rhythm with a PR interval of 260ms. Fundoscopy reveals a pigmented retina.

What is the diagnosis?

A. Myasthenia gravis
B. Thyroid eye disease
C. Oculopharyngeal musculodystrophy
D. Extraocular muscle fibrosis
E. Kearns-Sayre syndrome

E. Kearns-Sayre syndrome

This young patient demonstrates a combination of retinitis pigmentosa, chronic progressive external ophthalmoplegia, bilateral ptosis, mild cerebellar signs and a cardiac conductive defect (first degree heart block). These features are consistent with a mitochondrial disorder, in particular Kearns-Sayre syndrome. Some patient may also display proximal myopathy, cognitive impairment or short stature. The underlying cause is a genetic mitochondrial disorder. Patients rarely live beyond their 40s and there are no therapeutics currently available.

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A 23-year-old male presents with a 5-day history of increasing shortness of breath, exertional dyspnoea and bilateral ankle swelling. He has no previous past medical history and was previous a keen sportsman. On examination, his heart sounds I + II are present with a pansystolic murmur and a displaced apex. Auscultation of his chest demonstrates bibasal crackles. His ECG demonstrates a PR interval of 190 ms but sinus rhythm. Further neurological examination demonstrates bilateral partial ptosis noted associated with bilateral foot drop. You also note slow release of finger flexion.

What is the underlying diagnosis?

A. Myotonic dystrophy
B. Myasthenia gravis
C. Inflammatory myositis
D. Facioscapulohumeral dystrophy (FSD)
E. Guillain Barre syndrome

A. Myotonic dystrophy

The patient has presented with clinical heart failure on a background of systemic disease, involving structural heart disease, conductive heart block, bilateral ptosis bilateral foot drop and slow finger flexion release suggestive of myotonia. It would not be possible to distinguish at this stage between DM1 or DM2 myotonic dystrophy, the latter being less severe than DM1. The diagnosis would be clinched with genetic testing for an expanded CTG repeat in the DMPK gene: normally, 5-34 CTG repeats are found. Greater than 50 alleles is strongly diagnostic of disease manifestations.

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An 84-year-old male presents as a blue light ambulance call with a twelve hour history of sudden onset inability to move his right side. On examination, you note an expressive and receptive dysphasia associated with a dense right sensori-motor syndrome. Cardiovascular examination was unremarkable except for an irregular heartbeat at 80 per minute. A hyperacute CT head demonstrated a large area of ischaemia in a the left middle cerebral artery vascular territory. The patient was outside the window for thrombolysis and started on 300mg aspirin. Subsequent echocardiogram demonstrated 60% ejection fraction with no mural thrombus, 40% left and 35% right stenosis on carotid Doppler while a 24hr tape demonstrated new atrial fibrillation.

How can the risk of subsequent strokes be reduced?

A. Subcutaneous low molecular heparin at 48 hours after stroke
B. Warfarinisation at 48 hours after stroke
C. Warfarinisation at 14 days after stroke
D. Referral to vascular surgery for left carotid endarectomy
E. Insertion of permanent pacemaker

C. Warfarinisation at 14 days after stroke

This patient demonstrates an ischaemic infarct secondary to likely new atrial fibrillation, for which the optimum treatment is anticoagulation. The degree of carotid stenosis is not sufficiently significant to justify carotid endarectomy. The patient has no indications for a pacemaker. The treatment of choice for atrial fibrillation in such a high-risk patient for further strokes is anticoagulation.

In the absence of intracerebral haemorrhages or haemorrhagic transformation of infarcts, NICE guidelines recommend starting anticoagulation at 14 days after the onset of stroke . Due to the risk of haemorrhage as the infarcted brain matures, which is increased with the size of the initial infarct, early anticoagulation is not recommended. A recent meta-analysis collated the results of seven trials studying early anticoagulation in acute ischaemic strokes. Although the risk of further ischaemic strokes between days 7 and 14 is significantly reduced from 4.9 to 3%, the risk of symptomatic intracerebral haemorrhage was also significantly increased fro 0.7% to 2.5% . As a result, early anticoagulation before 14 days is not indicated by NICE or the American Heart and Stroke Associations .

Question 104 of 189

A 55-year-old female presents with 3 weeks of bilateral tingling sensation in her medial one and half digits at night. She has noted a clawing of her 4th and 5th digits and she is particularly concerned by the cosmetic elements. She also complains of a left sided foot drop present over the past 8 months. Her past medical history includes type 2 diabetes mellitus, for which she take metformin 850mg TDS and she admits to occasional poor compliance. Her last HbA1c was 53 mmol/mol. She has also had multiple admissions for surgery to her feet at childhood but she is unaware of further details. She was adopted and is unaware of her birth family history. On examination, she clinically has a left common peroneal palsy with bilateral thin calves, and loss of sensation in bilateral ulnar nerve territories.

What is the unifying diagnosis for her presenting paraesthesia and foot drop?

A. Hereditary neuropathy with liability to pressure palsies
B. Diabetic neuropathy
C. Chronic inflammation demyelinating polyneuropathy (CIDP)
D. Systemic lupus erythematosus (SLE)
E. Sarcoidosis

A. Hereditary neuropathy with liability to pressure palsies

The patient has thin calves and previous foot deformities requiring surgery, suggestive of CharcotMarie-Tooth disease or hereditary motor sensory neuropathy (HSMN), a disorder caused by deletion in the PMP22 gene, the same gene mutation responsible for hereditary neuropathy with liability to pressure palsies. Common peroneal nerve is the most commonly affected nerve (36%) followed by the ulnar nerve (28%). Diagnosis is confirmed by genetic testing.

Question 105 of 189

A 32-year-old female presents with four episodes of loss of consciousness within the past 4 weeks. She denies palpitations or chest pain but reports sudden onset binocular black dots in visual fields, occasional flashing lights, dysarthria and hearing loss, all of which resolves after about 60 minutes. She is unsure about the relevance of an occipital headache, onset with frequency of about three times per week for the past year. She denies any limb weakness, altered sensation or facial droop. She has no past medical history or family history of epilepsy. Your neurological examination, including fundoscopy is unremarkable. An EEG is unremarkable.

What is the likely diagnosis?

A. Cluster headache
B. Transient ischaemic attacks
C. Basilar migraine
D. Bilateral retinal detachment
E. Guillain-Barre syndrome

C. Basilar migraine

The symptoms are most likely to represent a rare migraine subtype with aura known as basilar migraine. A large Danish study identified up to 10% of all non-hemiplegic migraineurs as basilar migraines, most of whom report a brainstem-associated range of symptoms lasting for around one hour . 61% of patients report vertigo and over half dysarthria. One in four report episodes of loss of consciousness. Investigations include 24 hour tape and echocardiogram to exclude cardiac syncope and MRI to exclude posterior fossa space occupying lesion. An EEG may not exclude posterior epileptiform activity. Prevention can be offered with verapamil or topiramate.

Question 106 of 189

A 76-year-old male is referred by the GP to your Parkinson's disease clinic. The patient and his wife described an increasing frequency of falls, the most frightening of which occurred when he fell 4 steps down a flight of stairs last week, luckily with no lasting damage. On

examination, you note a slow pill rolling tremor equally on both hands and bilateral cog-wheeling. His cranial nerves are unremarkable except a poverty of upwards gaze. His speech appears distinct and nasal in character.

What is the most likely diagnosis?

A. Idiopathic Parkinson's disease
B. Vascular parkinsonism
C. Progressive supranuclear palsy (PSP)
D. Multi-system atrophy (MSA)
E. Corticobasal degeneration (CBD)

C. Progressive supranuclear palsy (PSP)

PSP is most likely in this scenario. A number of features should alert you to a Parkinson's-plus syndrome instead of idiopathic Parkinson's disease: bilateral, symmetrical symptoms are uncommon, particularly in early PD. In addition, eye signs are very unusual. PSP patients classically present with increasing falls near the stairs due to an impaired vertical gaze and a nasal 'DonaldDuck' voice secondary to pseudobulbar palsy. MSA patients classically present with significant cerebellar or autonomic symptoms. CBD present with higher order dysfunction such as apraxia and aphasia, but classically with 'alien hand syndrome', thought to be as prevalent as 60% of CBD patients, when the patient has no control over their own hand movements.

Question 107 of 189

A 32-year-old male presents with a sudden onset left sided weakness with new onset expressive dysphasia and dress apraxia. He has a seven year history of progressive cognitive impairment and seizures, lives in sheltered accommodation and was brought in after his relatives, who visit him on a weekly basis, noted a change from his baseline. The patient is a poor historian and could not remember the duration of symptoms. He reports a recent history of burning sensation when passing urine associated with increased frequency and reduced oral intake for the past four days. An MRI head demonstrates multiple areas of ischaemia within left and right cortex inconsistent with one single vascular territory. A urine dip is positive for leucocytes and nitrites, negative for ketones. A venous blood gas is taken:

pH	7.15
PaCO	2.4 kPa
Bicarbonate	6 mmol/l
Lactate	18 mmol/l
Anion gap	16 mmol/

What is the unifying diagnosis?

A. MELAS
B. Diabetic ketoacidosis
C. Ingestion of antifreeze
D. Severe dehydration secondary to urosepsis
E. Metformin induced lactic acidosis

A. MELAS

Young patients with significant cognitive impairment should raise suspicion. This patient presents with a significant lactic acidosis, with low bicarbonate likely secondary to chronic neutralisation in the presence of systemic acidosis. Multiple ischaemic infarcts inconsistent with vascular territories are suspicious of a metabolic disorder within the brain parenchyma. In this case, a mitochondrial genetic condition, mitochondrial encephalopathy with lactic acidosis and stroke like episodes (MELAS), is most likely. Focal and generalised seizures are common for MELAS patients, as is early onset dementia, focal weakness and cortical blindness from multiple ischaemic stroke episodes.Symptoms normally present at late childhood and early adulthood, with patients develop normally until then. Diagnosis is made through lactic acidosis, a progressive neurological and dementing clinical course and muscle biopsy for ragged red fibres. The majority of patients have been linked to an underlying mutation in transfer RNA .

In this case, the UTI is a red herring. While the patient may have a UTI, it does not produce a unifying diagnosis of cerebral and metabolic abnormalities. A normal anion gap rules out ingestion of inorganic acids or DKA. The patient is negative for urinary ketones. There is no suggestion he takes metformin.

Question 108 of 189

An 18-year-old man presents to the emergency department with pleuritic chest pain. He has no past medical history.

The examination is unremarkable.

A chest x-ray reveals a visible pleural edge with a distance of approximately 1.1cm from the pleural edge to the lung margin at the level of the hilum.

Given the likely diagnosis, what advice should he be given about flying?

A. He can fly 1 week post treatment with a chest x-ray demonstrating resolution
B. He can fly 6 weeks post treatment with a chest x-ray demonstrating resolution
C. He can fly immediately upon clinical improvement
D. Indefinite ban on flying
E. There is no contraindication to flying at any point

A. He can fly 1 week post treatment with a chest x-ray demonstrating resolution

Pneumothorax -> can fly 1 week after treatment if chest x-ray shows resolution

He can fly 1-week post-treatment with a chest x-ray demonstrating resolution is the correct answer. The patient has a small PTX < 2cm without any lung disease history. He is likely to be suitable for discharge and outpatient review. He can be advised that he can fly one week post any required treatment if a chest x-ray demonstrates resolution.

He can fly 6 weeks post-treatment with a chest x-ray demonstrating resolution is incorrect. This was previous BTS advice but has now been replaced by the above advice.

He can fly immediately upon clinical improvement is incorrect. We need to see chest x-ray evidence of resolution prior to flying. This would be more suitable advice if he had a pneumonia.

Indefinite ban on flying is not correct. A prior history of resolved PTX is not a contraindication to flying.There is no contraindication to flying at any point is incorrect. An active, unresolved PTX is an absolute contraindication to air travel.

Question 109 of 189

A 45 year old male presented with right shoulder pain and weakness. Three weeks ago he had been brought to the emergency department (ER) with the same complaint. The pain was so severe that it awoke him from sleep. At that time he took two paracetamol tablets but that gave him no relief. Eventually the ER team had been forced to give him morphine to relieve his agony having tried less potent analgesia. On further questioning he described the pain as sharp, exacerbated by movement of the shoulder, with numbness at the shoulder tip.

On examination, he was holding his right arm with his left one in adduction and internal rotation. Wasting of the deltoid muscle had been noted. The biceps tendon jerk was absent. Sensations were intact except in a small region over deltoid muscle. Lower limb examination was normal. One month ago he had a flu. The past medical history was otherwise of no significance.

He admitted to drinking alcohol at least four times a week. He has a ten year old son who is an insulin dependent diabetic.

The following investigations were ordered:

Hb	14 g/dl
Platelets	180 * 10^9/l
WBC	6 * 10^9/l
MCV	85 fl
MCH	0.6 fmol/cell
MCHC	21 mmol/l
Na+	135 mmol/l
K+	4 mmol/l
Creatinine	85 µmol/l
Urea	3.2 mmol/l
ESR	4 mm/hr
ANA	negative

What is the most likely diagnosis?

A. Syringomyelia
B. Brachial neuritis
C. Adhesive capsulitis
D. Polymyalgia rheumatica
E. Cervical radiculopathy

B. Brachial neuritis

Adhesive capsulitis usually presents with shoulder pain and restrictions of movement. It would not cause wasting nor neither lower motor neurological signs. Adhesive capsulitis is usually associated with diabetes and thyroid disorders.

Question 110 of 189

A 58 year old man progressively develops hand clumsiness, gait difficulty and dysphagia over several months. His voice has also become high pitched and nasal. Sensory examination has remained normal throughout. Plantars are up-going, with absent ankle reflexes bilaterally and wasting of the distal leg musculature.

Which treatment has been shown to lengthen survival for the underlying condition?

A. Intravenous immunoglobulin
B. Donepezil
C. Pyridostigmine
D. Non-invasive ventilation (NIV)
E. Steroid

D. Non-invasive ventilation (NIV)

The answer here is motor neurone disease (specifically Amyotrophic Lateral Sclerosis (ALS) in this case). There is mixture of diffuse upper motor neurone features (the up-going planters and pseudo-bulbar 'Donald duck' speech) along with lower motor neurone features (wasting and areflexia) . The giveaway that it is not other causes of mixed upper and lower motor neurone features e.g. cervical spine pathology/syringomyelia/conus medullaris lesion is the bulbar involvement.

NB: Always look for the presence of tongue fasciculations if you pick up a mixed upper and lower motor neurone picture. If they are evident, think ALS rather than these other causes as they are too low down anatomically to involve the tongue (and bulbar muscles).

Management of ALS involves an multidisciplinary team (MDT) approach. Don't forget to organised Speech and Language Team (SALT) assessment: poor swallow and aspiration pneumonia can kill. Additionally, NIV should be considered to treat respiratory insufficiency in ALS, both to lengthen survival and to slow the rate of Forced Vital Capacity (FVC) decline. It has been shown to increase survival by around 205 days. Riluzole has been shown to slow disease progression in patients with ALS by around 2-3 months.

Question 111 of 189

A 41 year old lady with a background of vitiligo and pernicious anaemia presents with weakness largely affecting her shoulders and hip muscles. The problem is variable throughout the day dependent on how much activity she has done. She does feel that symptoms are at their worst at the end of the day, when indeed her family also say her voice becomes quieter. Clinical examination demonstrates proximal muscle weakness after repeated testing. Electromyography demonstrates a decline in the amplitude of successive potentials (decremental response). Antibodies to the acetylcholine receptor (AChR) are negative on serum testing. Her CT thorax is normal.

Which of the following is likely to be most helpful in establishing a diagnosis?

A. Anti-smooth muscle antibodies
B. Anti-muscle specific kinase (MuSK) antibodies
C. Anti-striated muscle antibody
D. Anticardiolipin antibodies
E. Anti-transglutaminase antibodies

B. Anti-muscle specific kinase (MuSK) antibodies

The diagnosis here is Myasthenia Gravis (MG). Most (but not all) patients with MG are seropositive for the acetylcholine receptor (AChR) antibody. Antibodies to muscle-specific receptor tyrosine kinase (MuSK), a surface membrane component essential in the development of the neuromuscular junction, have recently been identified and are found in up to 50% of MG patients who are seronegative for AChR antibodies. Emerging data suggests that the patterns of weakness and response to certain treatments may be different from those with AChR positive MG. Antistriated muscle antibody usually positive in MG patients that have a thymoma.

Don't forget to recognise the autoimmune past medical history in this patient. It often gives useful diagnostic clues that the underlying condition is also an autoimmune one.

Question 112 of 189

A 36-year-old lady on natalizumab for relapsing-remitting multiple sclerosis develops left leg weakness. Over 3 weeks this progresses to left hemiparesis and visual impairment. She also notices poor memory and struggles to process numbers at work as an accountant. Examination reveals left-sided 4/5 power, right homonymous hemianopia, and an abbreviated mini-mental state score of 21/30. Routine blood tests are unremarkable. MRI brain shows new multiple confluent lesions in the parietooccipital and right motor white matter areas as well as the left occipital area, with no mass effect or enhancement.

Which of the following tests is likely to be most helpful in establishing a diagnosis?

A. Serum glucose
B. HIV serology
C. JC Virus serology
D. Cerebral angiogram
E. Full blood count (FBC)

B. HIV serology

The diagnosis here is progressive multifocal leukoencephalopathy (PML). This is a demyelinating disease of the CNS characterised by widespread lesions due to brain infection of oligodendrocytes by the JC Virus (JCV). These are visible on an MRI and have a characteristic appearance. The signs correspond to the areas occupied by the lesions. PML occurs almost exclusively in immunocompromised patients such as those with HIV or on immunotherapy. You should consider PML in your differential in such patients when they present with new odd neurology and an abnormal MRI scan.

This question's other aim is to introduce to you one of the newer therapies in multiple sclerosis (MS) called nataluzumab. It is actually very frequently used in MS and indeed now approved for the treatment of relapsing-remitting MS (RRMS) in more than 50 countries. It is a monoclonal antibody that inhibits the migration of leucocytes into the central nervous system, hence reducing inflammation and demyelination. It is given as an IV infusion once a month. It is indicated in patients getting relapses despite treatment with interferon-beta or patients who just have rapidly evolving severe RRMS. It has been shown to decrease the risk of progression of disability by 4254% and the annualised rate of relapse by 68%. The two principle side-effects are hepatotoxicity and increased PML risk, and we monitor patients for these with monthly liver function tests (LFTs) and 6-monthly JCV serology. It would not be unreasonable of them to expect you to know something about nataluzumab in the exam (especially to recognise PML as a side-effect) given that the drug is now in such common use.

Question 113 of 189

A 32 year old female is referred for a neurological opinion. She describes two separate occasions within the last six months of unilateral blurred visual loss. Her symptoms developed over a few days, comprising blurring of the vision and pain, particularly on eye movement. On both occasions this peaked within three days then gradually resolved over two weeks. Lumbar puncture reveals elevated protein level and CSF pleocytosis. Serum Aquaporin-4 is positive.

What is the most likely diagnosis?

A. Multiple sclerosis
B. Neuromyelitis optica
C. Leptomeningeal gliomatosis
D. Myasthenia gravis
E. Idiopathic intracranial hypertension

B. Neuromyelitis optica

Her symptoms are highly suggestive of Neuromyelitis optica (NMO, also known as Devic disease). NMO causes demyelination of the optic nerves or spinal cord due to autoimmune inflammation. Aquaporin-4 (AQP-4) is a water channel protein located in the astrocytic foot processes; NMO-IgG antibody attacks AQP-4 causing demyelination.

NMO typically presents with episodes of optic neuritis or transverse myelitis. The optic neuritis can be clinically indistinguishable from that seen in multiple sclerosis (MS), whereas the transverse myelitis is commonly more of a complete picture than that of MS. This is a result of the fact that the resulting spinal cord demyelination seen in NMO is longer and more extensive.

Question 114 of 189

A 25 year old man presents to A+E in a comatose state. He was found by his university flatmate collapsed on the floor. His flatmate states that the man had been behaving bizarrely in the last 24 hours and had been quite agitated and aggressive at times. When you examine him he has a GCS of 8 (E 2 V 1 M 5). He has a temp of 39.4ºC, heart rate 120/min, blood pressure 178/89 mmHg, sats 98% on room air and respiratory rate 20/min. Chest clear abdomen soft and non-tender, bowel sounds present. Globally greatly increased tone in all 4 limbs.

When looking at his previous electronic notes the only information that you can find is a recent admission to a psychiatric hospital where a diagnosis of paranoid schizophrenia was established.

CT Brain: normal

Hb	15.4 g/dl
Platelets	232
WBC	11.5
Na	143
K	4.1
Urea	8.1
Creatinine	101
Bilirubin	14
ALP	63 u/l
ALT	28 u/l
Calcium	2.64 mmol/l
Albumin	41 g/l
Creatine kinase	21,000 iu/l
Serum glucose	6.4 mmol/l

Lumbar Puncture:

Glucose	4.9 mmol/l
Protein	0.3g/l
Culture	nil organisms found
Opening pressure	21 mmHg

What is the most likely diagnosis?

A. Viral meningitis
B. Serotonin syndrome
C. Neuroleptic malignant syndrome
D. Bacterial meningitis
E. Malignant hyperthermia

C. Neuroleptic malignant syndrome

The answer is neuroleptic malignant syndrome. NMS is characterised by a triad of altered conscious state, hyperpyrexia and raised CK in a person on neuroleptic medications. Young males recently started on a high dose of a high potency antipsychotic are most at risk from this potentially fatal complication. Autonomic instability is also a feature.

Serotonin syndrome shares many clinical features with NMS but normally you would expect to see GI features and cerebellar symptoms in addition and rigidity and hyperthermia are usually more mild. In addition serotonin syndrome is associated with antidepressant medication. This is therefore a less likely diagnosis in an individual recently diagnosed with schizophrenia.

Malignant hyperthermia also shares many clinical features with NMS but this is a genetic disorder provoked by the use of anaesethic agents and there is nothing in the history to suggest this. A normal Lumbar Puncture MC+S essentially rules out bacterial meningitis and viral meningitis is unlikely to cause such severe symptoms.

Question 115 of 189

A 68-year-old female is admitted into the acute medical unit after suffering from a prolonged tonic-clonic seizure. She was diagnosed with status epilepticus which was terminated with lorazepam and a phenytoin infusion. Two days into the admission you are asked to review her due to ongoing confusion. She has a past medical history of depression which is currently treated with fluoxetine.

On examination of her cardiorespiratory system there is nothing of note. Her abdomen is soft and non tender. You note rapid blinking, which the nursing staff confirmed has been on-going over the past 24 hours.

Investigation results are as follows:

Hb	135	Na	137	Bilirubin	14 µmol/l
CRP	<4	Lactate	1.2	Albumin	32 g/l
Platelets	385	K	4.2	ALP	88 u/l
WBC	6.6 *	Urea	4.4	ALT	44 u/l
Neuts	4.2 *	Creatinine	75	γGT	68 u/l
Lymphs	2.2				
Eosin	0.2				

What is the most likely diagnosis?

A. Non-Convulsive status epilepticus
B. Psychogenic seizure
C. Tardive dyskinesia
D. Convulsive status epilepticus
E. Delirium

A. Non-Convulsive status epilepticus

Non-convulsive status epilepticus can present with subtle signs, such as twitching or blinking and/or fluctuating mental statusImportant for me Less important

Non-convulsive status epilepticus (NCSE) is defined as a prolonged (>30 mins) electrographic seizure activity with non-convulsive signs such as twitching or blinking. It can be a challenging diagnosis to make which often relies on EEG confirmation. It requires a high index of suspicion in individuals with risk factors and suggestive clinical features. Early recognition and treatment are essential to optimize response to treatment and to prevent neurological and systemic sequelae.

NCSE is very common in patients who have suffered convulsive status epilepticus, comatosed patients and in the general ICU population.

In this case, the prolonged post-ictal confusion and subtle motor signs are very suggestive of NCSE, and an urgent EEG should be arranged.

There is nothing on examination to suggest infection, and this is reassured with the normal inflammatory markers.

Given the history of depression, a reasonable differential includes a psychogenic seizure, however NCSE would be more likely and more important to rule out.

Tardive dyskinesia can cause blepharospasm however it is rarely caused by SSRIs.

Question 116 of 189

A 34-year-old female attends for review. She has a background of schizophrenia which is currently managed with quetiapine having failed treatment with olanzapine and risperidone in the past. She informs that she has on-going derogatory third person auditory hallucinations. You elect to start her on clozapine.

You perform a baseline blood tests which are as follows:

Hb	145 g/l
Platelets	320 * 10 /l
WBC	6.8 * 10 /l
Neutrophils	3.8 * 10 /

What other investigation is important to perform before starting clozapine?

A. Abdominal x-ray
B. Cardiac echo
C. Liver function tests
D. ECG
E. Urea and electrolytes

D. ECG

Clozapine can cause myocarditis. A baseline ECG should be performed before initiating treatment

Clozapine can cause myocarditis. Therefore, a baseline ECG should be performed before initiating treatment.

Clozapine requires differential white blood cell monitoring weekly for 18 weeks, then fortnightly for up to one year, and then monthly as part of the clozapine patient monitoring service.Prolactin, lipids, glucose, and body weight should also be regularly monitored.

It is important to be aware that clozapine can cause impairment of intestinal peristalsis, potentially resulting in constipation, intestinal obstruction, faecal impaction, and paralytic ileus. However there is no need to perform an abdominal x-ray as a baseline if not clinically indicated.

Question 117 of 189

A 64-year-old woman presents with a painful right eye which came on this evening. On examination, she is notably photophobic with a semi-dilated right pupil. Her visual acuity is 6/6 on the left and 6/12 on the right. Her past medical history includes urinary incontinence for which she takes solifenacin.

What is the definitive treatment for this condition?

A. Acetazolamide
B. Laser peripheral iridotomy
C. Laser trabeculoplasty
D. Pilocarpine
E. Steroid and cyclopentolate eye drops

B. Laser peripheral iridotomy

Laser peripheral iridotomy is the definitive treatment for acute angle-closure glaucoma Important for me Less important

Laser peripheral iridotomy is correct. The clinical features are highly suggestive of acute angle closure glaucoma (AACG). The patient takes an anti-muscarinic (solifenacin) drug which can precipitate AACG. Definitive management of this condition is with laser peripheral iridotomy.

Acetazolamide is incorrect. This drug can be used in the emergency setting to lower intra-ocular pressures, however, the definitive treatment is with laser peripheral iridotomy.

Laser trabeculoplasty is incorrect. Laser trabeculoplasty is performed as an additional measure to lower the intraocular pressure in patients with open-angle glaucoma. It can also be performed in a closed-angle glaucoma patient with a patent iridotomy (e.g. following a laser peripheral iridotomy).

Pilocarpine is incorrect. This drug can be used in the emergency setting to lower intra-ocular pressures, however, the definitive treatment is with laser peripheral iridotomy.Steroid eye drops and cyclopentolate is incorrect. This combination of medications can be used to treat anterior uveitis. Although this condition also presents with an acutely painful red eye, the presence of a semi-dilated pupil favours the diagnosis of AACG. In anterior uveitis, the pupil is classically oval and non-reactive.

Question 118 of 189

A 55-year-old woman is referred to the neurology outpatient department with a 3-month history of headaches. The headaches are localized to her right temporal region. They are sharp and stabbing in nature and occur approximately 15 times a day, lasting for 20 minutes on each occasion. She notices a runny nose and tearing of her eyes when they occur. This has occurred daily for the time period with no period of remission. She has no past medical history and is not on any regular medications.

The examination is unremarkable at the time of review.

What is the likely diagnosis?

A. Chronic paroxysmal hemicrania
B. Cluster headaches
C. Giant cell arteritis
D. Hemicrania continua
E. Migraine

A. Chronic paroxysmal hemicrania

Chronic paroxysmal hemicrania is characterised by multiple (5+) short (5-30 min) headaches centered around the eye. CPH headaches are shorter and more frequent than cluster headaches

Chronic paroxysmal hemicrania is the correct answer. This headache disorder is characterized by multiple (5+) short (5-30 minutes) headaches localized to the orbital, supraorbital or temporal region that are associated with ipsilateral autonomic features (rhinorrhoea, lacrimation).

Giant cell arteritis is incorrect. This typically causes a persistent rather than intermittent headache and is associated with scalp tenderness and other clinical features such as jaw claudication and visual obscuration.

Migraine is incorrect. Migraines usually last from 4-72 hours and occur with less frequency than chronic paroxysmal hemicrania. There may be an aura that precedes the headaches.Cluster headaches is incorrect. While this can also cause unilateral headaches with ipsilateral autonomic features, the headaches are typically longer (usually 1-3 hours) and less frequent (usually twice a day and rarely more than eight a day).

Hemicrania continua is incorrect. This is a continuous unilateral headache characterized by fluctuation in severity. There is no episodes of being headache free like in chronic paroxysmal hemicrania. It is also associated with ipsilateral autonomic features.

Question 119 of 189

A 45-year-old man is brought to the hospital via ambulance with a tonic-clonic seizure. He has a past medical history of epilepsy, gastro-oesophageal reflux and latent TB. His medications include sodium valproate, omeprazole, isoniazid and he commenced carbamazepine as a consequence of poor seizure control 4 weeks previously. He is known to drink moderate-large quantities of grapefruit juice.

At the time of review, he has recovered and his Glasgow coma scale is 15/15. A CT head is unremarkable.

The ingestion of which substance has caused his seizures?

A. Carbamazepine
B. Grapefruit juice
C. Isoniazid
D. Omeprazole
E. Sodium valproate

A. Carbamazepine

Carbamazepine is known to exhibit autoinduction. hence when patients start carbamazepine they may see a return of seizures after 3-4 weeks of treatment

Carbamazepine is correct. This medication exhibits autoinduction, which means that it may become less effective as a consequence. Due to this loss of response, patients may see a return of seizures 3-4 weeks after commencing treatment.

Grapefruit juice is incorrect. This is a cytochrome P450 enzyme inhibitor and therefore would increase rather than decrease the concentration of another drug.

Isoniazid is incorrect. This is a cytochrome P450 enzyme inhibitor and therefore would increaserather than decrease the concentration of another drug.

Omeprazole is incorrect. This is a cytochrome P450 enzyme inhibitor and therefore would increase rather than decrease the concentration of another drug.

Sodium valproate is incorrect. This is a cytochrome P450 enzyme inhibitor and therefore would increase rather than decrease the concentration of another drug.

Question 120 of 189

You are asked for advice from a GP regarding a 52-year-old woman with a diagnosis of Generalised Anxiety Disorder. She is having symptoms of free-floating anxiety for the past 5 years and this is causing work and relationship difficulties. The GP tells you that 5 years ago she was admitted to hospital with epigastric pain and anaemia and required a therapeutic endoscopy. A repeat endoscopy showed a small ulcer at D2 with a small amount of ooze which was treated with adrenaline. She still suffers from occasional heartburn.

She also has a history of alcohol dependency. She drank 20 units of alcohol per day for 20 years but has now cut down to 1-2 units per day. An ultrasound scan of the abdomen showed mild portal hypertension, hepatomegaly and no gastric varices. There is no history of deliberate selfharm or overdoses. She takes Vitamin B Co strong and thiamine 100mg PO OD and omeprazole 20mg PO OD and is compliant with medication currently but has a history of non-compliance with medication.

Which would be the safest therapy for treatment of her generalised anxiety disorder?

A. Clonazepam
B. Diazepam
C. Sertraline
D. Citalopram
E. Imipramine

E. Imipramine

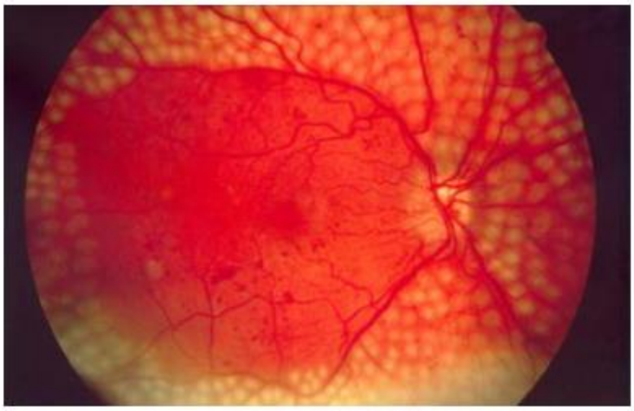
Clonazepam and Diazepam are benzodiazepines. They are not recommended in the treatment of generalised anxiety disorder due to the chronic course of the illness and the dangers of developing tolerance and dependence upon the drug. The risk is even higher in this lady, who has a history of alcohol dependence. In addition, benzodiazepines potentiate the effect of alcohol as they both act on the GABA receptor making it an unsafe medication in someone with a history of alcohol dependency who is still consuming alcohol.

Sertraline and Citalopram are SSRIs. SSRIs inhibit platelet aggregation and are known to increase the risk of upper GI bleeds. This individual has a history of UGI haemorrhage, with recent bleeding duodenal ulcer and continuing symptoms. She also has portal hypertension and continues to drinkalcohol putting him at risk of varices in the future. Whilst being on omeprazole will reduce this risk, there is a history of non-compliance with medication.

Imipramine is a TCA. TCAs are recommended when SSRIs are contraindicated in the treatment of generalised anxiety disorder. There is no increased risk of GI bleeds when on TCAs. It is potentially dangerous in overdose. However, there is no documented history of overdose with this individual

Question 121 of 189

A 67-year-old man with diabetes is attending the outpatient clinic complaining of a headache that has been bothering him for the last week. He reports he experienced some problems with the vision of his left eye two years ago, for which he was treated. Fundoscopic examination of his left eye appears as follows:



What treatment has this man taken for his previous visual problems?

A. Intravitreal ranibizumab
B. Intravitreal voriconazole
C. Laser photocoagulation
D. Oral voriconazole
E. Vitrectomy

C. Laser photocoagulation

Laser photocoagulation is the correct answer. In this fundus picture are dot and blot haemorrhages and neovascularisation of the disc and retina, suggesting background proliferative diabetic retinopathy. There are laser scars in the peripheral retina, which are seen after laser photocoagulation. The pathophysiology, classification and management of diabetic retinopathy are explained in the notes linked below.

Intravitreal ranibizumab is incorrect. Ranibizumab is a VEGF inhibitor, and anti-VEGF agents are used widely to treat some forms of diabetic retinopathy. Indications of anti-VEGF agents in diabetic retinopathy include diabetic macular oedema with visual impairment and proliferativediabetic retinopathy. The patient in this scenario could have had intravitreal ranibizumab administered during his treatment. However, findings in the fundus picture should be considered critical to the solution, making laser photocoagulation the correct answer.

Intravitreal voriconazole is incorrect. This is administered in cases of fungal endophthalmitis involving the vitreous. Fundus examination in cases of fungal endophthalmitis reveals fluffy chorioretinal infiltrates with irregular margins. Diabetic patients have a higher predisposition to develop fungal infections. However, the fundus picture in this case is more suggestive of laser photocoagulation.

Oral voriconazole is incorrect. This is prescribed for conditions such as fungal keratitis. The fundus would not show any specific findings in a case of keratitis, making this an unlikely answer.

Vitrectomy is wrong. Vitrectomy is a procedure employed for treating complications of proliferative diabetic retinopathy like vitreous haemorrhage. Other indications for performing vitrectomy include tractional retinal detachment and progressive neovascularization that does not respond to photocoagulation. The fundus picture shows laser scars, which makes vitrectomy unlikely.

Question 122 of 189

A 26-year-old female presents with a 4-month history of continuous left sided facial pain and frontotemporal headache. She reports it to be constantly present and throbbing in nature, with exacerbations of worsened severity every 3 days. She reports exacerbations to typically be associated with injected left eye and left nasal congestion, with occasional teariness in her left eye.

She has no past medical history or drug history except the oral contraceptive pill. Her routine blood tests are unremarkable. A MRI head organised by her GP also demonstrated no intracranial pathology. A trial indomethacin you have organised is positive.

What is the most likely diagnosis?

A. Short-lasting unilateral neuralgiform attacks with conjunctival injection and tearing (SUNCT)
B. Hemicrania continua
C. Cluster headaches
D. Paroxysmal hemicrania
E. Migraines

B. Hemicrania continua

The patient has a constant headache with exacerbations in the distribution of the left ophthalmic distribution of the fifth cranial nerve associated with autonomic symptoms: this represents one of the trigeminal autonomic cephalgias (TACs). TACs is a class of disorders differentiated by the frequency and duration of attacks, and their response to indomethacin.

SUNCT, paroxysmal hemicrania and cluster headaches present with attacks of approximate duration seconds, minutes to 30 minutes, up to 180 minutes respectively. Symptoms are not present in between attacks. Cluster headaches appear more commonly in men while young women are more predisposed to paroxysmal hemicrania. Hemicrania continua produces pain that is always present with exacerbations in severity. Crucially for diagnosis, only hemicrania continua and paroxysmal hemicrania are responsive to indomethacin, making it a useful diagnostic and therapeutic prophylactic drug.

Question 123 of 189

A 23-year-old investment banking intern presents to the urgent care centre complaining of progressive unsteadiness on walking over the past 6 months. He has no past medical history except type 2 diabetes, which is diet controlled, diagnosed one year ago. He has no family history of any diseases. He has been working very long hours for the past 2 years and reports high levels of stress at work, coupled a culture of 'binge-drinking' to team bond after work. Over the past 3 months, he has noticed a lack of articulation with his speech, which he assumed was secondary to alcohol. He estimates he drinks up to 30 units of alcohol a week. On examination, his cardiovascular, respiratory and abdominal systems are unremarkable. His finger-nose test is impaired bilaterally and is unable to tandem walk. He denies any neck stiffness or headache. He has a full range of eye movements. He has absent reflexes in his lower limbs and upgoing plantars bilaterally.

Which investigation will provide the definitive diagnosis?

A. MRI head with gadolinium contrast
B. CT angiography including posterior vessels
C. Lumbar puncture
D. Serum genetic testing
E. Muscle biopsy

D. Serum genetic testing

The patient describes cerebellar signs of dysarthria, poor coordination and ataxia, in addition to absent ankle jerks and upgoing plantars, presenting at an early age with progressive symptoms. Friedrich's ataxia and other spinocerebellar ataxias must be considered. A diagnosis of type 2 diabetes is odd in such a young patient. However, up to 1/3 of patients with Friedrich's ataxia experience impaired glucose tolerance or diabetes mellitus. These patients often present with cardiomyopathy as well, causing arrhythmias and heart failures.

This patient presents slightly late, most patients have symptoms by childhood. Up to 90% present by 25 years old . It is often assumed that spinocerebellar ataxias, in this case an autosomal recessive one, must be accompanied by a family history. In 40% of Friedrich's ataxia, there is no positive family history at all .

Diagnosis is by serum genetic testing of the GAA trinucleotide repeat. Treatment is limited but involves a multidisciplinary team of neurologists, cardiologists and endocrinologists. There are theories that iron chelation is important in Friedrich's ataxia but these are yet to be proven in large-scale studies2.

Question 124 of 189

A 58-year-old man presents with severe dyspnoea at rest. He has a past medical history of motor neurone disease which was diagnosed 9 months ago. On examination his chest sounds clear. His respiratory rate is 20 breaths per minute with a shallow depth.

You perform an arterial blood gas which is as follows:

PaO2	7.1 kPa
PCO2	6.3 kPa
pH	7.32
HCO3	38 mmol/l

How will you manage this patient?

A. BiPAP
B. CPAP
C. LTOT
D. Palliation of breathlessness with morphine
E. Intubation and ventilation

A. BiPAP

Non-invasive ventilation (NIV) should be the treatment of choice for patients with chest wall or neuromuscular disease causing type 2 respiratory failure. Additional LTOT may be required in case of hypoxaemia not corrected with NIV

The patient has developed type 2 respiratory failure as demonstrated by a PaO2 < 8 kPa and a respiratory acidosis. This is likely due to diaphragmatic weakness secondary to motor neurone disease. Non-invasive ventilation (NIV), in particular BiPAP, should be the treatment of choice for patients with chest wall or neuromuscular disease causing type 2 respiratory failure. Additional LTOT may be required in case of hypoxaemia not corrected with NIV.

Question 125 of 189

A 22-year-old woman presents with a worsening headache. She states that it is worse in the morning and exacerbated by straining. On examination, you note bilateral papilloedema with enlarged blind spots. Visual acuity is 6/6 in both eyes.

Blood results are as follows:

Hb	122 g/L	Male: (135-180) Female: (115 - 160)
Platelets	162 * 10 /L	(150 - 400)
WBC	9.6 * 10 /L	(4.0 - 11.0)
Na	140 mmol/L	(135 - 145)
K	3.9 mmol/L	(3.5 - 5.0)
Urea	5.2 mmol/L	(2.0 - 7.0)
Creatinine	64 µmol/L	(55 - 120)
CRP	2 mg/L	(< 5)
Glucose	5.8 mmol/L	(4-7)

A CT head is organised:

CT head	Papilloedema with flattening of the posterior sclera; bilateral venous sinus stenosis predominantly of the lateral segments of the transverse sinuses

A diagnostic lumbar puncture is organised:

Opening pressure	52 cm H2O	(7-18)
WBC count	3 cells/µL	(0-5)
Glucose	5.4 mmol/L	(> 0.7 x serum glucose)
Protein	22 mg/dL	(<45)

What treatment is indicated?

A. Acetazolamide
B. Optic nerve sheath decompression and fenestration
C. Serial lumbar punctures
D. Topiramate
E. Ventriculoperitoneal shunt

A. Acetazolamide

Acetazolamide is a carbonic anhydrase inhibitor that is used to treat idiopathic intracranial hypertension

The radiological and lumbar puncture results support a diagnosis of idiopathic intracranial hypertension (IIH).

The main principles of management of IIH are: to treat the underlying disease to protect the vision to minimise the headache morbidity

Acetazolamide is correct. The patient does not have an imminent threat to vision and should therefore be commenced on acetazolamide, and advised to lose weight if appropriate.

Optic nerve sheath decompression and fenestration is incorrect. There is evidence that many of the surgical procedures, such as CSF diversion and optic nerve sheath fenestration (ONSF), work well in the short term. However since the patient in this case does not have an imminent threat to vision, then medical management should be implemented in the first instance.

Serial lumbar punctures is incorrect. The relief from a lumbar puncture is typically short-lived as CSF is secreted from the choroid plexus at a rate of 25€‰mL/hour and consequently, the volume removed in a so-called therapeutic tap is rapidly replaced. Therefore, Serial lumbar punctures are not recommended for the management of IIH.

Topiramate is incorrect. Topiramate has carbonic anhydrase activity and can suppress appetite. There is growing evidence suggestive of the efficacy of this drug in treating IIH, however, it currently remains a second-line agent.

Ventriculoperitoneal shun is incorrect. Ventriculoperitoneal (VP) is the preferred CSF diversion procedure for visual deterioration in IIH, due to lower reported revisions per patient.

Question 126 of 189

An 80-year-old man has presented with his son who is concerned he is starting to become more confused and forgetful over the last few months. His son also reports that his previously independent father has required more help with activities of daily living, has lost his appetite and is having frequent falls. He has never drunk alcohol or smoked.

MMSE is 20/30. Upon neurological examination of the lower limbs, there is bilaterally increased tone, 3 out of 5 (MRC scale) global weakness, lack of sensation to light touch and vibration up to the knees, brisk knee-jerks and loss of ankle jerk reflexes and extensor plantar reflexes.

Hb	110 g/L	Male: (135-180)
MCV	110 fL	(80-100)

Nerve conduction studies demonstrate a normal conduction velocity but reduced amplitude.

What is the most likely diagnosis?

A. Hypothyroidism
B. Lead poisoning
C. Multiple sclerosis
D. Pernicious anaemia
E. Tabes dorsalis

D. Pernicious anaemia

When interpreting NCS, a reduced amplitude but normal conduction velocity is indicative of axonal pathology

Pernicious anaemia is the correct option due to being the most common cause of macrocytic anaemia in this age group (due to B12 deficiency). Subacute dementia and the subacute degeneration of the spinal cord (affecting the dorsal columns and peripheral nerves to present with the bilateral loss of sensation) with the classic triad of the brisk knee, absent ankle andextensor plantar reflexes can be explained by the B12 deficiency. This deficiency is a result of a chronic lack of intrinsic factors to metabolise dietary B12 to its active form. The lack of B12 has led to the axonal degeneration pattern seen with the reduced amplitude in the nerve conduction studies.

Hypothyroidism is incorrect. Untreated hypothyroidism would cause macrocytic anaemia and axonal neuropathy, however, would not explain the associated cord signs and symptoms described in this case.

Lead poisoning is incorrect, although it is another possible cause for an axonal pattern of nerve degeneration. However, in this case, the lead poisoning would not account for subacute dementia or cord signs.

Multiple sclerosis is incorrect as this does not usually result in cognitive impairment and would usually affect the corticospinal tracts also. Furthermore, it would not cause the axonal pattern in nerve conduction studies. The B12 deficiency is more likely in this case given the signs and symptoms.

Tabes dorsalis is incorrect. This is a result of tertiary syphilis and would explain the neurological signs seen here as it also affects the dorsal columns predominantly, however it would not explain the cause of the macrocytic anaemia.

Question 127 of 189

A 26-year-old woman presents with recurrent slurring of her speech that worsens as she continues to talk. She has double vision that worsens while watching TV and subsequently improves with rest, as well as intermittent weakness of the limbs.

On examination, she has visible ptosis - she explains this also improves with rest. Proximal muscle weakness is evident, with no wasting.

As well as the suspected neurological diagnosis, she demonstrates pyrexia and concurrent infection is suspected. The source is investigated.

Which antibiotic should be avoided, given the likely underlying neurological diagnosis?

A. Co-amoxiclav
B. Gentamicin
C. Meropenem
D. Nitrofurantoin
E. Vancomycin

B. Gentamicin

Aminoglycoside antibiotics are contraindicated in patients with myasthenia gravis due to the risk of fatal myasthenic crisis and respiratory failure

The history described above is highly suggestive of myasthenia gravis, given the history and signs found on examination. In a patient with this condition, a number of drugs may cause significant exacerbations and increase the risk of myasthenic crises. A number of antibiotics are contraindicated, including aminoglycosides such as gentamicin. Other classes generally avoided include macrolides, quinolones and tetracyclines.

Co-amoxiclav is generally considered safe to use in myasthenia gravis, as are other penicillinbased antibiotics.Meropenem is a beta-lactam antibiotic used in many situations where broad-spectrum treatment is needed. It is also considered safe to use in myasthenia gravis.

Nitrofurantoin is commonly used for the treatment of urinary tract infections. It is considered safe in patients with myasthenia gravis.

Vancomycin is a glycopeptide antibiotic, often used for complex or resistant infections. It is thought to be safe to use in myasthenia gravis.

Question 128 of 189

A 27-year-old man attends ambulatory care with a one day history of facial droop. He is a mountain bike instructor and is extremely anxious that this may be related to a fall last month. He hasn't been riding for the last month because he has felt unwell with aches and pains and some odd bruising up his leg.

On examination he has a bilateral VII nerve weakness but no other cranial nerve lesions detected. The arms and legs show no focal neurology.

Skin is intact with no skin lesions seen. There is no evidence of any joint effusions and all joints appear to have a full range of motion

Investigations:

Haemoglobin	127 g/L (130-180)
White cell count	10.0 Ã— 109/L (4.0-11.0)

Chest X-ray	Normal

What is the most likely diagnosis?

A. Sarcoidosis
B. Lyme disease
C. Syphilis
D. Bells palsy
E. Subdural haemorrhage

B. Lyme disease

This patient has bilateral seventh nerve palsy. The most likely diagnosis is Lyme disease. Lyme disease is caused by Borrelia burgdorferi bacteria that affects human from ticks that also feed on rodents.

Infected tick bites are common especially in the New Forest area of the UK. 70-80% of persons infected develop a rash (erythema migrans - target rash). Weeks after the initial bite thespirochetes spread via the bloodstream to joints, heart and nervous system.

Syphilis is on the rise but the more likely cause of the bilateral seventh nerve palsy is Lyme disease especially given the rash.

Subdural haemorrhage can occur after trauma - such as bike fall. Symptoms are likely to occur within days rather than having a first presentation one month after the accident.

Question 129 of 189

A 25-year-old lady attends her first neurologist appointment after moving areas. She has been referred by her GP with possible migraines for which she was receiving acetazolamide.

She attends reporting that the headaches are severe. They are typically up to 10/10 and are worse at the start of the day and improves as her day progresses. She finds sitting in a chair helpful and can sometimes vomit with them. Coughing and chewing hard food makes the headaches worse.

On examination, she has normal eye movements. Her optic discs appear a little blurred and there are no visual field defects. She refuses to lie flat and is non tender over her temporal scalp. She has normal observations and is afebrile. Her most recent tests are transferred to you

Na	132 mmol/l
K	3.1 mmol/l
Urea	4.2 mmol/l
Creatinine	76 µmol/l
HCO3	18mmol/l
MRI head	No mass lesion seen, increased subarachnoid space around the optic nerves

What is the most appropriate management?

A. Endovascular clipping
B. Lumbar puncture
C. Nifedipine
D. Sumatriptan
E. Refer to neurosurgery

B. Lumbar puncture

Idiopathic intracranial hypertension -> lumbar puncture is 2nd line treatment

The key to headaches is the history. This is not sudden and so is not a subarachnoid haemorrhage and nifedipine and endovascular clipping would therefore be inappropriate. Likewise, it does not sound entirely migrainous and sumatriptan would probably not help. There are no features that warrant a referral to neurosurgery. The enlarged subarachnoid space and postural headache suggest this is idiopathic intracranial hypertension. The first line is medicines like carbonic anhydrase inhibitors and if these fail then therapeutic lumbar puncture should be provided.

Question 130 of 189

A 50-year-old man is seen in the Emergency Department with fevers. He has been feeling generally unwell for the last 3 days and has just returned from a 2 week stay with his sister who lives on the other side of the U.K. He has had a mild non-productive cough and shortness of breath. He has had no vomitting, diarrhoea, dysuria or abdominal pain.

He has a past medical history of epilepsy, schizophrenia, hypertension and diet-controlled type 2 diabetes.

On examination he has a temperature of 38.3 ºC and is saturating at 94% on room air. His heart rate is 111 beats per minute and blood pressure is 118/75 mmHg. He looks clammy and pale but systems examination is otherwise unremarkable.

His chest x-ray shows clear lung fields. His blood tests are as follows:

Hb	140 g/l	Na	141 mmol/l
Platelets	443 * 10 /l	K	3.7 mmol/l
WBC	1.5 * 10 /l	Urea	7 mmol/l
Neuts	0.6 * 10 /l	Creatinine	98 µmol/l
Lymphs	0.5 * 10 /l	CRP	170 mg/l

He is started on broad spectrum antibiotics and fluids.

Which drug is most likely to have precipitated this condition?

A. Clobazam
B. Clozapine
C. Phenytoin
D. Sodium valproate
E. Venlafaxine

B. Clozapine

Agranulocytosis/neutropenia is a life-threatening side effect of clozapine - monitor FBC

This gentleman has neutropenic sepsis. The antipsychotic clozapine is well documented to cause agranulocytosis, neutropenia and lymphopenia. Patients on clozapine must be registered with the monitoring service specific to their brand of the drug and have regular drug levels and full blood counts. This gentleman has been travelling and has likely missed his monitoring appointment.

Clobazam side effects include drowsiness and confusion.

Phenytoin can cause leucopenia but this is unlikely in the absence of its other extensive side effect profile which includes drowsiness, tremor, gum hypertrophy, acne and hirsutism.

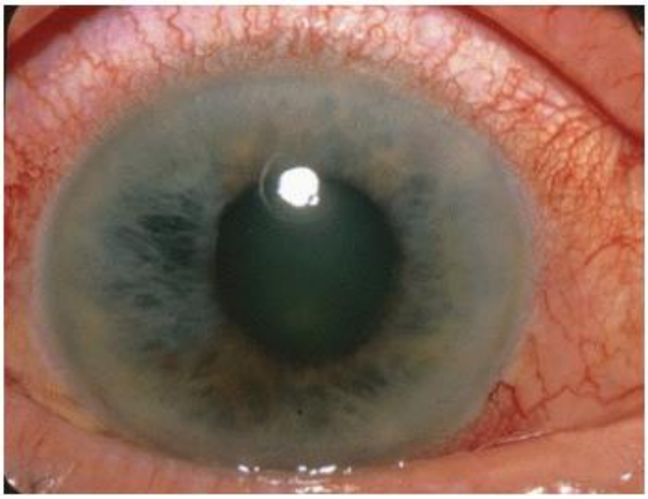
Sodium valproate also has a broad side effect profile which includes nausea, diarrhoea, weight gain, extra-pyramidal symptoms and hair loss. Again leucopenia is a documented side effect but rare.

Venlafaxine is associated with changes to serum cholesterol, hypertension, micturition disturbance and vasodilatation.

Question 131 of 189

A 50-year-old man presents to the emergency department with a painful right eye. The pain started approximately 4 hours ago whilst watching a movie. He also reports that his vision is blurry in the same eye. His past medical history includes type 2 diabetes and rheumatoid arthritis.

His eye is examined, as shown below:



What is the most appropriate management for this patient?

A. Reassurance and discharge
B. Topical aciclovir
C. Topical chloramphenicol
D. Topical corticosteroids
E. Topical timolol

E. Topical timolol

Topical timolol is correct. This patient's right eye is erythematous with a semi-dilated pupil. Combined with the clinical history and examination findings of reduced visual acuity, the patient has a likely diagnosis of acute angle-closure glaucoma. Hypermetropia (long-sightedness) is also a risk factor for acute angle-closure glaucoma. The management consists of reducing the intraocular pressure with a combination of medications to reduce aqueous humour production and increase uveoscleral outflow. Timolol is a beta-blocker eye drop that decreases aqueous humourproduction. Of note, acute angle-closure glaucoma is an ophthalmological emergency and requires urgent referral to ophthalmology. A combination of drugs is required in addition to timolol including, a muscarinic agonist eye drop (e.g. pilocarpine) and intravenous acetazolamide.

Reassurance and discharge is incorrect. Acute angle-closure glaucoma is an ophthalmological emergency and discharging home without intervention can result in blindness in the affected eye.

Topical aciclovir is incorrect. This is used in the management of viral ocular infections (e.g. herpes simplex virus). This usually presents with eye pain, watering and irritation. The eye may be erythematous and there are occasionally ulcers or vesicles seen along the eyelid margins. Examination of the eye with blue light after fluorescein administration may reveal the presence of a dendritic ulcer.

Topical chloramphenicol is incorrect. This is used in the management of bacterial conjunctivitis and keratitis. Keratitis is a potential differential diagnosis for this patient given the history of contact lens use. Symptoms of keratitis include erythema, photophobia, pain and lacrimation that are similar to this patient. However, it does not cause pupil dilatation making acute angle-closure glaucoma more likely.

Topical corticosteroids is incorrect. This is used in the management of anterior uveitis that presents as ocular pain, erythema and reduced visual acuity. Anterior uveitis is often associated with inflammatory conditions (e.g. ankylosing spondylitis). However, a change in pupil size is not a feature of anterior uveitis making it less likely to be the underlying diagnosis.

Question 132 of 189

A 80 year old woman is referred to neurology clinic after experiencing increasing diplopia, typically worsening during the course of the day. The patient reported no speech or swallowing problems and no limb weakness.

Past medical history included chronic obstructive pulmonary disease and ischaemic heart disease. The patient was able to mobilise around her flat with a frame but was required to use a wheelchair outside the home due to exertional breathlessness. Regular medications were inhaled salbutamol and tiotropium, aspirin, simvastatin, bisoprolol and ramipril. The patient lived with her husband and had once daily carers to assist with activities of daily living.

Examination in clinic was significant for the development of ptosis on prolonged upwards gaze. There was no significant weakness of facial muscles, palate or tongue. There was no evidence of fatiguable weakness in the arms or legs.

A summary of the patients investigations is given below.

Serum acetylcholine receptor antibodies	negative
Serum muscle specific tyrosine kinase	positive

Neurophysiology: no evidence of repetitive nerve stimulation

CT thorax: retro-sternal soft-tissue density mass equal in attenuation to muscle; mass demonstrates heterogeneous enhancement following contrast injection

What is the appropriate management of the retro-sternal mass?

A. Proceed to thymectomy
B. Proceed to thymectomy if patient does not respond to first line treatment
C. Surgical biopsy
D. No action required
E. Trans-bronchial biopsy

D. No action required

Thymectomy is a well recognised treatment for myasthenia gravis and should also be consideredin non-thymomatous generalised myasthenia in patients with antibodies to acetylcholine receptor who are aged under 50. Biopsy is not generally required prior to surgery.

However, thymectomy is not generally carried out in myasthenia gravis when patients have antibodies to MUSK, late onset disease or purely ocular disease as in this case. The patient's poor performance status would also argue against surgical intervention.

Repetitive nerve stimulation is a specific test for myasthenia gravis but has a relatively low sensitivity, especially in ocular only disease.

Question 133 of 189

A 24-year-old man presents with lower limb weakness. His past medical history is largely unremarkable, however, he does admit to intravenous drug use with heroin. He last injected heroin three weeks ago.

On examination, you note weakness of ankle plantar flexion (MRC grade 3), ankle dorsiflexion (MRC grade 3), knee flexion (MRC grade 4) and knee extension (MRC grade 4). The ankle jerk and knee jerk are only present on reinforcement. There is a loss of sensation to the mid-calves. A neurological examination was otherwise unremarkable.

What treatment is indicated?

A. Botulism antitoxin
B. Intravenous immunoglobulin (IVIG)
C. Plasma exchange
D. Steroids
E. Tetanus immunoglobulin

B. Intravenous immunoglobulin (IVIG)

IVIG is generally used first-line for Guillain-Barre syndrome

Intravenous immunoglobulin (IVIG) is correct. The most likely diagnosis is Guillain-Barre syndrome. IVIG is generally used as first-line management for Guillain-Barre syndrome.

Botulism antitoxin is incorrect. Botulism is characterised by a descending, flaccid paralysis, in contrast to Guillain-Barre syndrome which presents with an ascending paralysis. It is important to note that the patient is an intravenous drug user which is a risk factor for botulism.

Plasma exchange is incorrect. Although plasma exchange is just as effective as IVIG, it has more risks associated with it and is much more labour-intensive. For these reasons, IVIG is usually used first-line.Steroids is incorrect. Several trials have shown no benefit of steroids or immunosuppressants in Guillain-Barre syndrome.

Tetanus immunoglobulin is incorrect. Tetanus is a life-threatening disease characterised by a descending spastic paralysis. Although the causative bacteria Clostridium tetani has been found in contaminated heroin, the clinical features of an ascending paralysis are more suggestive of Guillain-Barre syndrome.

Question 134 of 189

A 23-year-old university student presented with a year long history of occipital headache. This was worse on coughing, sneezing and straining and partially relieved by lying flat. On one occasion the headache had been associated with vomiting. She had previously consulted her family doctor who was treating her for migraine. Over the past two months she had noticed pain in her both arms and felt unsteady on her feet.

On examination touching her arms caused pain and there was reduced appreciation of pinprick and temperature sensation throughout both arms. Tone, power and reflexes in the upper limbs were normal. On testing upper limb co-ordination there was some past-pointing and a very mild intention tremor. On inspection of the feet there was pes cavus and Rombergs test was positive. Again tone, power and reflexes were normal in the lower limbs and plantars were downgoing. Proprioception was impaired with absent joint position sense until the level of the knee. Vibration sensation was impaired in both feet.

Given the clinical findings outlined above which of the following is the most likely diagnosis?

A. Syringomyelia
B. Friedrichs ataxia
C. Brown-Sequard syndrome
D. Charcot-Marie-
E. Tooth disease Multiple sclerosis

A. Syringomyelia

Syringomyelia results from the formation of a fluid filled cavitation (called a syrinx) within the central canal of the spinal cord. The most common cause of a syrinx is obstruction to the flow of cerebrospinal fluid (CSF) by an Arnold Chiari malformation. An Arnold Chiari malformation is a developmental abnormality in which the cerebellar tonsils project through the foramen magnum and sit in a position which is normally occupied by the cervical spinal cord.

In this case the patient has features suggestive of an underlying Arnold Chiari malformation: occipital headache exacerbated by valsalva manoeuvre (coughing, sneezing and straining), pes cavus and cerebellar signs (past pointing and intention tremor in the upper limbs).

She also has features of an associated syringomyelia. The spinothalamic tracts are involved early inthe process due to their decussation within the cord. This means that there is loss of pain and temperature sensation corresponding to the level of the syrinx. This often occurs in a shawl-like distribution. Hyperalgesia and allodynia are also seen in the upper limbs in some cases.

As the syrinx enlarges the dorsal columns are affected resulting in loss of proprioception and vibration sensation in the lower limbs. This explains the positive Rombergs test which indicates that there is a proprioceptive problem.

If the syrinx expands further the anterior horn cells can be affected resulting in wasting and weakness in the hands.

Friedrichs ataxia is an autosomal recessive inherited condition in which there is progressive ataxia, dysarthria, loss of proprioceptive and vibration sensation and weakness.

Brown-Sequard syndrome results from a lateral hemisection of the spinal cord. It presents with a loss of pain and temperature sensation on the side of the body contralateral to the lesion and a hemiplegia and loss of vibration sensation and proprioception on the ipsilateral side to the lesion.

Charcot-Marie-Tooth (also known as Hereditary Sensory and Motor neuropathy) encompasses a group of inherited conditions which present in mid-adult life with peripheral neuropathy. Patients commonly have pes cavus and often present with foot drop.

Multiple sclerosis (MS) can present in a variety of ways with cerebellar features, optic neuritis, paraesthesiae and tranverse myelitis all being reported. Although in theory many of the clinical features in this question could be explained by an underlying diagnosis of MS, the clinical presentation here is typical of syringomyelia secondary to an underlying Arnold Chiari malformation.

Question 135 of 189

A 34-year-old man presents with insidious onset right hand weakness over the last week. He is right hand dominant and struggling to perform basic tasks such as opening a door with a key.

On examination he has marked weakness of thumb flexion at the interphalangeal joint and weakness of flexion of the index and middle finger. There is no detectable sensory deficit.

What is the likely diagnosis?

A. C8 nerve root radiculopathy
B. Motor neurone disease
C. Median nerve palsy
D. Carpal tunnel syndrome
E. Anterior interosseous syndrome

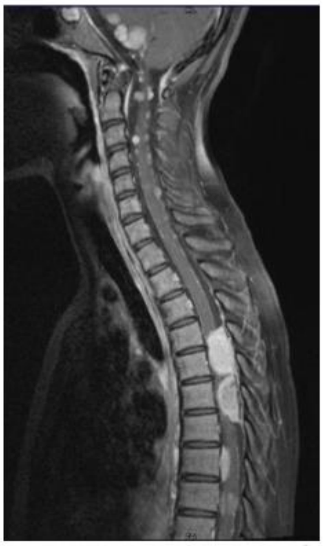
Question 136 of 189

A 24-year-old woman is referred urgently to neurology for lower limb weakness. This has been getting gradually worse over the past 4 weeks and she is now experiencing problems walking.

She also complains of bilateral tinnitus which has been present for the past 3 months. This is associated with episodes of vertigo.

On examination, both lower limbs have reduced power (3/5), with increased tone and hyperreflexia. The patient has one 1cm tan lesion on her torso but no other skin lesions are noted.

An MRI of her spine is requested:



What is the most likely diagnosis?

A. Multiple sclerosis
B. Neurofibromatosis
C. Von Hippel-Lindau disease
D. Tuberous sclerosis
E. Ataxia telangiectasia

B. Neurofibromatosis

This patient had neurofibromatosis type 2 (NF2). The MRI shows multiple tumours of the spinal cord in the context of the syndrome of multiple intracranial schwannomas, mengiomas and ependymomas.

Patients with NF2, in contrast to type 1, often have few skin changes. This patient had tinnitus due to the presence of bilateral acoustic neuromas.

The diagnostic criteria for NF2 are:

- Bilateral vestibular schwannomas
- A first degree relative with NF2 AND
    - Unilateral vestibular schwannoma OR
    - Any two of: meningioma, schwannoma, glioma, neurofibroma, posterior subcapsular lenticular opacities

Unilateral vestibular schwannoma AND

- Any two of: meningioma, schwannoma, glioma, neurofibroma, posterior subcapsular lenticular opacities

Multiple meningiomas AND

- Unilateral vestibular schwannoma OR
- Any two of: schwannoma, glioma, neurofibroma, cataract

Question 137 of 189

A 65-year-old male presents to the neurology outpatient department with a history of recurrent bouts of unsteadiness and vomiting over the last 10 years, with partial resolution. He has also had episodes of visual problems, which he describes as the sudden loss of vision in the right eye, with an almost complete recovery of vision over the course of the next few weeks. He has a history of type 2 diabetes and is hypertensive and is generally non-compliant with his treatment. He is also a smoker with a 50 pack year history.

His medication includes glimepiride 2mg daily and metformin 500mg TDS. He also takes telmisartan 40mg daily.

On examination, he has nystagmus in the right eye with the fast component towards the right. His gait is ataxic and he has evidence of spasticity in both lower limbs with exaggerated reflexes and bilateral ankle clonus. Fundoscopic examination revealed a pale optic disc.

MRI brain shows diffuse lesions in multiple sites. The report queried demyelinating plaques vs multiple infarcts.

Which of the following would be the most appropriate next investigation?

A. MRI spinal cord
B. CSF examination for oligoclonal bands
C. MR angiography of the verterbrobasilar system
D. Titres of anti-Hu antibodies
E. Visual evoked potentials

A. MRI spinal cord

The diagnosis, in this case, is that of multiple sclerosis and the question aims to assess the candidate's ability to plan further investigations if the MRI of the brain is inconclusive.

An MRI of the brain is a sensitive imaging modality for the detection of plaques in patients with MS, but it lacks specificity. Over the age of 50, small ischaemic lesions may be difficult to distinguish from demyelination. The presence of spinal cord lesions, however, is quite specific for inflammatory disorders such as MS rather than ischaemic lesions. Thus cord imaging is useful when there is diagnostic difficulty.

Oligoclonal bands are found in the CSF in >90% of cases of MS but are not specific for MS. They are also found in

- Lyme disease
- Systemic lupus erythematosus
- Neurosarcoidosis
- Subacute sclerosing panencephalitis
- Subarachnoid hemorrhage
- Syphilis
- Primary central nervous system lymphoma
- Sjogren's syndrome
- Guillain-Barre syndrome
- Meningeal carcinomatosis
- Neuromyelitis optica

Question 138 of 189

A 55-year-old cachectic male is brought into hospital after having been found on the floor by his sister, who visits him once a week. She reports a long history of depression and his appetite to have been poor for a number of years. He denies any loss of consciousness episodes, palpitations, chest pain, dysphagia or presyncopal symptoms. When asked how he ended up on the floor, he vaguely reports to have slowly fallen from being generally weak and denies head injury. He does report a recent sore throat and dry cough. He is alert and orientated. On examination, he scores 3 and 4 out of 5 power in all movements. Lower limb reflexes could not be elicited with a tendon hammer but bilateral upgoing plantars are noted.

Which treatment might reverse the underlying condition most rapidly?

A. IV methylprednisolone
B. IV immunoglobulin infusion
C. Plasmapheresis
D. IV thrombolysis
E. IM vitamin B12 + IV pabrinex

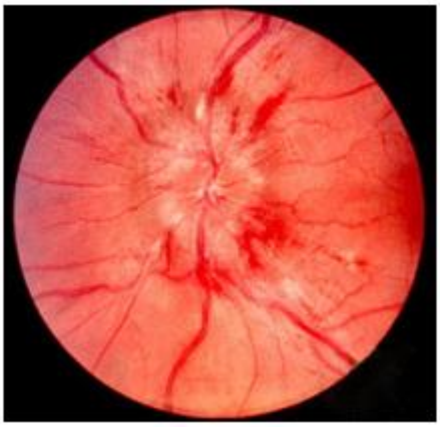
E. IM vitamin B12 + IV pabrinex

The patient presents with upgoing plantars and absent ankle jerks. This should alert all MRCP candidates to 5 classic diagnoses: subacute combined degeneration of the cord (SCDC), motor neurone disease, Friedrich's ataxia, tabes dorsalis (syphilis) and dual peripheral with central pathology. In the context of cachexia and poor oral intake should alert a vitamin B12 deficiency, leading to a combination of peripheral and dorsal column degeneration, known as subacute combined degeneration of the cord. Vitamin B12 can in some cases reverse the neurology very rapidly.

Question 139 of 189

A 44-year-old woman presents to the emergency department with a 2-week history of worsening headaches. She also complains of occasional bouts of dizziness and double vision. Her past medical history includes borderline type 2 diabetes and her regular medications include metformin and the combined contraceptive pill.

On examination, her observations are within normal limits. Her BMI is 32 kg/m². There is no focal neurology to find on examination. Fundoscopy is carried out (as shown below):



Given the finding from this patient's fundoscopy, what is the next step in the management of this patient?

A. CT head
B. Reassurance and routine ophthalmology follow up in 2 weeks
C. Topical corticosteroids
D. Urgent lumbar puncture
E. Urgent referral to ophthalmology

A. CT head

This patient has papilloedema on fundoscopic examination, as seen with the enlarged optic disc with blurred margins and venous congestion. Papilloedema arises secondary to raised intracranial pressure (e.g. inflammation, infection, malignancy or idiopathic intracranial hypertension). Given this finding on examination, in addition to the symptoms of headache, dizziness and blurredvision, it is most important to complete a CT head to rule out any space-occupying lesion that may be causing raised intracranial pressure.

Reassurance and routine ophthalmology follow-up in 2 weeks is incorrect. It is not appropriate to discharge with follow-up at this stage as a combination of papilloedema and headaches can be suggestive of serious underlying pathology such as an intracranial tumour. Therefore, a CT head must be requested.

Topical corticosteroids are incorrect. Topical corticosteroids are used for the treatment of anterior uveitis, a condition that causes a painful red eye with photophobia and blurred vision. Patients also require an urgent review by ophthalmology. However, topical corticosteroids have no role in the treatment of papilloedema.

Urgent lumbar puncture is incorrect. A lumbar puncture is a reasonable investigation to request given the possibility of this patient having idiopathic intracranial hypertension (given the presence of an obese woman taking oral contraceptives) or an infectious cause of her headache. However, a lumbar puncture must not be completed until after a CT head is performed to rule out a spaceoccupying lesion that may be responsible for raised intracranial pressure.

Urgent referral to ophthalmology is incorrect. Although papilloedema is a worrying feature to find on examination, it does not require an urgent referral to ophthalmology. Rather, it requires neuroimaging to rule out intracranial pathology.

Question 140 of 189

A 24-year-old woman is admitted with a fever and headache. She reports this began acutely and worsened over the last 24 hours, now being associated with difficulty looking at lights.

She is febrile and photophobic with neck stiffness present. Her other observations are normal. GCS is 15 and no seizure activity is reported. There are no focal neurological deficits and no rash is observed.

She is commenced on IV ceftriaxone and dexamethasone for presumed bacterial meningitis. A lumbar puncture is performed which yields the following results.

Opening pressure	14cm H2O (6-15)
WBC	127 cells/mm³ (<5)
WBC Differential	85% lymphocytes, 15 % polymorphs
CSF Protein	0.7g/L (0.2-0.4)
CSF Glucose	4.9mmol/L (>2/3 serum)
Serum glucose	5.6mmol/L (3.3 - 5.8)
Culture	No organisms isolated
Bacterial PCR	Negative for *Neisseria meningitidis*, *Streptococcus pneumoniae* and *Haemophilus influenzae*
Viral PCR	Positive for Herpes simplex virus type 2

What is the most appropriate course of action?

A. Add IV aciclovir
B. Continue dexamethasone for 4 days and ceftriaxone for 10 days
C. Stop ceftriaxone and dexamethasone, start IV aciclovir for 14 days then repeat LP
D. Stop ceftriaxone and dexamethasone, start oral valaciclovir
E. Stop ceftriaxone and dexamethasone, supportive treatment only

E. Stop ceftriaxone and dexamethasone, supportive treatment only

Herpes simplex meningitis does not require treatment. This is in contrast to herpes simplex encephalitis which requires 14-21 days of intravenous aciclovirImportant for me Less important

The correct answer is stop ceftriaxone and dexamethasone, supportive treatment only. This patient's clinical presentation is consistent with a meningitic process which presents with fever, headache, neck stiffness, and photophobia. Viral meningitis, in stark contrast to viral encephalitis or bacterial meningitis, generally does not require treatment. Antibiotics should be discontinued and with supportive treatment, the condition generally resolves in 2-5 days. Enteroviruses are the most common cause, but HSV type 2 is recognised and associated with a condition now called recurrent benign lymphocytic meningitis.

Add IV aciclovir is incorrect. IV aciclovir is the treatment of choice for viral encephalitis caused by herpes simplex virus type 1. This condition involves inflammation of the brain parenchyma rather than the meninges. It results in an altered conscious level, agitation, seizures, and/or focal neurological deficits. None of these are described in this case. There would also be no rationale for continuing ceftriaxone and dexamethasone with negative cultures and a CSF examination consistent with a viral infection.

Continue dexamethasone for 4 days and ceftriaxone for 10 days is incorrect. This is the treatment recommendation for pneumococcal meningitis. This more commonly affects older adults, with bacterial meningitis in younger adults more commonly meningococcal. CSF in pneumococcal meningitis generally shows a predominance of polymorphs with raised protein and low glucose. Cultures may be negative where antibiotics have been given before lumbar puncture, but the negative bacterial PCR is reassuring.

Stop ceftriaxone and dexamethasone, start IV aciclovir for 14 days then repeat LP is incorrect. This would be the treatment of choice for HSV encephalitis, usually caused by HSV type 1. As discussed above, this presents with an altered conscious level, agitation, seizures, and/or focal neurological deficit. If the repeat LP at 14 days remains PCR positive, treatment is extended for a further 7 days.

Stop ceftriaxone and dexamethasone, start oral valaciclovir is incorrect. Valaciclovir is an alternative agent to oral aciclovir which can help with compliance, as it is administered twice daily rather than 5 times daily. However, its use is in oral or genital HSV infection rather than CNS infection. Also, as discussed above, there is no need for treatment in the case described.

Question 141 of 189

A 65-year-old woman presents to her general practitioner with a two-month history of a severe, 'all-over' throbbing headache, which varies in intensity over the course of the day. She has a past medical history of obesity and a traumatic fracture of the knee three months previously. She takes codeine regularly. She smokes 10 cigarettes per day.

The examination is unremarkable and the fundoscopy is normal. Her body mass index is 42 kg/m². A CT of her brain shows no abnormality.

Blood tests: CRP 3mg/L (<5)

Given the likely diagnosis, what is the appropriate management?

A. Acetazolamide
B. Prednisolone
C. Therapeutic lumbar puncture
D. Withdraw codeine gradually
E. Withdraw codeine now

D. Withdraw codeine gradually

Medication overuse headache

simple analgesia + triptans: stop abruptly

opioid analgesia: withdraw gradually

Withdraw codeine gradually is the correct answer. The patient presents with a new daily headache. As she has had a recent fracture, it is likely that codeine treatment was started at the same time. This new headache occurring after commencing opioid analgesia is likely reflective of a medication overuse headache. It varies over the course of the day as the dose of codeine wears off before the next dose is taken. Management of a codeine-associated medication overuse headache is to withdraw the medication gradually.Acetazolamide is incorrect. She is overweight and this would put her at risk of developing idiopathic intracranial hypertension (IIH). However, fundoscopy has excluded papilloedema and so this makes this diagnosis unlikely. Furthermore, headaches associated with raised intracranial pressure are typically specifically worse in the morning rather than varying over the course of the day.

Therapeutic lumbar puncture is incorrect. This is an option for the treatment of IIH if confirmed. However, that diagnosis is unlikely in the setting of a normal fundoscopic examination.

Prednisolone is incorrect. Any new headache in someone > 50 should prompt consideration of giant cell arteritis (GCA). However, the headache of GCA is typically unilateral and associated with scalp tenderness. Additionally, it is extremely unlikely to have this condition and a normal CRP (now felt to be a more accurate marker of systemic inflammation than ESR in this condition).

Withdraw codeine now is incorrect. Withdrawal of opioids in medication overuse headache should be gradual rather than abrupt to avoid withdrawal and a worsening of the clinical situation.

Question 142 of 189

A 19-year-old lady comes to her clinic appointment and is distraught. She has had severe headaches for the past year and this has led to her dropping out of her university degree. These often stop her doing anything else. She has vomits most days of the week when the pain is at its worse and her vision feels blurry. She has been managed by her GP who has tried sumatriptan, NSAIDs, paracetamol and relaxation techniques all without much benefit. She is currently taking sertraline for depression and she does not smoke.

Her headache are a constant severe frontal headache with radiation around the eyes. She can vomit at times and this is commonly before with her breakfast. She has rarely found relief from these but does note that her symptoms are better in the evenings.

On examination, she has normal visual fields. All her limbs have equal strength and normal tone. There is no sensory loss. Her fundi show no bleeds or exudate but the disc is blurred. She has a raised body mass index of 26kg/m² and afebrile. She has no sinus tenderness and not inflammation of the upper respiratory tract mucosa can be seen.

Na	138 mmol/l
K	4.3 mmol/l
Urea	5.1 mmol/l

Creatinine	87 µmol/l
CT head	no intracranial haemorrhage, mass effect or lesions seen
MRI head	flattening of the sclera of the eye, partially empty sella turcica, enlarged subarachnoid space around the oculomotor and optic nerves

What is the likely diagnosis?

A. Ependymoma
B. Idiopathic intracranial hypertension
C. Venous sinus thrombosis
D. Myopia
E. Chronic sinusitis

Postural headache but normal imaging -> idiopathic intracranial hypertension

This is a chronic postural headache suggested by its improvement as the day progresses. Myopia and chronic sinusitis would not cause a postural headache. Ependymoma and a venous sinus thrombosis would both be seen on imaging. Therefore, by exclusion, this headache is idiopathic intracranial hypertension.

Question 143 of 189

A 23-year-old man presents to you in General Practice with symptoms of low mood, anhedonia, irritability and disturbed sleep. He denies feelings of self-harm or suicidal ideation.

His PHQ-9 score is 10 indicating moderate depression. You refer him for cognitive behavioural therapy (CBT) and decide to commence him on fluoxetine.

What is the most appropriate next step in follow-up for this patient?

A. Follow up review in the community within 2 weeks
B. Follow up review in the community within 7 days
C. Follow up review in the community within 72 hours
D. No routine follow up required - patient to represent if needed
E. Referral to mental health services

B. Follow up review in the community within 7 days

Patients ≤ 25 years who have been started on an SSRI should be reviewed after 1 week

This patient has moderate depression as indicated by their PHQ-9 score. Patients under the age of 25 who are started on an SSRI should be followed up within 1 week due to the increased risk of suicide within this age group and therefore option 2 - follow-up review in the community within 7 days is the correct answer. Patients over the age of 25 who are started on antidepressant therapy should be followed up in the community within 2 weeks.

Follow-up in the community within 2 weeks and follow up in the community within 72 hours are incorrect as they are not the recommended time scales for follow-up.

No routine follow-up required - patient to represent if needed is incorrect as all patients who are initiated on antidepressant therapy should have planned follow-up due to the risk of worsening symptoms when initiating treatment.Referral to mental health services is incorrect as there is no indication for referral to mental health services at this stage.

Question 144 of 189

A 60 yr old woman went swimming with her family, and after finishing her swim and having a shower, her daughter noted that she became confused suddenly, repetitively asking if they had gone for a swim today. She could not remember events that occurred in the past 24 hours, and when told the answers to her questions, would ask the same question 5 minutes later. There was no change in her personality, no change in her speech, nor any muscle weakness. She is able to recall her address, the names of her daughters and husband, and her date of birth. Her daughter said her mother did not suffer any trauma during the swim, and did not lose consciousness anytime throughout the day. The patient's past medical history includes hypertension and depression, and she takes ramipril 2.5mg once daily, and citalopram 20mg once daily.

On examination the patient was alert, but constantly asked where she was and why was she in hospital. She was afebrile, heart rate 80 bpm, blood pressure 138/68 mmHg, respiratory rate of 18 breaths per minute, and oxygen saturation of 99% on air. Neurological examination was unremarkable, but her abbreviated mental test score was 6/10.

Her investigation results were as follow:

C Reactive protein	4 mg/l
Haemoglobin	14.8 g/dl
White cell count	5.6 x 10^9/L
Na+	142 mmol/l
K+	4.3 mmol/l
Urea	4.2 mmol/l
Creatinine	68 µmol/l
Corrected calcium	2.32 mmol/l
Plasma glucose	5.8 mmol/

Computer Tomography (CT) head scan No acute intracranial pathology. Over the next 12 hours, her memory improves and she is discharged from the observation ward.

What is the best advice for the patient with regards to driving in the future?

A. She can drive 4 weeks after the event if the cause has been identified and treated
B. DVLA need not be notified, no driving restrictions
C. She must be symptom free for 1 year before being eligible to drive
D. DVLA must be informed, and her licence will be revoked for 6 months
E. She must cease driving until there is satisfactory control of her symptoms

This lady has suffered an episode of transient global amnesia, a neurological condition characterised by acute short term memory loss, lasting no more than 24 hours. Patients often developed perseveration, with repetitive questioning as they are unable to keep short term memory, but are able to recall long term memories. The aetiology of this condition is unknown.

DVLA guidelines for transient global amnesia are that as long as epilepsy, any sequelae from head injury and other causes of altered awareness have been excluded, there are no restrictions on driving and the DVLA need not be informed.

Question 145 of 189

A 43-year-old right-handed female legal secretary has presented into your general medical clinic in an extremely distressed state. Over the past 2 weeks, she has been 'unable to write'. When questioned further, she reports that she 'wants to write but my hand just stops as soon as I pick up the pen.' She has no past medical history, lives with her husband and is a non-smoker and nondrinker. On examination, her neurological examination is unremarkable. You ask her to write. Her hand and fingers suddenly flex, resulting in illegible handwriting.

What is the most likely diagnosis?

A. Psychogenic functional neurology
B. Focal dystonia
C. Dominant parietal lobe space occupying lesion
D. Right median nerve palsy
E. Brachial plexopathy

B. Focal dystonia

The patient describes a history suggestive of writers cramp, a focal dystonia characterised by flexion, extension or rotation of the muscles of the hand. The underlying pathophysiology is unclear but is thought to relate to a change in the plasticity of cortical networks.

Question 146 of 189

A 68-year-old female presents via blue light ambulance to the emergency department with a first seizure, witnessed by her husband. He describes a sudden onset limb jerking lasting for around 6 minutes, associated with urinary incontinence and tongue biting, with a period of confusion and drowsiness immediately after. He is not aware of her having had any previous seizures, she has no other past medical history and does not take drugs on a medical history. He, however, does describe her to be 'not quite herself' over the past four weeks when she appears to have been extremely agitated and occasionally extremely paranoid. He had thought it to be linked to her not feeling very well, during a period when she had complained of a 'flu-like' headache, generalised muscle ache and a non-productive cough.

You examine her, finding significant gait and limb ataxia with no truncal ataxia. Her blood tests are unremarkable except for positive anti-NMDA antibodies. Her MRI scan demonstrates swelling in bilateral limbic cortices and other no other intracranial abnormalities. She has declined a lumbar puncture and is thought to retain capacity.

Which investigation would most likely provide the underlying diagnosis?

A. CT abdomen/pelvis with contrast
B. Lumbar puncture
C. Nerve conduction studies and EMG
D. MRI spine
E. Muscle biopsy

A. CT abdomen/pelvis with contrast

This middle-aged woman has presented with seizures on a background of personality change, agitation, paranoia and cerebellar ataxia. The diagnostic test is her positive anti-NMDA antibody, with MRI confirm limbic encephalitis as a result of a paraneoplastic syndrome. The underlying diagnosis is classically an ovarian teratoma, presenting antigens to the body's immune system similar to the brain limbic areas, driving an immune-related encephalitis. Treatment requires immunosuppression with intravenous steroids, immunoglobulin's or plasma exchange, in addition to removal of the underlying tumour by surgery.

Question 147 of 189

A 44-year-old male presents to your headache clinic with a 6-month history of left-sided daily headaches located in the frontal and retroorbital area. He denies any pain-free periods. The headaches are of moderate 5/10 severity with unpredictable exacerbations of severe pain going up to 9/10 severity. He describes to you what sounds to you like left-sided conjunctival injection and lacrimation often occurring alongside the headaches.

Which of the following is most likely to aid you in making a diagnosis?

A. a trial of high flow 100% oxygen
B. Lumbar puncture
C. a trial of indomethacin
D. CT brain
E. MRI brain

C. a trial of indomethacin

A headache with absolutely no pain-free periods otherwise described like this (i.e. like a trigeminal autonomic cephalalgia (TAC)) is hemicrania continua.

The trigeminal autonomic cephalalgias should be side-locked (i.e. one side only) and typically but not exclusively in the retroorbital/temporal region. They should include some autonomic parasympathetic feature such as eye-watering, red eye, running nose, a sensation of fullness in the ear, or miosis. These should be ipsilateral to the pain. They are a spectrum and most conveniently differentiated by how long the headache lasts for and how frequent the attacks happen per day. Some features are quite noticeable in TAC, e.g in a cluster headache (aka 'suicide headaches') people are usually very animated and pace about/hold their head in pain/want to smash their head against the wall (contrast this to migraine: which is not a TAC and usually involves sitting still in a dark room and not making any noise).

It is worth looking up the International Classification of Headache Disorders diagnostic criteria for TACs. Here are some important features:

- Cluster headaches: attacks have a frequency between one every other day and 8 per day and last 15-180 minutes.
- Paroxysmal hemicrania: attacks have a frequency above five per day and last 2-30 min. The condition responds absolutely to indomethacin
- Hemicrania continua: a constant form of paroxysmal hemicrania (no headache-free periods). Also responds absolutely to indomethacin.

- Short-lasting unilateral neuralgiform headache attacks: could get 20 attacks in a day, each last around 1600 seconds.

Notice the differences between how long each lasts and how many attacks there can be per day. The rest of the features are quite similar to each other between the headaches, so learn the general features of a TAC then memorise how long each should last and its frequency to help you differentiate them.

Returning to the question: hemicrania continua and paroxysmal hemicrania clearly are the two that completely resolve with indomethacin. The history, therefore, helps differentiate which is which. Practically speaking, you bring these patients in, on two separate days, for a trial injection of indomethacin or placebo (water) where patient (and nurse if possible) is blinded to which is which and we see which one of the two days was associated with resolution of headaches at a later date in clinic (give the patient a headache diary to take home with). Note the 100% oxygen trial would be used for cluster headaches, which tends to respond to this.

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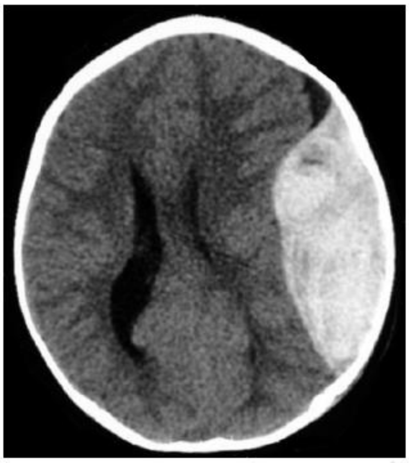
You are asked to review a 32-year-old man who was brought in by ambulance after an assault. A witness states that he lost consciousness before waking up fully. He has subsequently become more drowsy. His current GCS is 9/15 (E2 V3 M4).

Neurological examination is difficult due to GCS. Of note, pupils are symmetrical and there is an extensor right plantar.

A cross-section of his CT brain is shown below.



Which of the following parts of the history is associated with a poorer prognosis?

A. Extensor plantar response
B. Low GCS at presentation
C. Lucid period
D. Symmetrical pupils
E. Young age

B. Low GCS at presentation

The above history describes a reduced level of consciousness following an assault. The CT brain shows a biconvex hyperdensity in the left temporal region which is in keeping with an extradural haematoma. There is evidence of mass effect with midline shift and effacement of the left lateral ventricle which, combined with a reducing conscious level, is concerning for raised intracranial pressure (ICP).

A low GCS at presentation is correct. Low GCS is likely associated with raised ICP and/or traumatic brain injury and therefore carries a poorer prognosis.

Extensor plantar response eludes to an upper motor neuron lesion, in this case, an epidural haematoma, and would be an expected finding but does not denote a poorer prognosis.

Poorer prognosis is associated with patients that do not experience a lucid period. Asymmetrical pupils are associated with a poor prognosis and are a sign of raised ICP. Young age is a protective factor compared to old age which carries a worse prognosis.

Question 149 of 189

A 52-year-old woman presents to your clinic with a history of vertigo.

She describes short-lived episodes of true vertigo which often occur whilst lying down in bed, turning over or sitting up.

The episodes last between ten to twenty seconds and then quickly dissipate.

You perform the Dix-Hallpike test in the clinic which gives a positive result and you make a diagnosis of BPPV.

Whilst consenting the patient to perform an Epley manoeuvre to treat her BPPV the patient asks how successful this is at treating her condition.

In what percentage of cases is this successful?

A. 25%
B. 50%
C. 60%
D. 80%
E. 99%

D. 80%

The Epley manoeuvre is successful in treating around 80% of cases of BPPV

The Epley manoeuvre is successful in treating around 80% of cases of BPPV. Repeated treatments with the Epley manoeuvre can be helpful if it fails at the first attempt. Patients can also be shown how to do 'home Epley' manoeuvres to complete by themselves.

Question 150 of 189

A 76-year-old man was brought into the emergency department by his wife. She was concerned because she had noticed that he had been slurring his words at breakfast that morning at 07:00. She had also noticed he was suffering from left-sided weakness. These symptoms persisted and he was struggling to feed himself when they ate lunch at 13:30.

He had a past medical history of hypertension for which he takes amlodipine. He did not smoke and was independent in his activities of daily living.

On examination, he was dysarthric and had hemisensory loss of his left side. Power was 2/5 in his left leg and left arm. He had normal sensation and power on the right side of his body. His pulse was irregular.

Observations were as follows:

- oxygen saturations 98% on room air
- respiratory rate 14/min
- heart rate 67bpm
- blood pressure 135/78mmHg
- temperature 37.1ºC.

A CT angiogram was performed with perfusion imaging which revealed the presence of a thrombus in the right middle cerebral artery. There was a small area of infarct present and a large area of surrounding ischaemic penumbra.

What is the most appropriate management of this patient?

A. Load with aspirin and admit to the stroke ward
B. Thrombectomy
C. IV thrombolysis with alteplase
D. IV heparin infusion
E. Decompressive hemicraniectomy

B. Thrombectomy

For thrombectomy in acute ischaemic stroke, an extended target time of 6-24 hours may be considered if there is the potential to salvage brain tissue, as shown by imaging such as CT perfusion or diffusion-weighted MRI sequences showing limited infarct core volume

This patient has had an ischaemic stroke, likely as a result of inadequate anticoagulation in the context of atrial fibrillation. As the symptoms were first noticed during breakfast, this patient is outside the 4.5 hour time window in which thrombolysis can be attempted.

Despite this, his CT perfusion scan shows only a small area of infarct, surrounded by a large area of potentially salvageable ischaemic tissue (the penumbra). NICE guidelines suggest that thrombectomy should be offered as soon as possible to patients who have suffered an ischaemic stroke (as demonstrated by CTA) within 6 to 24 hours (including wake-up strokes) if there is the potential to salvage brain tissue (as shown on CT perfusion imaging or diffusion-weighted MRI).

Patients who have a pre-existing disability (e.g. frailty, poor functional baseline) often have poor outcomes even if thrombectomy is successful, so NICE recommends that thrombectomy is reserved for those with a pre-stroke Modified Rankin score of <3. This patient meets these inclusion criteria given his good baseline function.

Patients who are outside the time window or otherwise do not meet the criteria for thrombolysis/thrombectomy should be managed on a stroke ward with antiplatelet agents and control of risk factors (e.g. blood pressure control, anticoagulation for atrial fibrillation commenced 2 weeks after the stroke).

IV heparin infusions are not used to treat ischaemic strokes. Unfractionated heparin is used for rapid anticoagulation (in the treatment of thromboembolic disease for example) in patients who are at high risk of bleeding (the anticoagulant effect can be terminated rapidly by stopping the infusion).

A decompressive hemicraniectomy is reserved for malignant middle cerebral artery syndrome

Question 151 of 189

A 25-year-old man presents with a four-day history of anorexia, feverishness and vertigo. He has had intermittent difficulty balancing and staying upright when walking and has had episodes of mild vertigo lasting 10-20 minutes at a time. His hearing is unimpaired. On examination, he has some cervical lymphadenopathy. The examination is otherwise unremarkable.

What is the likely diagnosis?

A. Meniere's disease
B. Benign Paroxysmal Positional Vertigo
C. Otitis media
D. Vestibular neuronitis
E. Multiple sclerosis

D. Vestibular neuronitis

Vestibular neuritis stereotypical history: recovering from an upper respiratory tract infections presents with recurrent attacks of vertigo associated with nausea and vomiting. There is no hearing loss or tinnitus

Vestibular neuronitis may be preceded by viral symptoms. Vestibular neuronitis is associated with spontaneous onset vertigo and imbalance often associated with nausea and vomiting. Unlike labyrinthitis, it does not cause tinnitus or hearing loss.

Urgent referral should be considered with any of the following red flags: Neurological symptoms or signs, acute deafness, a new type or new onset headache, vertical nystagmus.

Question 152 of 189

A 55-year-old male presents with a 3-week history of speech slurring, dysphagia and droopiness of his eyelids. His only past medical history includes type 2 diabetes mellitus and hypertension, both of which are well controlled. On examination, you note bilateral ptosis with a full range of eye movements. When asked to count upwards from his speech begins to slur at 15. In addition, he spits out upper airway secretions three times during you examination but is able to swallow half a glass of water. Neurological

examination of the rest of his cranial nerves, upper and lower limbs are unremarkable. FVC is 85% of predicted.

What is the most appropriate treatment?

A. Intravenous immunoglobulins
B. Plasmapheresis
C. Oral pyridostigmine
D. Oral prednisolone
E. Oral azathioprine

C. Oral pyridostigmine

Pyridostigmine is a long-acting acetylcholinesterase inhibitor that reduces the breakdown of acetylcholine in the neuromuscular junction, temporarily improving symptoms of myasthenia gravis

The patient describes a clear history of bulbar and ocular myasthenia gravis. The key distinction is that this patient is not in crisis when respiratory function is compromised or complete dysphagia is noted. During a crisis, IVIg and plasmapheresis are indicated but not in a new diagnosis setting. The appropriate treatment is pyridostigmine, an acetylcholinesterase inhibitor. An immunomodulating agent is added only if the patient remains symptomatic with pyridostigmine. Steroids such as prednisolone or azathioprine, ciclosporin or mycophenolate mofetil are reasonable choices. Neither would immediately be required in a new diagnosis

Question 153 of 189

Which of the following patients fits a diagnosis of neurofibromatosis type 1 according to the National Institute of Health (NIH) criteria?

A. A 9-year old male with 2 lisch nodules and 6 cafe au lait macules larger than 5mm diameter
B. A 15-year old female with a dysplastic naevus and a 1st-degree relative with neurofibromatosis
C. A 22-year old male with a shagreen patch and 10 cafe au lait macules larger than 20mm diameter
D. A 25-year old female with axillary freckling and 10 cafe au lait macules larger than 10mm diameter
E. A 30-year old male with an acoustic neuroma an a cutaneous neurofibroma

A. A 9-year old male with 2 lisch nodules and 6 cafe au lait macules larger than 5mm diameter

The NIH consensus development group published some criteria to aid diagnosis of neurofibromatosis type 1 in 1988, as neurocutaneous disorders can be difficult to distinguish. It is important to note that these criteria focus on cafe au lait macule size in adults and children. A child with 6 or more cafe au lait macules larger than 0.5 cm would fit one of the criteria but they would need to be 1.5 cm or larger in an adult. Other criteria include axillary freckling, 2 or more cutaneous/subcutaneous neurofibromas or one plexiform neurofibroma, optic pathway glioma, bony dysplasia, 2 or more Lisch nodules or a 1st-degree relative with neurofibromatosis type 1. At least two of these criteria are required to reach clinical diagnosis.

Acoustic neuromas are associated with neurofibromatosis type 2.

Question 154 of 189

A 32-year-old lady develops a progressive loss of central vision in both eyes over two days. Her eyes are also painful to move. 24 hours later she develops additional paraesthesia in both arms with an associated weakness of both arms. On examination, she has central scotoma of both eyes. She also has a 3/5 global weakness of both arms with hyperreflexia and patchy sensory loss. In the following days, this progresses to paraesthesia and weakness in both lower limbs plus extensor plantar responses.

Raised serum levels of which of the following would be most helpful in establishing the suspected underlying diagnosis? helpful in establishing the suspected diagnosis?

A. Anti-Hu antibodies
B. Antinuclear antibodies
C. N-methyl-d-aspartate (NMDA) receptor antibodies
D. Anti-Aquaporin-4 antibodies
E. Voltage gated potassium channel antibodies

D. Anti-Aquaporin-4 antibodies

Neuromyelitis optica is associated with aquaporin 4 positive serum antibody

The diagnosis is neuromyelitis optica (NMO). It's features are best remembered by what they describe: the co-occurrence of optic neuritis and myelitis. Myelitis describes swelling of the spinal cord and in NMO it characteristically extends over three or more vertebral segments, and the limb features correspond to the level affected. The optic neuritis in NMO is bilateral.

The clinical course of NMO is variable. It may occur as a monophasic illness that is either fulminant and fatal or associated with varying degrees of recovery. Polyphasic courses characterised by relapses and remissions also occur.

A curative treatment for NMO does not exist to date and management, like in MS, focuses on remission and prevention of relapse.

Treatment of an acute attack is with steroids: 5 days of IV methylprednisolone at a dose of 1g, followed by an oral steroid taper. Maintenance therapy is normally with azathioprine, rituximab, or mycophenolate mofetil.

Question 155 of 189

A 45-year-old man presents to the emergency department with the worst headache he has ever head. He reports he was sitting with his wife when he felt sudden onset pain at the back of his head. The pain reached 10/10 intensity rapidly. He also feels nauseous but has not vomited yet.

His past medical history includes adult dominant polycystic kidney disease and hypertension for which he takes ramipril. On examination, his Glasgow coma scale is 15/15 and there is no focal neurology.

You are concerned regarding an intracranial bleed and request a non-contrast CT head, which demonstrates hyperdensity in the subarachnoid space and ventricles.

Given the diagnosis, what is the most appropriate definitive intervention?

A. Thrombectomy
B. External ventricular drain
C. Haematoma evacuation
D. Aneurysm clipping
E. Aneurysm coiling

E. Aneurysm coiling

Following a subarachnoid haemorrhage, most intracranial aneurysms are now treated with a coil by an interventional neuroradiologist

The description given is of a thunderclap headache which is synonymous with a subarachnoid haemorrhage. Further evidence to support this is the CT findings of fresh blood in the subarachnoid space. The likely aetiology is an aneurysmal bleed given the background of ADPKD which is associated with Berry aneurysms. Most aneurysms are now managed by interventional radiology with coils, therefore is the most appropriate definitive intervention.

Thrombectomy is used in at management of acute ischaemic stroke.External ventricular drains are used to treat complications such as hydrocephalus and therefore is not treating the aneurysm itself.

Haematoma evacuation via craniotomy is a neurosurgical procedure used in the management of moderate to large-sized subdural haematomas.

Aneurysm clipping by neurosurgery is also used to manage aneurysmal bleeds but is not the firstline treatment option.

Question 156 of 189

A 44-year-old man presents with a fever. He states that it came on a couple of days ago. Today he developed a headache. The headache is frontal, dull in character and was gradual in onset. He explains that he generally keeps well however did experience an episode of fever with associated joint pain a few weeks ago. On examination he has nuchal rigidity. You also note the presence of sensory loss within the right ulnar nerve distribution.

Blood results are as follows:

Hb	134 g/l	Na	143 mmol/l
Platelets	388 * 10 /l	K	3.7 mmol/l
WBC	14.2 * 10 /l	Urea	5.8 mmol/l
Neuts	7.4 * 10 /l	Creatinine	51 µmol/l
Lymphs	4.7 * 10 /l	CRP	82 mg/l
Glucose	6.2 mmol/l		

An ECG was performed:

12 lead ECG	First degree heart block

CSF results are as follows:

White cell count	104/mm*
White cell differential	95% lymphocytes
Glucose	2.8 mmol/l
Proteins	1250 mg/L (normal 15-450

What is the most likely pathogen?

A. Neisseria meningitidis
B. Streptococcus pneumoniae
C. Borrelia burgdorferi
D. Cryptococcus neoformans
E. Mycobacterium tuberculosis

C. Borrelia burgdorferi

Central nervous system Lyme disease can be diagnosed with CSF analysis. The results are similar to the 'typical' bacterial picture with low glucose and high protein, however in Lyme disease we would expect a lymphocytic pleocytosis

The patients CSF results are suggestive of a bacterial infection with a glucose < 50% of plasma and a very high protein. Common bacterial infections (such as Pneumococcus and Meningococcus) usually result in a neutrophilic pleocytosis. However in this case the patient has a lymphocytic pleocytosis. Causes of this type of picture include partially treated bacterial infection, Lyme disease, tuberculosis, and Cryptococcus.

The extra-CNS clinical features of arthralgia, peripheral neuropathy and first degree heart block are suggestive of Lyme disease. This diagnosis is further supported by the CSF results. Borrelia burgdorferi is the bacteria which causes Lyme disease. Intravenous administration of ceftriaxone is the treatment of choice for disseminated Lyme disease.

A partially treated bacterial infection (e.g. Pneumococcus and Meningococcus) could results in the CSF results given, however this patient has yet to be started on treatment.

Mycobacterium tuberculosis and Cryptococcus neoformans could also result in this CSF picture, however they are less common generally, and tend to occur in specific patient cohorts with certain risk factors such as HIV. They are less likely diagnoses in this case

Question 157 of 189

A 67-year-old woman presents to her GP with progressive numbness and difficulty walking. Furthermore her daughter who was present mentions that she has been behaving strange over the past few months.

She is otherwise fit and well, apart from a ileal resection for treatment-resistant Crohn's disease 9 years ago.

Laboratory tests showed a low haematocrit and mean corpuscular volume of 110 fL. Blood smear analysis noted macrocytic red blood cells with hypersegmented neutrophils.

Which of the following is the most likely cause of the patient's presentation?

A. Ferrochelatase deficiency
B. Folate deficiency

C. Intrinsic factor deficiency
D. Iron deficiency anaemia
E. Cobalamin deficiency

E. Cobalamin deficiency

The ileal resection suggests that the patient is not absorbing the vitamin B12-intrinsic factor complex, leading to vitamin B12 deficiency (aka. cobalamin deficiency), and subsequently subacute combined degeneration. This is further supported by the macrocytic anaemia with hypersegmented neutrophils. Folate deficiency causes the same blood film picture but does not present with neurological symptoms.

Question 158 of 189

A 85-year-old lady presents to her GP with patchy dysesthesia affecting her feet bilaterally. She doesnt complain of any back pain and there has been no change in her bowel habit. She has stress incontinence, but no recent change in her urinary symptoms. On examination there is no weakness and reflexes are preserved. Her past medical history includes hypertensive nephropathy and diet controlled type two diabetes. She takes amlodipine 10mg, ramipril 5mg and bendroflumethazide 2.5mg for her hypertension and for the last year nitrofurantoin 100mg as prophylaxis for urinary tract infections.

Na	135 mmol/l
K	4.2 mmol/l
Urea	10.8 mmol/l
Creatinine	186 µmol/l
Glucose	8.3 mmol/l

What is the most likely cause for her symptoms?

A. Nitrofurantoin induced peripheral neuropathy
B. Uraemic peripheral neuropathy
C. Diabetic neuropathy
D. B12 deficiency
E. Thiazide induced peripheral neuropathy

A. Nitrofurantoin induced peripheral neuropathy

Nitrofurantoin can cause a peripheral neuropathy, especially in patients with pre-existing renal impairment. Painful neuropathy can be caused by both uraemia and diabetes but would not be expected without more advanced disease.

Question 159 of 189

A 64-year-old woman attends neurology clinic after referral from her GP with intermittent episodes of facial pain. She had first noticed symptoms around 10 months previously and recalled her first attack had occurred when taking her grand-children to a fireworks display. Pain episodes were described as a severe 'sawing' pain affecting the right side of her face that lasted one to two minutes before resolving completely. Since the onset the patient reported suffering from at least one attack on most days, with the frequency seeming to increase over time. On direct questioning she reported that on some occasions her nose had become blocked and she had felt sweaty during an episode but that these symptoms were not a major concern in comparison to the severe pain. The patient denied any other symptoms either at the time of the attack and was otherwise in good health with no recent history of weight loss. There was no family history of neurological disease.

A full neurological examination was performed with particular findings of normal pupillary reflexes, visual acuity and colour vision. There was no evidence of facial sensory deficit or facial nerve palsy. A peripheral neurological examination was likewise unremarkable.

Helpfully (although uncomfortably for the patient), a new episode of pain began during the course of the consultation. A further examination was conducted and conjunctival injection and slight eyelid oedema of the right eye were noted. In addition, the patient was noted to be sweating, markedly more profuse on the right side of her face. She also confirmed the onset of a blocked nose sensation at the onset of pain.

What is the most likely diagnosis causing the patients symptoms?

A. Trigeminal neuralgia
B. Multiple sclerosis
C. Horners syndrome
D. Short unilateral neuralgiform pain with autonomic symptoms
E. Atypical trigeminal neuralgia

D. Short unilateral neuralgiform pain with autonomic symptoms

The patients history certainly has features suggestive of trigeminal neuralgia, in particular the character and episodic nature of the pain and that the first episode was particularly memorable. However, the presence of autonomic features associated with attacks points strongly towards thevariant short unilateral neuralgiform pain with autonomic symptoms (SUNA) as the correct diagnosis. The distinction is clinically relevant as individuals with autonomic symptoms achieved less benefit from microvascular decompression compared to individuals with trigeminal neuralgia.

Atypical trigeminal neuralgia is the diagnosis made when an individual suffers from persistent lower intensity pain between exacerbations, in contrast to the complete resolution of pain seen in trigeminal neuralgia. Horners syndrome would not cause episodic autonomic symptoms and is not normally associated with pain. Multiple sclerosis is an important differential for trigeminal neuralgia but the patients normal baseline neurological examination and prominent autonomic symptoms make this less likely. If uncertainty persists after clinical assessment, MRI scanning to assess for demyelination plaques is the appropriate investigation.

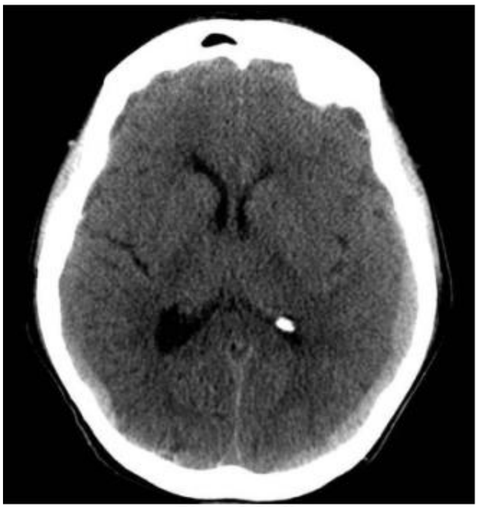
Question 160 of 189

An 81-year-old woman attends the emergency department following a mechanical fall. She has sustained a head injury and a CT head is arranged as part of her trauma survey. Her past medical history includes osteoarthritis of the right hip, recurrent falls, and atrial fibrillation for which she is anticoagulated.

On examination, her respiratory rate is 16/min and is saturating at 96% on air. Her heart rate is 81/min and her blood pressure is

91/64mmHg. Neurological examination elicits a Glasgow coma score of 14 due to confusion but there are no focal neurological deficits. She has no spinal, limb, chest, or abdominal tenderness, and a full painless range of movement in all four limbs.

Her CT head is displayed below:



What is the diagnosis in this instance?

A. Extradural haematoma
B. Intracerebral haematoma
C. Normal appearances
D. Subarachnoid haemorrhage
E. Subdural haematoma

E. Subdural haematoma

This woman has sustained a subdural haematoma. She is at increased risk of sustaining such an insult due to her advanced age and anticoagulation. Note that alcohol excess is also considered a risk factor. The diagnosis is confirmed on the CT scan which shows a concave bleed posteriorly.

An extradural haematoma typically presents with a convex-shaped bleed as it requires more force to detach the dura mater from the cranium. This CT shows a concave shape and is therefore consistent with a subdural rather than an extradural haematoma.

An intracerebral haematoma, as the name suggests, refers to a bleed within the brain parenchyma. As the bleed in this CT is of convex shape along the edge of the cranium, this is not indicative of an intracerebral haematoma.

This CT does not have normal appearances as it displays a haematoma posteriorly. This is therefore an incorrect answer.

A subarachnoid haemorrhage is not the correct answer here. CT images in subarachnoid haemorrhage show blood within the sulci of the brain parenchyma which is not the case here where the bleed is confined to the meninges.

Question 161 of 189

A 32-year-old male presents to clinic with shooting pain down both legs for one week and a mildly weak right hand. He does not recall ever having any other remarkable neurological symptoms and has not been diagnosed with any chronic medical illnesses.

He is right handed, and on examination, he has reduced fine motor control in the right hand with a brisk brachioradialis reflex on the right side. He also has subjective sensory disturbance over his trunk, but no objective sensory loss.

You suspect multiple sclerosis (MS), which findings are required to make the diagnosis of relapsing remitting MS?

A. A gadolinium enhancing lesion on MRI in a typical region for MS 1 not)
B. At least 2 separate MRI lesions typical of MS that are of different ages (i.e 1 enhancing and
C. Oligoclonal bands in CSF
D. Optic neuritis on examination and abnormal visual evoked potentials
E. Observe the patient and await a second clinical episode before a diagnosis can be made

B. At least 2 separate MRI lesions typical of MS that are of different ages (i.e 1 enhancing and

McDonald's criteria are MRI criteria used in the diagnosis of multiple sclerosis. Introduced in 2001, revised in 2005 and again recently in 2010. The latest revision improves sensitivity from 46-74% with a slight trade-off in specificity (slight deterioration from 94-92%)

The diagnosis of multiple sclerosis requires establishing disease disseminated in both space and time.

Dissemination in space:

Dissemination in space requires at least 1 T2 bright lesion in two or more of the following locations:

- periventricular
- juxtacortical
- infratentorialspinal
- cord Dissemination in time:

Dissemination in time can be established in one of two ways:

- A new lesion when compared to a previous scan (irrespective of timing) - T2 bright lesion and/or gadolinium-enhancing lesion
- ii) Presence of an asymptomatic enhancing lesion and a non-enhancing T2 bright lesion on any one scan (i.e 2 lesions of differing ages that satisfy the dissemination in space criteria)

Question 162 of 189

A 28-year-old lady presents to the emergency department with a 4-day history of generalised headache that is worse on lying down. She reports it has gradually become worse and she has also noticed blurred vision since yesterday. Her past medical history includes chronic back pain, acne and anxiety. On examination, she is noted to be overweight. Fundoscopy shows papilloedema. Further investigations point to a diagnosis of idiopathic intracranial hypertension.

Which of the following of her medications is associated with this condition?

A. Tramadol
B. Tetracycline antibiotics
C. Diazepam
D. Amitriptyline
E. Ibuprofen

A. Tetracycline antibiotics

Associated medications include, but are not limited to:

- tetracycline antibiotics
- isotretinoin
- contraceptives steroids
- levothyroxine
- lithium
- cimetidine

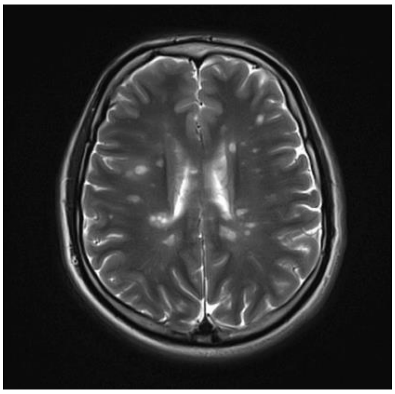
Question 163 of 189

A 24-year-old man is referred to the neurology clinic by his GP. He describes a four month history of left-sided numbness and intermittent tingling. This mainly affects his left arm but he occasionally has symptoms in the left leg. There is no history of headaches, visual problems or weakness.

His GP has performed a series of blood tests including a full blood count, urea and electrolytes, vitamin B12 and C reactive protein, all of which were normal.

Neurological examination today was normal other than reduced sensation in the left C6/7 dermatome.

A MRI head is performed. The T2 images are shown below:



What is the most likely diagnosis?

A. Creutzfeldt-Jakob disease
B. Multiple sclerosis
C. Adrenoleucodystrophy
D. Cerebral toxoplasmosis
E. Gliolastoma multiforme

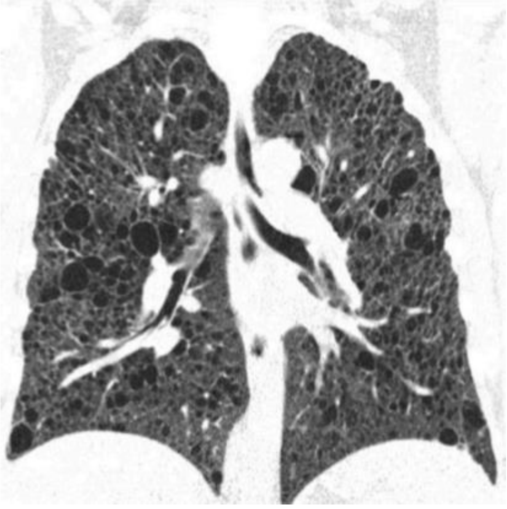
B. Multiple sclerosis

The MRI scan is consistent with multiple sclerosis. Widespread periventricular, juxtacortical, post fossa and upper cervical cord high T2 regions are noted. Note the difference in the lesions with varying degrees of contrast enhancement and restricted diffusion indicating active/recent demyelination. This satisfies the diagnostic criteria in terms of separation in terms of time space.

Question 164 of 189

A 39-year-old woman with known tuberous sclerosis is referred to the respiratory clinic after developing progressive dyspnoea. Her GP had requested a chest x-ray which showed significant changes.

The CT scan is shown below:



What complication has developed?

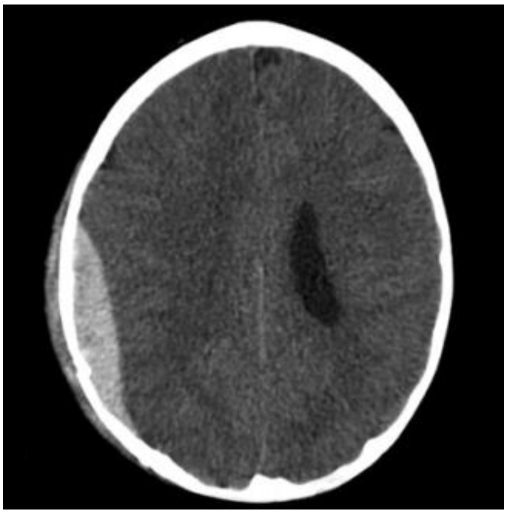
A. Metastatic angiomyolipoma
B. Lymphangioleiomyomatosis
C. Lung rhabdomyomas
D. Lung angiofibromas
E. Subependymal giant cell astrocytoma

B. Lymphangioleiomyomatosis

The CT demonstrate innumerable small regular lung cysts diffusely distributed throughout the lungs, the typical appearance of lymphangioleiomyomatosis (LAM). This disorder may occur alone on in association with tuberous sclerosis.

Question 165 of 189

A 7-year-old boy falls from the top of a slide and is taken to the Emergency Department. He is crying, complaining of a severe headache and regularly vomiting. A CT head is performed:



What does the scan show?

A. Subarachnoid haemorrhage
B. Brain contusion
C. Extradural haematoma
D. Subdural haematoma
E. Meningioma

C. Extradural haematoma

The CT scan demonstrates a bi-convex (lentiform) extra-axial hyperdense collection consistent with an extradural haematoma.

Question 166 of 189

A 62-year-old woman comes to the gastroenterology clinic for review. She has a history of watery diarrhoea over the course of the past 6 months, where she is opening her bowels some 4-6 times per day. There is other past medical history of hypertension, ischaemic heart disease, Type 2 diabetes and depression.

Investigations

Hb	11.5 g/l	Na	139 mmol/l	Bilirubin	12 µmol/l
Platelets	207 * 10 /l	K	3.9 mmol/l	ALP	95 u/l
WBC	8.9 * 10 /l	Urea	7.2 mmol/l	ALT	23 u/l
Neuts	5.6 * 10 /l	Creatinine	100 µmol/l	γGT	56 u/l
Lymphs	1.8 * 10 /l	Albumin	38 g/l		
Eosin	0.5 * 10 /l				

Colonoscopy: Mild mucosal oedema only, biopsy reveals lymphocytic infiltration

Which of the following agents is most likely to be the cause of her colonoscopy findings?

A. Amlodipine
B. Atorvastatin
C. Lisinopril
D. Metformin
E. Sertraline

E. Sertraline

Sertraline is recognised as highly likely to be associated with lymphocytic colitis, (microscopic colitis associated with lymphocytic infiltration). This conclusion results from a mix of association / epidemiology studies, and case series where removal and re-introduction of medication has been correlated with symptoms and changes in biopsy appearance.

Calcium channel antagonists are not thought to be associated with microscopic colitis, but are uncommonly reported as leading to change in bowel habit including diarrhoea. Metformin leads to bile salt malabsorption which may result in diarrhoea. Statins may be associated with microscopic colitis, although withdrawal and re-challenge evidence is less strong than for sertraline. ACE inhibitors are not known to be associated with microscopic colitis. Diarrhoea was a commonly reported adverse event in lisinopril clinical trials, but this is not thought to be due to microscopic colitis.

Question 167 of 189

A 32-year-old woman presents to clinic with a constant headache. She has attempted to control this by taking daily paracetamol, ibuprofen and codeine for the last two months. Her headache has not improved. She describes the headache as being present almost every single day but it is not disabling. There is a concern that she may have a medication overuse headache.

What is the most appropriate management?

A. Wean off codeine. Stop paracetamol and ibuprofen abruptly
B. Stop codeine abruptly. Reduce paracetamol and ibuprofen gradually
C. Wean off all analgesia gradually
D. Wean off codeine and ibuprofen.
E. Stop paracetamol abruptly Stop all medications abruptly

A. Wean off codeine. Stop paracetamol and ibuprofen abruptly

The duration of the headache and regular use of codeine makes the diagnosis of medication overuse headache most likely.

The correct answer is to stop paracetamol and ibuprofen abruptly, but gradually wean off codeine due to the adverse effects of acute opioid withdrawal. Whilst codeine and triptans are the most common cause, other types of analgesia can contribute. Improvement is often seen within a few days.

Question 168 of 189

A 62-year-old Afro-Caribbean female is brought to the Emergency Department by husband after a 3-day history of unusual, odd behaviour at home. The family had hosted a dinner party that evening and the patient was reported to have been very disinhibited, agitated and describing hallucinations around her guests. In the past 3 weeks, she had two episodes of generalised seizures witnessed by her husband, each lasting for up to 5 minutes before spontaneously terminating, associated with urinary incontinence and tongue biting. She has no history of epilepsy and is not on any regular medications, but was diagnosed with ovarian teratoma two years ago. On examination, she has no focal neurology but you note a dystonic orofacial movement disorder. A CT head was unremarkable, with no acute infarct, haemorrhage or space occupying lesion demonstrated.

Which investigation is most likely to produce the diagnosis?

A. MRI head
B. Lumbar puncture
C. Anti-NMDA receptor antibodies
D. Anti-MuSK antibodies
E. Anti-GM1 antibodies

D. Anti-MuSK antibodies

The syndrome described is strongly suggestive of an autoimmune-mediated limbic encephalitis.

Question 169 of 189

A 32-year-old female is seen in neurology clinic due to a 4-month history of headaches. She described having headaches most days on waking up, which were throbbing in nature. She found that they eased off after mobilising and that coughing made them worse. She reported a couple of episodes of blurred vision on waking, but no nausea or vomiting. Her general practitioner arranged for an outpatient magnetic resonance head scan which was normal. Her body mass index (BMI) had been 27 kg/m², but on advice from her general practitioner she had lost weight, and her BMI was now 23 kg/m². She was not on any regular medications, other than paracetamol and ibuprofen which she had been using regularly for her headaches.

There was no focal neurological deficit on examination, and her visual acuity was normal. On fundoscopy, there was a mild degree of papilloedema. Blood pressure was 125/82mmHg. Blood tests were unremarkable.

What is the most appropriate next step in management?

A. Lumbar-peritoneal shunt
B. Optic nerve fenestration
C. Cessation of paracetamol and ibuprofen
D. Acetazolamide
E. Sumatriptan

D. Acetazolamide

The postural nature of this patient's headaches, and the fact that they are exacerbated by coughing are suggestive of raised intracranial pressure. In a young woman with a normal magnetic resonance scan, this is suggestive of idiopathic intracranial hypertension.

In overweight patients, the initial management strategy is weight loss, which may be enough to relieve symptoms. This patient has already lost weight and has a normal body mass index, but her headaches persist, so additional measures are required. Cessation of any causative agents would also be important (e.g. tetracyclines, retinoids, lithium).

Acetazolamide is useful in controlling mild disease that is resistant to the conservative measures mentioned above. Repeated lumbar puncture may also be used to lower intracranial pressure. In acute disease, corticosteroids are sometimes beneficial. If there is significant visual loss despitemedical therapy surgical interventions such as optic nerve sheath fenestration or lumbarperitoneal shunt insertion may be necessary.

Analgesia-induced headache and migraine are important causes of recurrent headache, but would not cause the postural association seen in this case, so cessation of analgesic agents and commencement of sumatriptan are not appropriate treatments.

Question 170 of 189

A 35 year old mechanic attends the emergency department following an injury at work. He has suffered a serious laceration to the upper arm. While suturing, the doctor notices multiple cuts and burns on both arms.

On examination there is marked wasting of brachioradialis and the small muscles in both hands, with mild hyporeflexia of the biceps and brachioradialis tendons. He is weak in both arms, distally more so. His lower limb and cranial nerve examination is unremarkable. On testing upper limb sensation, vibration and proprioception are intact but there appears to be reduced pain and temperature sensation over the C3/C4/C5 deramatomes.

What is the most useful investigation?

A. Lumbar puncture
B. Nerve conduction studies
C. MRI Brain
D. Electromyography
E. MRI cervico-thoracic spine

E. MRI cervico-thoracic spine

The presence of loss of pain and temperature sensation in a 'cape like' distribution is highly suggestive of syringomyelia. A fluid filled cavity within the cord develops, typically between C2 to T9, compressing the cord from inside to out. Consequently the spinothalamic tracts are the first to be affected in a symmetrical distribution.

There are a number of aetiologies, but it should be noted that therw is an association with Arnold Chiari malformation Type 1. It is sensible to investigate for a compressive cord lesion in the first instance; imaging of the brain may be considered if syringomyelia is detected. An MRI cervicethoracic cord would be the most sensitive method of detecting syringomyelia.

Question 171 of 189

A 78-year-old man is brought to see you in clinic by his daughter. He has a diagnosis of Alzheimer's dementia and although currently coping well, has significantly impaired short-term memory.

Currently, his daughter attends daily for all meals, cleaning, and shopping. He continues to live in his own home and is alone overnight. She has, however, become concerned as he continues to drive 5 miles three times a week and she is unsure if this is safe.

Which of the following is the best course of action regarding his driving licence?

A. He is disqualified from driving
B. He is allowed to drive, but only if daughter is present
C. There is no need to contact the DVLA, continue to drive
D. Assess his risk factors, report to the DVLA and await advice
E. He is safe if his mini mental state examination score >11

D. Assess his risk factors, report to the DVLA and await advice

The DVLA guidance states this is a difficult decision to make. The presence of impaired short-term memory, disorientation or loss of insight suggests someone with dementia is not fit to drive.

In this case, the correct answer would be to contact the DVLA, assess the risk factors and take further advice. He should not be immediately disqualified; he warrants a more careful assessment and decision with support from the DVLA. The factors that suggest someone with dementia may not be safe to drive include significantly impaired short-term memory, poor attention and concentration, and impairment of planning.

Question 172 of 189

A 24 year-old university student presents with a 10 day history of visual disturbance. He reports changes in his visual acuity where he has been experienced some blurring of his vision which is considerably worse after having been for a run. He thinks it is related to some pain he developed on his right eye prior to the onset of his vision loss. He has no previous history of visual problems and is normally fit and well. He takes no regular medications.

On further questioning he reports that 6 months ago he had some sensory changes on his lower limbs. He felt they were numb and this persisted for a few weeks. He thought it was due to his sporting activities, as he is an avid hockey player.

He drinks 20 units of alcohol at the weekends and does not smoke. His blood results are shown below:

Hb	134 g/l
Platelets	250 * 10 /l
WBC	8 * 10 /l
Na	134 mmol/l
K	4.1 mmol/l
Urea	4 mmol/l
Creatinine	60 µmol/l

Which investigation below would be most useful to diagnose this patient?

A. EEG
B. CT head
C. HIV serology
D. Contrast MRI brain and spine
E. Anti-NMO antibodies

D. Contrast MRI brain and spine

This young man presents with the classic symptoms of optic neuritis. Changes in vision(usuallyblurriness but can occasionally present as loss of red colour vision). He describes Uhthoff's phenomenon.

His history of a previous episode of paraesthesia is consistent with a previous first CIN episode. This gentleman requires urgent investigations. He should have a lumbar puncture which would show oligoclonal bands- which should be paired with a serum sample for comparison.

The correct answer is contrast MRI brain and spine and this will show a gadolinium enhancing consistent with an acute demyelinating lesion. Most likely it would also show an old lesion in his cord which would be diagnostic of MS.

Neuromyelitis optica is a demyelinating condition associated with NMO antibodies (Aquaporin 4 antibodies) which presents with demyelination of the optic nerve and spinal cord. It follows a more severe course than traditional MS. In this instance, an MRI with contrast will be the most appropriate investigation to yield a diagnosis.

Question 173 of 189

A 31-year-old woman is referred to the neurology team after experiencing two seizures, having had no prior episodes. After a consultation and discussion, they agree for her to trial carbamazepine. She has a past medical history of asthma and gastro-oesophageal reflux disease, for which she uses salbutamol and beclometasone inhalers, montelukast, cimetidine and omeprazole; she also takes the combined oral contraceptive pill.

Three weeks later, she experiences several further seizures, despite being fully adherent to the carbamazepine regime.

Which medication may have led to the return of her episodes?

A. Carbamazepine
B. Cimetidine
C. Combined oral contraceptive pill
D. Montelukast
E. Omeprazole

A. Carbamazepine

Carbamazepine is known to exhibit autoinduction. hence when patients start carbamazepine they may see a return of seizures after 3-4 weeks of treatment

Carbamazepine is a commonly-used antiepileptic medication. It is also used for trigeminal neuralgia and bipolar disorder. Crucially, it is known to induce the P450 enzyme system, which breaks down the drug itself, and so it is considered an auto-inducer. As such, 3-4 weeks after treatment, it may lead to the return of seizures.

Cimetidine is an antihistamine H2-receptor antagonist, used for the control of gastric acid production. It is a P450 inhibitor, not an inducer, and so would not reduce the effectiveness of carbamazepine.The combined oral contraceptive pill is metabolised by the P450 system. It is not known to significantly induce or inhibit the P450 system and so is incorrect.

Like the pill, montelukast is also known to be metabolised by the P450 system. Similarly, it is not known to induce or inhibit the system and so is not the correct answer.

Omeprazole is considered a P450 inhibitor, rather than inducer. As such, it would not cause a reduction in carbamazepine levels and so would not be the cause of seizures returning.

Question 174 of 189

A 51-year-old patient with a history of schizophrenia presents after being found on the floor of his sheltered accommodation home surrounded by many packets of medications. There is no collateral history. On examination, the patient is only verbally responsive to pain. There is evidence of vomitus around the oral cavity. You note that he is generally tremulous. Observations show heart rate 110/min, blood pressure 101/61 mmHg.

On the 12 lead ECG the P waves are present followed by QRS complexes. PR interval 0.12 seconds, QRS 0.12 seconds, corrected QT 0.48 seconds.

What is the most likely causative agent?

A. Citalopram
B. Chlorpromazine
C. Diazepam
D. Digoxin
E. Cocaine

A. Citalopram

Other side effects of citalopram overdose to be aware of include:

- Nausea Vomiting Tremor
A. Nystagmus Convulsions

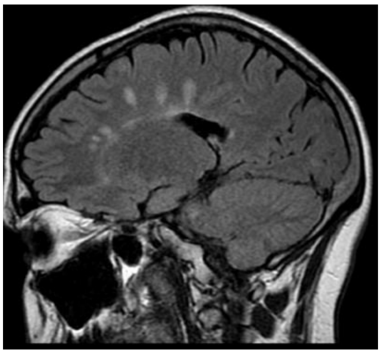
Management of SSRI poisoning is generally supportive. Convulsions can be managed with benzodiazepines.

Question 175 of 189

A 31-year-old woman is reviewed in the neurology clinic. For the past 6 months she has been presenting to her GP with a variety of symptoms including lethargy, heat intolerance, pins/needles and limb numbness. For the past 4 weeks she has also been complaining of shooting pains in her right hand.

Her GP has arranged a number of blood tests including thyroid function tests and vitamin B12 which were reported as normal.

A neurological examination today demonstrates no consistent neurological findings. A MRI is requested:



What is the most likely diagnosis?

A. Multiple sclerosis
B. Pituitary tumour
C. Ependymoma
D. Acute disseminated encephalomyelitis (ADEM)
E. Systemic lupus erythematosus

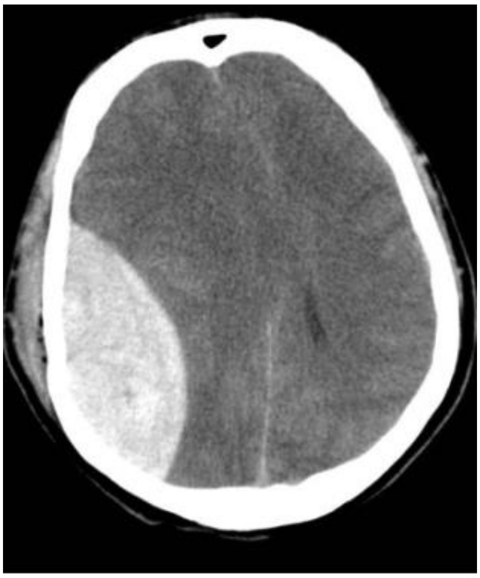
A. Multiple sclerosis

The MRI shows multiple white matter plaques penpendicular to the corpus callosum giving the appearance of Dawson fingers - a classic MRI finding in multiple sclerosis.

Lethargy is a clue in this question. It is very common feature in patients with multiple sclerosis. Other features such as pain and heat intolerance and also seen.

Question 176 of 189

A 27-year-old woman is admitted to the Emergency Department following a road traffic accident. Primary survey reveals a right-sided haemothorax but she is haemodynamically stable. She is only responding to voice. A CT scan of her head (with contrast) is performed:



What does the scan show:

A. Subdural haematoma

B. Subarachnoid haemorrhage
C. Extradural haematoma
D. Brain contusion
E. Meningioma

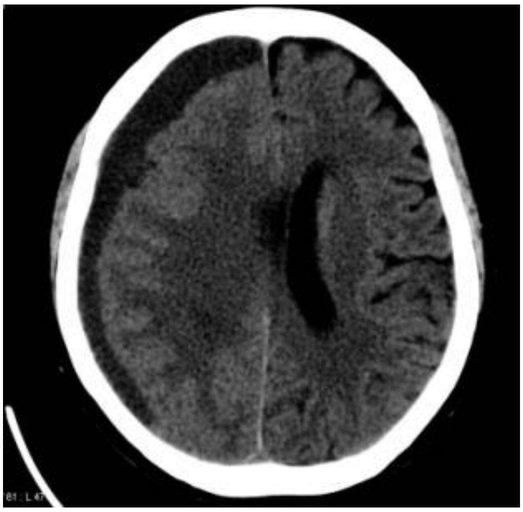
C. Extradural haematoma

The CT scan shows a extra-axial biconvex collection associated with fracture suggestive of extradural haematoma.

Question 177 of 189

A 73-year-old man who is a chronic alcoholic is brought to the Emergency Department by an ambulance. He was found collapsed in the street. On admission his airway, breathing and circulation are satisfactory. His GCS is 13/15 (eyes = 3, verbal = 4, movement = 6) although his level of consciousness appears to be fluctuating. There are no obvious signs of external head injury.

A CT head (without contrast) is performed:



What is the most likely diagnosis?

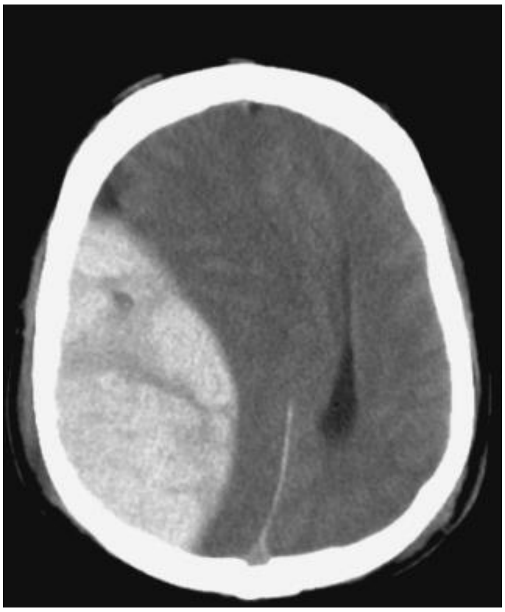
A. Subarachnoid haemorrhage
B. Wernicke's encephalopathy
C. Subdural haematoma
D. Meningioma
E. Extradural haematoma

C. Subdural haematoma

CT scan demonstrates a right sided chronic subdural haemorrhage resulting in midline shift.

Question 178 of 189

A 30-year-old man who has been seriously assaulted is brought to the Emergency Department. His GCS is 5/15 on arrival and he is immediately intubated and transferred for a CT head (with contrast):



Which blood vessel(s) is most likely to have ruptured?

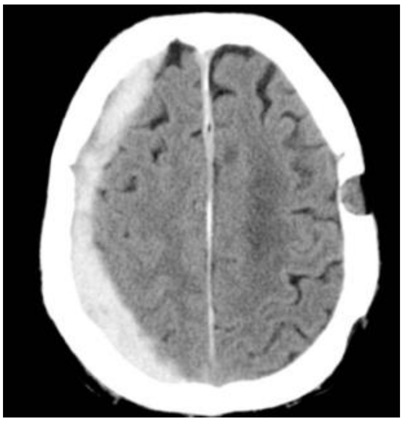
A. Anterior communicating artery
B. Emissary veins
C. Temporal artery
D. Middle meningeal artery
E. Posterior communicating artery

D. Middle meningeal artery

This scan shows a massive extradural haemorrhage, most likely caused by a ruptured middle meningeal artery.

Question 179 of 189

An 85-year-old complains of severe headaches and lethargy over the past 3 weeks. His past medical history includes atrial fibrillation for which he takes warfarin. A CT head (with contrast) is performed:



What is the most likely diagnosis?

A. Normal age-related changes
B. Extradural haematoma
C. Subdural haematoma
D. Meningioma

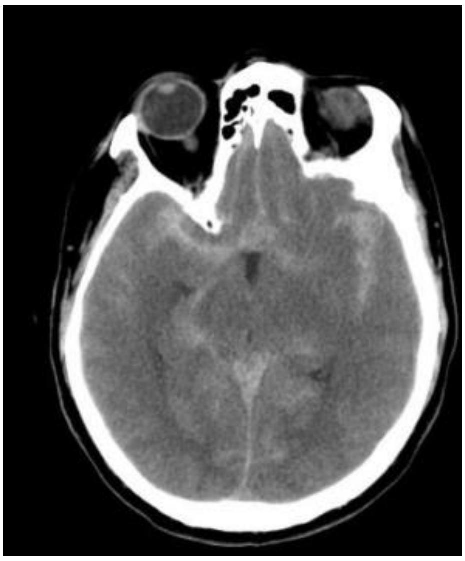
E. Subarachnoid haemorrhage

C. Subdural haematoma

The CT scan shows a crescent-shaped (concave surface away from the skull) extra-axial collection seen with subdural haematomas. A burr hole can also be seen on the left.

Question 180 of 189

A 33-year-old woman presents to the Emergency Department with a severe headache. She describes falling to the ground whilst walking to work and since having a severe occipital headache. Her GCS is 15/15 and her vital signs are stable, other than a low grade pyrexia of 37.5ºC. Her headache fails to improve with analgesia and a CT head with contrast is performed:



What does the scan show?

A. Extradural haematoma
B. Brain contusion
C. Subdural haematoma
D. Meningioma
E. Subarachnoid haemorrhage

E. Subarachnoid haemorrhage

The correct answer is Subarachnoid haemorrhage. This condition typically presents with a sudden onset, severe headache (often described as a 'thunderclap' headache). The patient's symptoms of collapsing and having an occipital headache are consistent with this. A subarachnoid haemorrhage can also cause fever due to blood being irritating to the meninges, which could explain the patient's low-grade pyrexia. On CT imaging, subarachnoid haemorrhage appears as areas of high density in the subarachnoid space.

The first incorrect option is Extradural Haematoma. This usually results from trauma leading to bleeding between the dura mater and skull. It often presents with a lucid interval followed by rapid neurological deterioration. The CT appearance is typically a lens-shaped hyperdensity that does not cross suture lines, which does not match the image shown.

The second incorrect option is Brain contusion. This refers to bruising of brain tissue following head injury. The clinical presentation might include loss of consciousness at the time of injury and focal neurological deficits depending on the area affected. On CT imaging, brain contusions appear as irregular areas of low density due to oedema and high density due to haemorrhage; however, these findings are not evident in this case.

Subdural Haematoma, the third incorrect option, occurs when there is bleeding into the space between the dura mater and arachnoid membrane, most commonly due to tearing of bridging veins during trauma. Clinically, patients may present with fluctuating consciousness level or focal neurological deficits. On CT scan, it appears as a crescent-shaped hyperdensity that can cross suture lines but not dural reflections - again inconsistent with our image.

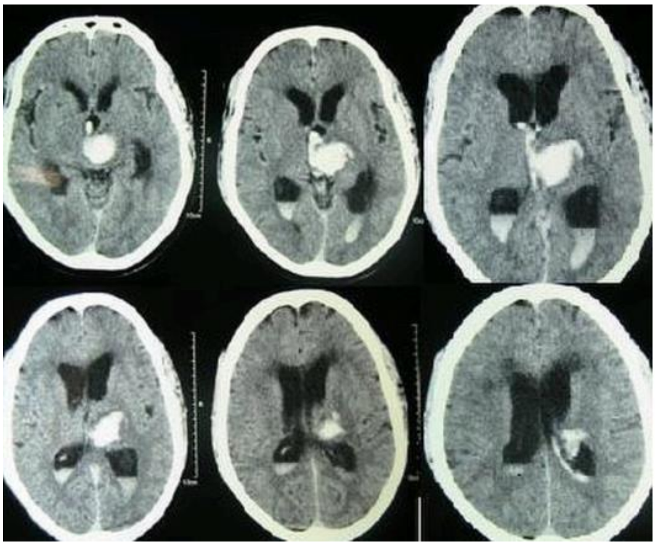
Lastly, Meningioma, another incorrect option, refers to benign tumours arising from meningeal layers surrounding the brain and spinal cord. Patients usually present with slowly progressive symptoms related to increased intracranial pressure or focal neurologic deficits based on tumour location. Meningiomas on CT scans appear as extra-axial masses that are typically homogenous and isodense or slightly hyperdense compared to brain parenchyma - features not seen in this case.

Question 181 of 189

A 72-year-old woman patient presents to the emergency department with a sudden onset of severe headache, worse than any she's ever experienced, which started an hour ago. She also reports weakness on the right side of her body and difficulty speaking. There is no history of trauma.

Her medical history reveals longstanding hypertension, which she admits to not managing well in recent months. On examination, she appears drowsy. Her blood pressure is 195/110 mmHg. Her neurological examination reveals right-sided hemiparesis and expressive aphasia.

Urgent head imaging is ordered.



What is the most likely diagnosis?

A. Brain tumour

B. Intracerebral and intraventricular hemorrhage
C. Intracerebral and subarachnoid haemorrhage
D. Ischaemic stroke
E. Subdural haemorrhage

B. Intracerebral and intraventricular hemorrhage

The correct answer is intracerebral and intraventricular haemorrhage. The CT shows bright hyper-dense intracerebral and intraventricular lesions, due to the presence of blood, which has a characteristic bright white appearance on CT imaging. An intracerebral hemorrhage is caused by the sudden rupture of a blood vessel within the brain. This leads to the accumulation of blood, causing damage to the surrounding brain tissue. Intraventricular hemorrhage represents bleeding inside the brain's ventricular system, which consists of interconnected spaces filled with cerebrospinal fluid (bleeding here is located within the lateral ventricles). Common presentations include a sudden severe headache, weakness, altered consciousness, and focal neurological deficits.

Brain tumour is incorrect. The rapid onset of symptoms in this case, paired with the CT findings and lack of previous history, are not in keeping with a brain tumour. Sometimes an intracerebral haemorrhage can be caused by an underlying brain tumour and further imaging is sometimes considered after the resolution of the haemorrhage.

Intracerebral and subarachnoid haemorrhage is an incorrect choice. While there are intraparenchymal and intraventricular haemorrhages on CT scans, no blood exists in the subarachnoid spaces. Subarachnoid haemorrhage typically results from the rupture of cerebral aneurysms, or, less commonly, from a sustained head trauma. Patients with subarachnoid haemorrhage typically present with a thunderclap headache, described as a sudden-onset headache usually occipital that is the worst headache of their life often accompanied by sensitivity to light (photophobia), signs of meningitis (meningism), and focal neurological deficits.

Ischaemic stroke although a potential differential, is ruled out in this case by the CT findings. The earliest CT sign visible for ischemic stroke is a hyperdense segment of a vessel, representing direct visualization of the intravascular thrombus/embolus and as such is visible immediately. Other signs include areas of low density in the grey and white matter of the territory. These changes may take time to develop and the CT may be normal initially. Clinically ischaemic stroke can be difficult to distinguish from haemorrhagic stroke.

Lastly, subdural haemorrhage is characterized by bleeding between the brain's surface and its outer covering, the dura mater, often caused by head trauma. They are thought to result from thestretching and tearing of bridging cortical veins as they traverse the subdural space to connect with a nearby dural sinus for drainage. It can lead to symptoms such as fluctuating levels of consciousness, headache, dizziness, and in severe cases coma. On CT, it presents with a crescentshaped homogeneously hyperdense extra-axial collection that spreads diffusely over the affected hemisphere.

Question 182 of 189

A 32-year-old woman presents with reduced sensation. She has noticed that slowly over six months she has not felt when hot water has splashed on her hands, despite blistering occurring afterwards. Her husband has become concerned and asked her to seek a medical opinion. She denies any other problems, including weakness, weight-loss or her activities of daily living being affected. She has a past medical history of asthma but only rarely needs her salbutamol inhaler. She has no other medications or allergies. On examination, she has sensory loss over her hands and arms when tested for temperature and pain. There is a dermatomal distribution affecting dermatomes C4 to C6 which is symmetrical. Cranial nerve and lower limb examination is normal. Spinal examination shows no tenderness.

What is the most likely diagnosis?

A. Multiple sclerosis
B. Cervical disc prolapse
C. Vasculitis
D. Myasthenia gravis
E. Syringomyelia

E. Syringomyelia

The correct answer is syringomyelia. The fact that she has primarily loss of spinothalamic function in a cervical nerve distribution with a distinct sensory loss makes syringomyelia the most probable diagnosis. Weakness can occur as well. Syringomyelia is caused by a tubular cyst within the central spinal cord, most commonly affecting the cervical region. It compresses corticospinal and spinothalamic tract as well as anterior horn cells. Presentation can be variable causing loss of temperature sensation, pain, paralysis, stiffness and weakness. Due to the common location of the cyst, this is most likely to affect hands, arms and shoulders. In half of the patients, there is only mild or no disability.

Multiple sclerosis is unlikely as there is no evidence of lesions separated by attacks. The absence of neck pain or tenderness makes a disc prolapse unlikely, whilst the absence of systemic features makes vasculitis unlikely. Myasthenia is associated with fatigability rather than a sensory loss.

Question 183 of 189

A 45-year-old Bangladeshi male presents with a 6-month history of bilateral reduced sensation on the tips of both his feet, which has gradually progressed on both legs to his low shins. His past medical history includes type 2 diabetes, diagnosed 7 years ago and reports good medication compliance with metformin 500mg BD alone, with a HbA1c of 48 mmol/mol (6.5%) two weeks ago. He is also currently in his ninth month of anti-tuberculosis treatment, having initially presented with a chronic cough, night sweats and weight loss. An induced sputum subsequently cultured positive for acid-fast bacilli. He did not bring in his medications but remembers being told they are 'the standard four then two drugs'. He takes no other medications and has no known drug allergies. On examination, tone, power and gait of his lower limbs are unremarkable. He demonstrates reduced sensation to light touch to his left lower shin and right mid-shin. Ankle jerks are absent bilaterally, plantars are downgoing bilaterally.

What is the most likely diagnosis?

A. Guillain-Barre syndrome (GBS)

B. Chronic inflammatory demyelinating polyneuropathy (CIDP)
C. Drug induced peripheral neuropathy
D. Diabetic neuropathy
E. Diabetic amyotrophy

C. Drug induced peripheral neuropathy

The patient describes a chronic sensory deficit in a peripheral nerve distribution. This is most likely isoniazid-induced peripheral neuropathy, classically interfering with vitamin B6 via an unknown mechanism. As a result, pyridoxine should be regularly prescribed in all patients taking isoniazid. GBS and CIDP classically present as combined motor and sensory syndromes, of duration less than and more than 4 weeks respectively. While diabetes-related peripheral neuropathy is possible, his glycosylated haemoglobin measurement, an accurate measure of the patient's serum glucose levels over the past 3 months, reflects very good control.

Question 184 of 189

A 28 year old female is referred to general medical clinic for review. She has a one year history of very severe right sided headaches. These are associated with eyelid swelling and lacrimation and never occur on the left. Paroxysms last around 2 minutes at a time and attacks occur upwards of 10 times per day. Attacks tend to occur every day for a week or two with a moth or so between attacks. She describes no nausea or vomiting associated with her headaches.

What treatment should you offer?

A. Low dose carbamazepine
B. Trial of indomethacin
C. Verapamil
D. Topirimate
E. Prednisolone

B. Trial of indomethacin

The history given above is one of severe, paroxysmal, unilateral headaches with associated autonomic features. The two most likely diagnoses would be cluster headache or paroxysmal hemicrania (PH), with the latter being more likely. It would be unusual for cluster headache attacks to last for less than 5 minutes and a daily frequency of greater than 10 is unusual for cluster headache but not PH. In addition, cluster headaches have a male predilection but PH tends to affect females. Paroxysmal hemicrania (or chronic paroxysmal hemicrania if attacks occur for over one year) tends to be highly responsive to treatment with indomethacin, so much so that a trial of this may be considered a diagnostic strategy.

Low dose carbamazepine is the treatment of choice for trigeminal neuralgia. Verapamil is first line for prophylaxis of cluster headaches. Topiramate may be used in migraine prophylaxis and oral steroids have been used to prevent cluster headaches but have little evidence to support their use.

It may be argued that due to the relative rarity of PH compared with cluster headaches that even with the history above the former may be more likely, however due to the diagnostic response to indomethacin seen in PH it would be appropriate to trial this first.

Question 185 of 189

A 42-year-old man presents to the emergency department with a sudden-onset, severe headache that he describes as the 'worst headache of his life'. He had a similar headache of lesser severity one week ago.

On examination, he appears to be in significant distress due to his pain and has significant neck stiffness. Full neurological examination is otherwise normal. Computerised tomography (CT) scanning of the brain is done within an hour, showing blood in the basal cisterns and sulci. CT angiography (CTA) confirms a posterior communicating artery aneurysm.

What is the most appropriate management for this patient?

A. Craniotomy and clipping
B. Digital subtraction angiography (DSA)
C. Endovascular coiling
D. Intravenous mannitol
E. Oral nimodipine

Question 186 of 189

A 48-year-old man presents to the emergency department with sudden onset, severe right eye pain and a headache. The pain started earlier this morning and worsened while watching television in a dark room. He also had 2 episodes of non-projectile vomiting. He has no significant past medical history except for migraine.

His vital signs are a temperature of 37 ºC, blood pressure 140/85 mmHg, pulse 55 beats/min and oxygen saturation 98% on room air. On examination, he is conscious and oriented but in distress due to severe pain. The pupils are unequal; the right pupil is semi-dilated and non-reactive to light.

Given the likely diagnosis, what is the definitive treatment for this patient?

A. Intravenous mannitol
B. Laser peripheral iridotomy
C. Laser trabeculoplasty
D. Oral propranolol
E. Tropicamide eye drops

B. Laser peripheral iridotomy

Laser peripheral iridotomy is the definitive treatment for acute angle-closure glaucoma

This scenario describes a patient with acute angle-closure glaucoma, for which peripheral laser iridotomy is the definitive treatment option. A history of sudden-onset eye pain precipitated by mydriasis (watching TV in a dark room), vomiting, and pupillary findings in this scenario point towards the diagnosis. This is confirmed by measuring intra-ocular pressure via tonometry and visualisation of the angle by gonioscopy. Emergency management includes drugs that reduce the secretion of aqueous humour (acetazolamide and timolol) and drugs that increase the outflow of aqueous humour (pilocarpine and apraclonidine). Laser peripheral iridotomy is the definitive treatment option as it creates a tiny hole in the peripheral iris, allowing aqueous humour to flow to the angle.Intravenous mannitol is not the definitive treatment for angle-closure glaucoma. Diuretics like mannitol and acetazolamide are sometimes used in medical management of acute angle-closure glaucoma to lower the intraocular pressure and symptomatic relief.

Laser trabeculoplasty lowers the intraocular pressure in open-angle glaucoma and has no role in treating acute angle-closure glaucoma. Unlike the patient's symptoms in this scenario, open-angle glaucoma exhibits no symptoms in the initial stage and only presents with problems in peripheral vision.

A sudden-onset headache and past history of migraine may suggest an acute migraine attack, which makes oral propranolol a reasonable option in this scenario, but a semi-dilated, nonreactive pupil points away from this.

Tropicamide eye drops will worsen this patient's symptoms as tropicamide causes pupillary block and worsens angle-closure glaucoma. Tropicamide is one of the causes of drug-induced acute angle-closure glaucoma.

Question 187 of 189

A 60-year-old woman presents to the Emergency Department with a 2-day history of vertigo and vomiting on movement. Each episode of vertigo lasts for around 1 minute after head movements and then resolves if she remains still. She awoke in the night with the symptoms and they have remained unchanged since. She was completely well prior to this and has no other past medical history. Her hearing is unaffected and she has no other symptoms affecting her ears, nose, or throat.

What proportion of patients with this condition will have an improvement in their symptoms after a single particle repositioning manoeuvre?

A. 10 %
B. 100 %
C. 20 %
D. 50 %
E. 80 %

E. 80 %

The Epley manoeuvre is successful in treating around 80% of cases of BPPV Important for me Less important

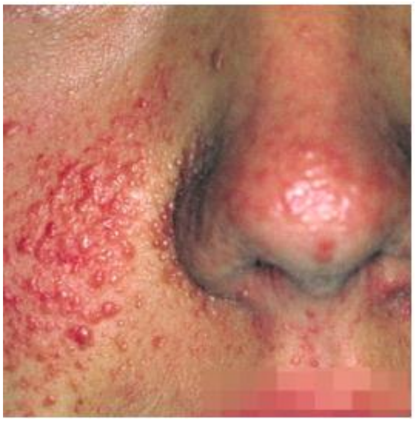
This patient has benign paroxysmal positional vertigo (BPPV). This condition causes the onset of vertigo after head movements and is due to the presence of calcium debris loose within the semicircular canals. This condition is more common in women, and in people over the age of 60. Without treatment, episodes tend to last for around 2 weeks. Episodes often recur.

The most effective treatment is the Epley manoeuvre. This procedure involves a set series of movements of the patient's trunk and head and is designed to encourage the debris to move out of the semicircular canal.

A single manoeuvre leads to complete symptom resolution in 70-80% of patients, and many of the remaining patients will respond to a second or third manoeuvre.

Question 188 of 189

The patient below is being treated for epilepsy:



What is the most likely underlying diagnosis?

A. HIV
B. Neurofibromatosis
C. Arteriovenous malformation
D. Tuberous sclerosis
E. Lennox-Gastaut syndrome

D. Tuberous sclerosis

Tuberous sclerosis - adenoma sebaceum

The correct answer is Tuberous sclerosis. Tuberous sclerosis is a genetic disorder that causes noncancerous (benign) tumours to grow in different parts of the body. It often affects the brain and can lead to conditions such as epilepsy. The image shows characteristic adenoma sebaceum on the nose.

HIV is not the most likely underlying diagnosis in this case. While HIV can have neurological manifestations due to direct effects of the virus or secondary to opportunistic infections, it does not typically present with benign brain tumours. Neurological complications of HIV usually include cognitive impairment, myelopathy, neuropathy, and potentially opportunistic infections of the CNS like toxoplasmosis or progressive multifocal leukoencephalopathy (PML).

The next option, Neurofibromatosis, is a genetic disorder causing tumours to form on nerve tissue. These tumours can develop anywhere in your nervous system, including your brain and spinal cord. However, these tumours are typically neurofibromas and would have a different appearance on imaging compared to those seen in tuberous sclerosis.

Arteriovenous malformation (AVM) is an abnormal connection between arteries and veins, bypassing the capillary system. This vascular disorder disrupts normal blood flow and oxygen circulation. Brain AVMs could possibly cause seizures but they would create a different pattern on imaging studies than what is presented here.

Finally, Lennox-Gastaut syndrome is a type of childhood-onset epilepsy that results in multiple seizure types and cognitive decline. However, it does not cause benign brain tumours as seen in the provided image.

In conclusion, based on UK guidelines for managing patients with epilepsy and considering the characteristics shown in the image provided - benign brain tumours - Tuberous Sclerosis represents the most likely underlying diagnosis for this patient's condition.

Question 189 of 189

A 35-year-old lady was referred to the neurology clinic for investigation of facial weakness. Over the past 2 months, she noticed that her eyelids had tended to drop towards the end of the day, and she had occasional diplopia. She also felt that the corners of her mouth drooped a bit, and she reported some difficulty smiling. There was no limb weakness, and she had not had any difficulty swallowing.

On examination, there was a bilateral facial droop, and bilateral partial ptosis. She was sitting with her head tilted up to compensate for this. She had almost complete ptosis after trying to keep her eyes in elevation for more than 15 seconds. Eye movements were otherwise normal. Palatal movement was equal on both sides, and there were no abnormalities in tongue movements. Tone, power, reflexes and sensation were normal in the upper and lower limbs.

What is the most appropriate initial management?

A. Mycophenolate mofetil
B. Thymectomy
C. Prednisolone
D. Intravenous immunoglobulin
E. Pyridostigmine

E. Pyridostigmine

Pyridostigmine is a long-acting acetylcholinesterase inhibitor that reduces the breakdown of acetylcholine in the neuromuscular junction, temporarily improving symptoms of myasthenia gravis

This lady presents with weakness affecting her facial and extraocular muscles, demonstrating fatigability. This is characteristic of myasthenia gravis. Treatment choice depends on the severity of symptoms.

In mild disease such as in this case, acetylcholinesterase inhibitors such as pyridostigmine are useful in control of symptoms. In more severe disease, with limb weakness or bulbar dysfunction immunomodulatory agents are often required. Steroids are often employed, with the addition of steroid-sparing agents such as mycophenolate mofetil, ciclosporin or azathioprine if necessary.Intravenous immunoglobulin and plasma exchange are useful in myasthenic crises. Thymectomy improves symptoms in cases associated with thymoma, and may also be beneficial in young patients with recent onset of symptoms, but would not be used as an initial treatment option.

PASSMEDICINE
RESPIRATORY

INTERNAL MEDICINE

EDITED BY

DR. JAMIL ALRAJHI

2024

773-555-493
jameelalrajhi@gmail.com

PASSMEDICINE PART 2 RESPIRATORY 2024

Question 1 of 231

A 60-year-old male is admitted to hospital under the medical team with breathlessness. The patient is able to give a history of rapid onset breathlessness this afternoon, with no associated chest pain. He has recently started an an ACE inhibitor for hypertension. On examination, there are bilateral crepitations to the mid-zones. There are no added heart sounds. The patient is tachypnoeic and saturations are 90% on room air. Urinalysis is negative. A chest X-Ray demonstrates acute pulmonary oedema. An ECG shows sinus rhythm with a rate of 95. Blood results show:

Na +	135 mmol/l
K +	5.1 mmol/l
Urea	12.6 mmol/l
Creatinine	188 µmol/l

Which investigation will confirm the most likely underlying diagnosis?

A. Magnetic Resonance (MR) Renal Angiography
B. Computed Tomography (CT) Pulmonary Angiography
C. Coronary Angiography
D. Echocardiography
E. High Resolution Computed Tomography (HRCT)

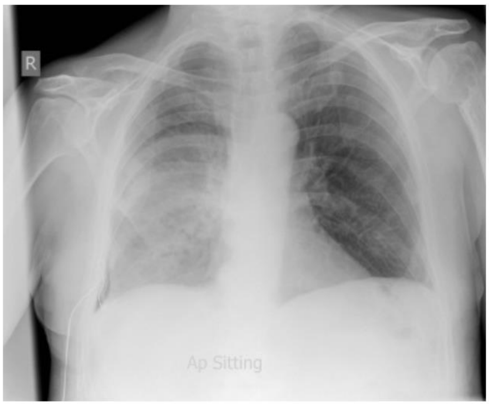
A. Magnetic Resonance (MR) Renal Angiography

This patient gives a history of flash pulmonary oedema. The clue in the question as to the cause is the recent initiation of an ACE inhibitor. In patients with undiagnosed renal artery stenosis, initiation of ACE inhibitor therapy is a recognised cause of flash pulmonary oedema.

Although a echocardiography would be part of the initial work-up, it would not reveal the underlying diagnosis. From the history, and based on the unremarkable ECG, cardiogenic pulmonary oedema is unlikely to be the cause in this scenario.

Question 2 of 231

A 76-year-old man presents with dyspnoea. A chest x-ray is performed:



What is the main finding on the film?

A. Right upper lobe consolidation
B. Right sided pneumothorax
C. Pulmonary oedema
D. Right middle + lower lobe consolidation
E. Bronchiectasis

D. Right middle + lower lobe consolidation

The presence of consolidation above the horizontal fissure and the obscured right heart border suggest the presence of right middle lobe consolidation. There is some loss of definition (silhouette sign) of the medial aspect of the right hemidiaphragm suggesting some right lower lobe consolidation as well.

A chest drain is also seen on the right hand side. This had been inserted to drain an associated pleural effusion.

Question 3 of 231

A 75-year-old male presents to his GP with three weeks of shortness of breath. He developed a cold a few weeks prior and has remained short of breath since. He was bringing up green sputum but the cough is now dry. He has no pains in his chest and no fevers. He is now retired but remains a keen gardener with an allotment of vegetables and tubers. He previously worked as an aerospace engineer making and fixing electronic parts. He is unsure of any asbestos exposure.

On examination, he has clubbing and appears cachectic. There are bilateral apical crepitations and he appears breathless with activity. Oxygen saturation on air is 94% and respiratory rate is 28/min.

Na +	138 mmol/l
K +	4.2 mmol/l
Urea	4.3 mmol/l
Creatinine	78 µmol/l
Hb	135 g/l
Platelets	450 * 10 /l9
WBC	14 * 10 /l9
C-reactive protein	12 mmol/l
Chest x-ray	reticular shadowing prominent at the apices

What is the likeliest diagnosis?

A. Aspergillosis
B. Asbestosis

C. Berylliosis
D. Hypersensitivity pneumonitis
E. Idiopathic pulmonary fibrosis

C. Berylliosis

Aeronautical engineers -> risk of berylliosis

This gentleman has shortness of breath and reticular changes consistent with a form of interstitial lung disease (formerly pulmonary fibrosis). The cause is reliant on the history. Asbestosis is usually a lower lobe fibrosing process. Aspergillosis typically occurs in weakened immunosuppressed individuals. Hypersensitivity pneumonitis classically occurs in those exposed to moulds and hay which is possible in a gardener but not common. Berylliosis is linked to beryllium heavily used in the aeronautical industry.

Question 4 of 231

A 35-year-old female banker from Scotland presents with breathlessness on exertion. She is a non smoker and has two budgies at home. She had severe pneumonia 5 years ago and required intubation on intensive care for 5 days. She recovered but has reported ongoing dyspnoea and on examination has an inspiratory wheeze. This has not improved despite her GP starting inhaled steroids for asthma. Her chest X-ray was unremarkable, peak flow was reduced and eosinophil count was normal.

What is the most likely diagnosis?

A. Cystic fibrosis
B. Severe asthma
C. Posterior subglottic stenosis
D. Chronic obstructive pulmonary disease
E. Allergic bronchopulmonary aspergillosis

C. Posterior subglottic stenosis

Subglottic stenosis can occur from birth, idiopathically and in patients who have been intubated. It worth considering in patient with lung function tests that show an obstructive picture and as a differential diagnosis in the 'difficult asthma clinic'.

This patients lack of response to inhaled steroids and predominance of inspiratory rather expiratory wheeze may asthma less likely. A patient with cystic fibrosis would probably have an abnormal CXR by the age of 35 and the normal eosinophil count makes ABPA less likely. COPD is unusual in non smokers but plausible if their is a family history of early onset dyspnoea (ie alpha- 1-antitrypsin deficiency).

Question 5 of 231

A 64-year-old man presents to the respiratory clinic for review. He has been diagnosed with idiopathic pulmonary fibrosis which has not responded to a course of oral corticosteroids. He is finding it difficult to manage the stairs more than once per day and is finding it difficult to get out of the house. On examination his blood pressure is 138/88 mmHg, his pulse is 80 beats per minute and regular. There are inspiratory crackles on auscultation of the chest consistent with pulmonary fibrosis, and his O2 saturation is 92% at rest. His FVC is 65% of predicted.

Which of the following is the next most appropriate intervention?

A. Cyclophosphamide
B. Infliximab
C. Methotrexate
D. Nintedanib
E. Rituximab

D. Nintedanib

Nintedanib is a small molecule tyrosine kinase inhibitor including the receptors platelet-derived growth factor receptor (PDGFR) and , fibroblast growth factor receptor (FGFR) 1-3, and VEGFR 1-3. It is licensed for the treatment of idiopathic pulmonary fibrosis, and is endorsed by NICE guidelines when:

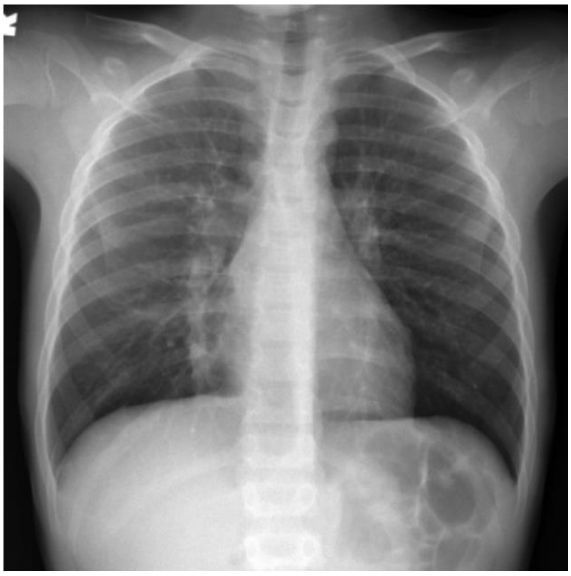
a. **The person has a forced vital capacity (FVC) between 50% and 80% of predicted**

b. **The company provides nintedanib with the discount agreed in the patient access scheme and**

c. **Treatment is stopped if the disease progresses (a confirmed decline in percent predicted FVC of 10% or more) in any 12month period.**

Question 6 of 231

A 9-year-old is referred to the Children's Ward with pyrexia and cough:



What does the x-ray show?

A. Right middle lobe consolidation
B. Perihilar lymphadenopathy
C. No abnormality
D. Left lingual consolidation
E. Right upper lobe consolidation

A. Right middle lobe consolidation

The correct answer is **Right middle lobe consolidation**. This is because the x-ray image shows an area of increased opacity in the right middle zone of the lung, which corresponds to the location of the right middle lobe. The loss of the right heart border is a common finding in right middle lobe consolidation. Consolidation refers to a region of lung tissue filled with liquid instead of air, often due to infection such as pneumonia. The child's symptoms of pyrexia and cough also support this diagnosis.

The option **Perihilar lymphadenopathy** is incorrect because it refers to enlargement of lymph nodes around the hilum, a region where blood vessels and nerves enter or exit an organ. In this case, it would be seen as enlarged shadows near the centre (mediastinum) of the chest x-ray. However, there are no such findings in this image.

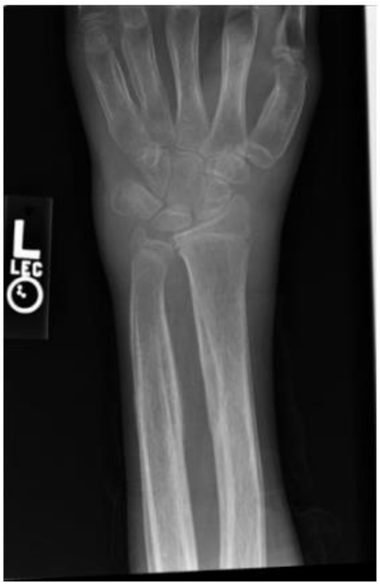
The option **No abnormality** is incorrect as there is clearly an area of increased opacity in the right middle lobe indicating a pathological process.

Left lingual consolidation is also incorrect. The lingula is a part of the left upper lobe that corresponds anatomically to the right middle lobe. However, there are no signs on this x-ray that suggest any abnormalities in this area.

Lastly, **Right upper lobe consolidation** would typically show increased opacity in the upper part of the right lung field on an x-ray image. However, in this case, it's clear from the image that there's an increase in density more centrally located which corresponds with a right middle lobe consolidation rather than upper.

Question 7 of 231

A 75-year-old man is admitted to hospital with haemoptysis and dyspnoea. He is a life-long smoker and a coin lesion is seen in the right middle zone on chest x-ray. He also complains of pain in his left forearm. An x-ray is requested:



What is the most likely cause of the patients arm pain?

A. PTHrP-induced subperiosteal bone resorption and osteopaenia
B. Pathological fracture of the radial head
C. Long-standing osteoarthritic changes
D. Hypertrophic pulmonary osteoarthropathy
E. Metastatic lung cancer

D. Hypertrophic pulmonary osteoarthropathy

The x-ray shows the periosteal reaction on the shafts of the ulnar and radius. This is typical of hypertrophic pulmonary osteoarthropathy.

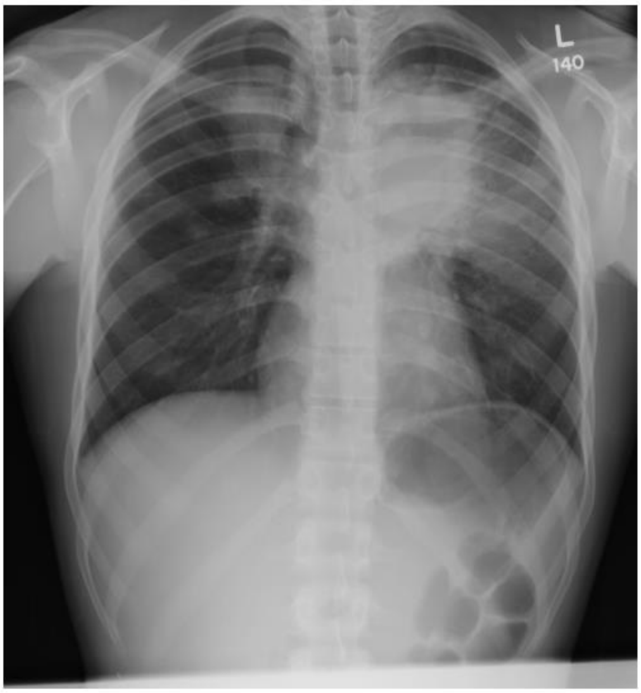
Question 8 of 231

A 23-year-old man presents with dyspnoea and haemoptysis to the Emergency Department. He initially thought his symptoms were due to a chest infection but a course of amoxicillin from his GP has not helped. He now feels short of breath on mild exertion and has coughed up fresh blood on a number of occasions today.

On examination his oxygen saturations are 92% on air. On examination he has crackles predominately in the upper and lower zones of the left lung. Temperature is 37.7ºC.

Hb	13.1 g/dL
WCC	11.3 *10^9/l
Sodium	137 mmol/l
Potassium	4.2 mmol/l
Urea	6.4 mmol/l
Creatinine	123 µmol/l
CRP	188 mg/L

Urine dip: blood +++, leucocytes + A chest x-ray is requested:



Later tests during his admission show the following;

c-ANCA	Positive
p-ANCA	Positive
Anti-GBM	Negative

What is the most likely diagnosis?

A. Granulomatosis with polyangiitis
B. Cryptogenic organizing pneumonia

C. Churg-Strauss syndrome
D. Tuberculosis
E. Goodpasture's disease

A. Granulomatosis with polyangiitis

This patient has both the pulmonary and renal manifestations (raised creatinine, heamaturia) of granulomatosis with polyangiitis.

Question 9 of 231

A 67-year-old gentleman comes for review following a two-week-wait referral to the respiratory clinic. He is a current smoker with a 60 pack-year history. He has been suffering from a worsening cough and haemoptysis for the last month and has had a weight loss of 3kg over one month. On examination he looks cachectic, his fingers are clubbed and tar-stained. His wrists are tender. His chest shows reduced expansion on the right side with reduced breath sounds and dullness.

What is the wrist tenderness likely to indicate?

A. Osteoarthritis
B. Osteoporosis
C. Osteopenia
D. Paraneoplastic rheumatoid-like disease
E. Hypertrophic pulmonary osteoarthropathy

E. Hypertrophic pulmonary osteoarthropathy

The correct answer is hypertrophic pulmonary osteoarthropathy. This is a patient who is very likely to have lung cancer. He has a heavy smoking history, haemoptysis and weight loss with worrying examination features. Wrist tenderness is likely to be secondary to this in the form of a paraneoplastic hypertrophic pulmonary osteoarthropathy due to squamous cell lung cancer. This is suggested by the clubbing, caused in a similar way. Wrist tenderness is unlikely to occur with osteoarthritis, osteopenia and osteoporosis. Paraneoplastic rheumatoid-like disease is more likely to be asymmetrical and affect the lower limbs.

Question 10 of 231

A 44-year-old man attends with a one-week history of fever and cough. A systematic enquiry was otherwise unremarkable. On examination, you note right-sided coarse crepitations. The examination was otherwise unremarkable.

Blood results are as follows:

Hb	115 g/L	Male: (135-180)Female: (115 - 160)
Platelets	524 * 10 /L 9	(150 - 400)
WBC	18.2 * 10 /L 9	(4.0 - 11.0)
Neuts	14.8 * 10 /L 9	(2.0 - 7.0)
Lymphs	3.0 * 10 /L 9	(1.0 - 3.5)
Mono	0.2 * 10 /L 9	(0.2 - 0.8)
Eosin	0.2 * 10 /L 9	(0.0 - 0.4)
Na +	132 mmol/L	(135 - 145)
K +	4.8 mmol/L	(3.5 - 5.0)
Urea	24.2 mmol/L	(2.0 - 7.0)
Creatinine	381 µmol/L	(55 - 120)
CRP	22 mg/L	(< 5)
ANCA PR3	8.2 IU/ml = Positive	(< 2)
Total serum IgE	246 KU/l	(5 - 120)
Aspergillus specific IgG or IgE	Positive	

A CT chest is performed:

CT chest	Hazy consolidation or ground-glass infiltrates in the right lower lobe lung parenchyma suggestive of an intra-alveolar haemorrhage

What is the most likely diagnosis?

A. Allergic bronchopulmonary aspergillosis (ABPA)
B. Churg-Strauss syndrome
C. Goodpasture's disease
D. Hypereosinophilic syndrome
E. Wegener's granulomatosis

E. Wegener's granulomatosis

Consider granulomatosis with polyangiitis when a patient presents with ENT, respiratory and kidney involvement

Wegener's granulomatosis is correct. This condition is characterised by necrotizing granulomatous inflammation of the upper and/or lower respiratory tract, and vasculitis of smallto-medium-sized vessels. The presence of a pulmonary haemorrhage and renal failure, combined with a strongly positive ANCA makes this the most likely diagnosis.

Allergic bronchopulmonary aspergillosis (ABPA) is incorrect. Although the patient has a positive aspergillus-specific antibody result and raised total serum IgE, the clinical features, in this case, do not support a diagnosis of ABPA. ABPA presents with shortness of breath, cough, and wheezing. As *Aspergillus fumigatus* spores are common in the environment and almost everyone is exposed to them, low levels of antibodies to *A. fumigatus* are found in many patients and are often not clinically significant.

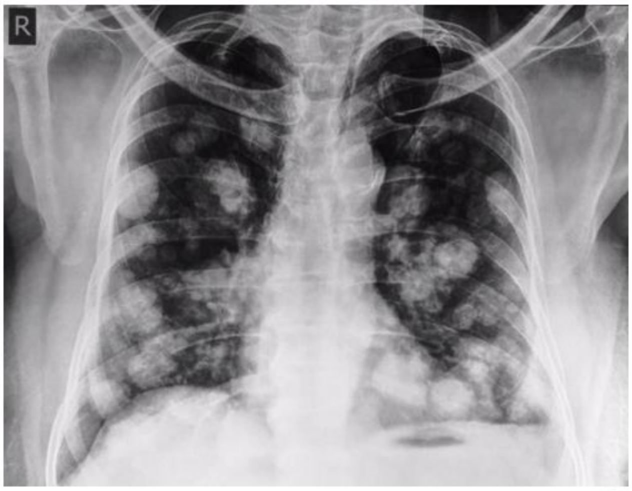
Churg-Strauss syndrome is incorrect. Churg-Strauss syndrome, also known as allergic granulomatous angiitis, is a rare disease characterised by the triad of asthma, pulmonary infiltrates, and hypereosinophilia. Although this remains within the differential diagnosis, the acute onset, presence of renal failure, and strongly positive ANCA with a PR3 subtype favours the diagnosis of Wegener's granulomatosis. Churg-Strauss syndrome is classically associated with the MPO subtype of ANCA.

Goodpasture's disease is incorrect. Pulmonary haemorrhage and renal failure are classical features of this condition. However, this condition is associated with anti-GBM antibodies rather than ANCA, making this a less likely diagnosis.

Hypereosinophilic syndrome is incorrect. Hypereosinophilic syndrome (HES) is a diagnosis of exclusion and should be made only after reactive and clonal eosinophilia have been excluded. The absence of eosinophilia makes this a less likely diagnosis.

Question 11 of 231

A 53-year-old man is admitted to the Emergency Department complaining of a 'chest infection'. His symptoms have not improved despite a course of antibiotics from his GP. The admission chest x-ray is shown below:



What is the most appropriate next-line investigation?

A. High resolution CT thorax
B. CT abdomen
C. Testicular ultrasound
D. Bronchoscopy
E. Sputum culture

B. CT abdomen

The chest x-ray shows cannonball metastases which are most commonly caused by renal cell cancer. A CT abdomen is the most appropriate test to investigate this possibility.

Other tests should of course be considered including a prostate specific antigen.

Question 12 of 231

A 52-year-old man presents with a 5 day history of cough, feeling hot and facial pains. He is generally fit and well although does currently take sertraline for anxiety and depression. He describes a cough productive of pale yellow sputum. He also describes difficulty breathing through his nose and pain in his face, particularly when coughing on leaning forward.

On examination he is alert, pulse rate is 84/min, temperature is 37.3º and respiratory rate is 16/min. His blood pressure is 122/74 mmHg. Chest auscultation is unremarkable. He is tender over the maxilla.

What is the most appropriate next step in management?

A. Check serum urea for CURB-65 scoring
B. Oral amoxicillin + review in 3-4 days if not improving
C. Advise paracetamol for symptoms + review in 3-4 days if not improving
D. Arrange a chest x-ray
E. Oral clarithromycin + review in 3-4 days if not improving

C. Advise paracetamol for symptoms + review in 3-4 days if not improving

This patient has a viral upper respiratory tract infection. His examination findings are essential normal other than some facial pain associated with rhinosinusitis. He requires supportive treatment only as per NICE guidelines.

Question 13 of 231

A 63-year-old man with a new diagnosis of idiopathic pulmonary fibrosis is reviewed is respiratory clinic following initial baseline investigations. He has been suffering from progressive shortness of breath for nine months with a non-productive cough. His GP has tried to give him inhalers and antibiotics but this has not helped. He has a past medical history of hypertension, depression and gout. He is concerned about his new diagnosis, especially about his prognosis.

What test is most useful in determining prognosis?

A. Forced vital capacity
B. 6-minute walking test
C. High resolution CT of the chest
D. Carbon monoxide transfer factor
E. FEV1/FVC ratio

D. Carbon monoxide transfer factor

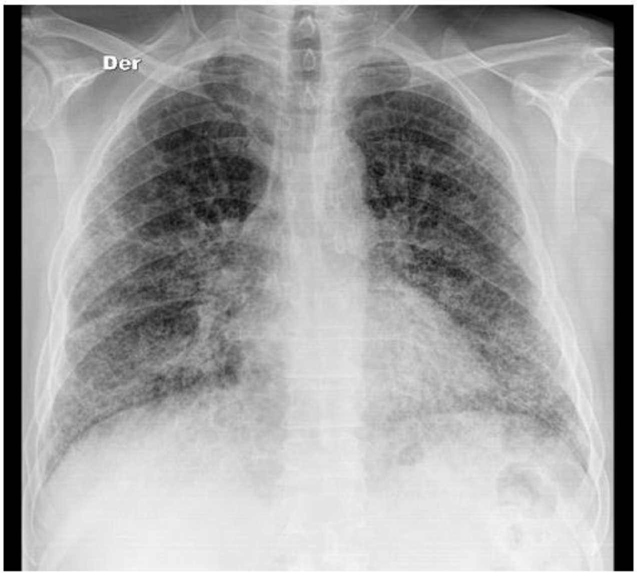
The correct answer is carbon monoxide transfer factor. The 6-minute walking test is not useful for prognosis, whilst CT is more useful in determining aetiology. The FEV1/FVC ratio would confirm a restrictive pattern consistent with fibrosis, but this would not help defining prognosis. Serial measurements of forced vital capacity can be helpful in understanding the disease course and establishing progression, but at the first appointment this is unlikely to be helpful.

Question 14 of 231

A 63-year-old retired car mechanic is referred to the respiratory clinic by his GP due to progressive dyspnoea and low oxygen saturations. The GP had performed an ECG and BNP level, both of which were within normal limits.

The dyspnoea had been getting progressively worse for the past 2 years and is now limiting his daily activities. He also describes a dry cough which is worse after walking. There is no history of chest pain, On examination there is no clubbing, pulse is 84/min, respiratory rate 18/min and oxygen saturations are 91% on room air.

An x-ray is requested:



What is the most likely diagnosis

A. Sarcoidosis
B. Extrinsic allergic alveolitis
C. Silicosis
D. Bronchiectasis
E. Idiopathic pulmonary fibrosis

E. Idiopathic pulmonary fibrosis

The chest x-ray shows reitcular opacities predominantly in the bases, a classic finding of idiopathic pulmonary fibrosis.

His occupation is not a risk factor for silicosis. This condition also most commonly affects the upper zones of the lungs.

There is no hilar lymphadenopathy to support a diagnosis of sarcoidosis.

Whilst there is some traction bronchiectasis of the lungs (seen most easily on the associated CT below) this is not the primary diagnosis and the history is not supportive.

With extrinsic allergic alveolitis there is often a relevant occupational history and the upper/mid zones are more commonly affected.

Question 15 of 231

An 81-year-old man is seen in the respiratory clinic due to poor control of his chronic obstructive pulmonary disease (COPD). Despite being prescribed inhaled salbutamol as required, he remains breathless on minimal exertion which is limiting his ability to carry out daily activities. He reports that these symptoms are consistently present with no diurnal variation.

He has a 50 pack year smoking history. His past medical history includes hypertension for which he takes ramipril and he has no allergies. Recent blood tests performed by the general practitioner show the following:

Hb	141 g/L	Male: (135-180)Female: (115 - 160)
Platelets	301 * 10 /L 9	(150 - 400)
WBC	6.3 * 10 /L 9	(4.0 - 11.0)
Neuts	4.1 * 10 /L 9	(2.0 - 7.0)
Lymphs	1.7 * 10 /L 9	(1.0 - 3.5)
Mono	0.4 * 10 /L 9	(0.2 - 0.8)
Eosin	0.1 * 10 /L 9	(0.0 - 0.4)

Which of the following would be most appropriate to initiate?

A. Add a leukotriene receptor antagonist
B. Add a long acting beta agonist + muscarinic antagonist (LABA + LAMA)
C. Add an inhaled corticosteroid (ICS)
D. Add prophylactic antibiotics
E. Assessment for long term oxygen therapy

B. Add a long acting beta agonist + muscarinic antagonist (LABA + LAMA)

COPD - still breathless despite using SABA/SAMA and **no** asthma/steroid responsive features → add a LABA + LAMA

This man has presented with COPD that has not been adequately controlled by a SABA/SAMA. The next step in management depends on whether he shows asthma/steroid-responsive features. A lack of any history of asthma/atopy, variation in symptoms (including diurnal), and a normal eosinophil count indicates that this man is unlikely to be steroid responsive. Therefore the most appropriate second-line treatment is to add a LABA + LAMA.

Leukotriene receptor antagonists are not used second line in COPD.

ICS is indicated second line if there are any features suggestive of asthma/steroid responsiveness. As these are not present in the history or blood results, a LABA + LAMA is more appropriate.

Prophylactic antibiotics could be considered in cases of recurrent infective exacerbation however this is not the case here.

This man is not yet on optimal medical therapy so an assessment for long-term oxygen therapy would be premature at this stage.

Question 16 of 231

A 52-year-old woman presents with worsening exercise tolerance. Her GP has recently increased her asthma treatment but this has not helped, nor does she have any relief from her salbutamol inhaler. She has no other known medical history. On examination, her lung fields sound clear on auscultation but she has an early diastolic murmur. She has already had a chest X-ray demonstrating enlarged proximal pulmonary arteries.

Which investigation is most likely to provide the correct diagnosis?

A. ECG
B. Renal function blood tests
C. Echocardiogram
D. High-resolution CT chest
E. Right heart catheterisation

E. Right heart catheterisation

The correct answer is right heart catheterisation to diagnose pulmonary arterial hypertension. The history is primarily of worsening shortness of breath in a middle-aged person with clear lung fields and a diastolic murmur. The chest X-ray with enlarged proximal pulmonary arteries is highly suggestive. Whilst an ECG or echocardiogram may show features of pulmonary arterial hypertension right heart catheterisation is the gold standard and would be most diagnostic.

Question 17 of 231

A 69-year-old man with a history of rheumatoid arthritis well controlled on methotrexate presents with gradual pain and swelling in his wrists and ankles.

The pain is described as a dull ache that is intermittent, often worse in the evenings. There is associated swelling which can sometimes feel warm. He has tried regular paracetamol but this has had limited effect.

He also describes a chronic cough and shortness of breath on exertion for the past 6 months that he has not mentioned to his GP. There has been no haemoptysis and he denies any fevers. His wife has noticed that he has been losing weight recently.

He has a past medical history of rheumatoid arthritis, hypertension, hypercholesterolaemia and type 2 diabetes mellitus. He currently takes methotrexate weekly, folic acid 5mg weekly, ramipril 5mg, simvastatin 20mg at night, metformin 500mg three times a day and paracetamol 1g four times daily.

He is a retired accountant and a current smoker with a 50 pack year smoking history. He drinks approximately 30 units of beer a week. He denies any recent foreign travel.

On examination, he is cachectic and short of breath on exertion. His pulse is 80/min and regular, blood pressure 140/93 mmHg, oxygen saturations of 93% on air. He has marked fingernail clubbing. Examination of his wrists reveals slightly swollen and tender joints. Swan neck and ulnar deviation deformities are noted in both hands. Other than his wrists, no other joint abnormalities are detected. Examination of the peripheral nervous system is normal.

Examination of his chest is normal with no focal consolidation.

Initial bloods are as follows:

Na+	134 mmol/L
K+	3.9 mmol/L
Urea	7.8 mmol/L
Creatinine	105 µmol/L
Hb	100 g/L
WBC	6.0x10^9/L
Platelets	200x10^9/L
LFTs	Normal
Serum uric acid	410 µmol/L
CRP	12 mg/L

Chest X-ray reveals a discrete opacification at the periphery of the right middle lobe.

What is the most likely cause of his joint pains?

A. Rheumatoid arthritis flare
B. Gout
C. Hypertrophic pulmonary osteoarthropathy secondary to bronchogenic carcinoma
D. Reiter's syndrome
E. Osteoarthritis

C. Hypertrophic pulmonary osteoarthropathy secondary to bronchogenic carcinoma

This man has a history of intermittent swelling and pain in his wrists and to a lesser degree in his ankles. He has a past medical history of rheumatoid arthritis with some evidence of joint deformities but seems to be well controlled on methotrexate. There is no synovitis present in the typical pattern of rheumatoid arthritis.

He has a strong smoking history, a 6-month history of cough, weight loss, is clubbed and has an abnormal chest x-ray all highly suspicious of an underlying lung malignancy. Hypertrophic pulmonary osteoarthropathy occurs in about 5% of case of lung cancer and typically presents with intermittent aching and swelling in the wrists and ankles.

This man's serum uric acid is normal and gout typically presents in the first metatarsophalangeal joint. He has no previous history of gout but does have some risk factors for this.

Osteoarthritis is a possible diagnosis but does not typically present with signs of synovitis.

Reiter's syndrome is a seronegative arthritis characterised by the triad of urethritis, conjunctivitis and arthritis which doesn't fit with this patient's clinical picture.

Question 18 of 231

A 57-year-old male is seen in the respiratory clinic with a three months history of a productive cough, weight loss and drenching night sweats. He reports that he is producing approximately 1 cup of green sputum each day and has lost 4 kgs during the same time period. He has smoked 20 cigarettes a day since he was 15 years old. He last traveled abroad 2 years ago, visiting family in North America.

The GP has performed routine blood tests which are unremarkable and a HIV test is negative. Several sputum samples have been sent for routine microscopy, culture & sensitivities (MC&S) growing only normal respiratory flora. Following a normal chest X-ray, you perform a high resolution CT (HRCT) scan. This demonstrates right middle lobe and left lower lobe bronchiectasis with multiple nodules in both lungs.

What other initial investigation would you perform to help reach a diagnosis?

A. Sweat test
B. PET-CT Scan
C. Repeat HRCT scan in 3-6 months
D. Sputum for acid fast bacilli (AFB)
E. Bronchoscopy +/- biopsies

D. Sputum for acid fast bacilli (AFB)

The correct answer is to send sputum for acid fast bacilli (AFB).

Whilst the causative organism could potentially be either tuberculosis or non-tuberculosis mycobacterial (NTM) infection, the latter is the more appropriate here given the radiological findings on HRCT, which are classically seen in atypical mycobacterial infection. This question tests your understanding of the common presentations of pulmonary non-tuberculous mycobacterium (NTM) infection in immunocompetent individuals. NTM infection in the non-HIV population is becoming increasingly common and is often a challenging diagnosis to make due to the environmental nature of NTM.

Question 19 of 231

A 24-year-old woman with a history of cystic fibrosis comes to the endocrinology clinic for review. She has been losing weight and a recent fasting plasma glucose has been measured at 8.1 mmol/l. Over the past year there has been three admissions to hospital with exacerbations of her CF. On examination her blood pressure is 122/81 mmHg, pulse is 71 beats per minute and regular. Auscultation of the chest reveals coarse crackles and scattered wheeze. Abdomen is soft and on tender, her body mass index is 19.5 kg/m².

Which of the following is the best way to manage her diabetes?

A. Low calorie diet
B. Insulin and high calorie diet
C. Metformin and low calorie diet
D. Gliclazide and standard calorie diet
E. Dapagliflozin and low calorie diet

B. Insulin and high calorie diet

Patients with cystic fibrosis suffer significant difficulties in maintaining their weight even before or if they never develop diabetes. Weight loss in cystic fibrosis is closely allied to both risk of exacerbations of the disease and an increase in overall mortality. For this reason it's important for patients to maintain a high calorie diet and take insulin to allow them to convert those calories into stored energy.

All of the other options which include either a standard or low calorie diet are incorrect because they won't allow the patient to maintain her body weight. Dapagliflozin promotes spilling of carbohydrate into the urine, and hence leads to a calorie deficit, this is therefore incorrect. Gliclazide can be used in the initial period of diabetes associated with cystic fibrosis, although it's important in this situation to also encourage patients to maintain a high calorie intake.

Question 20 of 231

A 73-year-old man is referred to the respiratory outpatient clinic with exertional shortness of breath. He has no past medical history and does not take any regular medications. He does not smoke or drink alcohol. He worked for 51 years at a paper mill.

On examination, he is mildly dyspnoeic at rest. There are bibasal inspiratory crackles heard on auscultation. The fingers are clubbed. Cardiovascular examination is unremarkable.

A high-resolution CT chest demonstrates parenchymal bands, traction bronchiectasis and honeycomb lung. There is no involvement of the pleura.

Given the likely diagnosis, what treatment is likely to be recommended?

A. Chemotherapy
B. Conservative management
C. Prednisolone
D. Radiotherapy
E. Surgery

B. Conservative management

Asbestosis is treated conservatively - no interventions offer a significant benefit

Conservative management is the correct answer. The patient presents with exertional shortness of breath and crackles and undergoes an HRCT chest, which demonstrates classic changes of fibrotic lung disease (parenchymal bands, honeycombing and traction bronchiectasis). The most likely cause of fibrosis is asbestos exposure. Historical work in a paper mill is a significant occupational risk for asbestos exposure.

Chemotherapy is incorrect. Asbestos exposure puts the patient at risk of developing mesothelioma, which is a malignant cancer of the pleura. Chemotherapy is an option in this condition. However, we are specifically told there is no pleural involvement and therefore this is unlikely.

Prednisolone is incorrect. This is often trialled in interstitial lung disease if the condition is felt to be steroid-responsive. However, asbestosis is not felt to respond to medical treatment in this way. Additionally, HRCT appearances of ground glass (suggesting inflammation) rather than honeycombing (suggesting established fibrosis) tend to be more steroid responsive.

Radiotherapy is incorrect. This may be an option for treating lung cancer or mesothelioma. However, the HRCT chest does not describe pleural involvement or a primary lung malignancy and therefore this is not likely to be recommended.

Surgery is incorrect. Occupational exposure to asbestosis has been associated with lung cancer, which in some cases can be treated surgically. However, the description, in this case, is of pulmonary fibrosis, rather than a malignancy.

Question 21 of 231

A 24-year-old woman with cystic fibrosis (CF) is reviewed in the endocrinology clinic. She has lost weight over the past few months despite maintaining a high-calorie diet and wakes three times per night to pass urine. Over the past year she has suffered two exacerbations of her CF and has seen a more rapid deterioration in her lung function. On examination her blood pressure is 122/82 mmHg, her pulse is 82 beats per minute and regular, and her body mass index is 19 kg/m². There are coarse crackles and wheeze on auscultation of the chest. A fasting blood glucose is 8.1 mmol/l, and her HbA1c is 60 mmol/mol.

Which of the following is the most appropriate way to manage her diabetes mellitus?

A. Switch to a low calorie diet
B. Start metformin monotherapy and continue high calorie diet
C. Start gliclazide monotherapy
D. Start sitagliptin monotherapy
E. Start basal bolus insulin and continue high calorie diet

E. Start basal bolus insulin and continue high calorie diet

Patients with cystic fibrosis and diabetes should follow a high calorie diet and use extra insulin to control their blood sugar

It's clear that the development of diabetes in patients with cystic fibrosis is associated with weight loss and the development of more frequent respiratory exacerbations. As such it's crucial to maintain adequate nutrition with a high calorie diet and adequate insulin to control blood glucose levels. This is at odds with management of diabetes in other conditions.

Gliclazide is of value in early diabetes associated with cystic fibrosis, although it rarely controls blood glucose levels adequately to allow patients to maintain a high calorie diet, hence insulin is preferred

. Insulin resistance isn't the primary problem in CF related diabetes, therefore metformin is of little value, and DPPIV inhibitors are less effective in lowering blood glucose versus sulphonylureas in this situation.

Question 22 of 231

A 54-year-old man presents to the respiratory clinic. He was found to have three pleural plaques on a chest X-ray when being screened for tuberculosis due to immigration purposes. He worked as a builder for four years 21 years ago. He has no shortness of breath or chest pain. A diagnosis of benign pleural disease is made. He is keen to seek compensation.

What is the most appropriate advice?

A. Seek compensation by criminal law suit
B. Seek compensation by civil law suit
C. Seek compensation via GP
D. Seek compensation via department of social security
E. Unlikely to receive compensation currently

E. Unlikely to receive compensation currently

The correct answer is that he is unlikely to receive compensation currently. This is a gentleman who been identified to have pleural plaques, likely secondary to asbestos exposure but who is suffering no symptoms. These patients do not qualify for compensation. If a diagnosis of asbestosis or mesothelioma is made then the patient would qualify. Compensation can be obtained through Industrial Injuries Disablement Benefit from the Department of Social Security or a civil law claim for damages from the employer under which the exposure occurred. Patients should be advised to consult their local Department of Work and Pensions benefit agency for advice

Question 23 of 231

A 61-year-old man with known lung cancer presents to the emergency department with shortness of breath. He was diagnosed with squamous cell lung cancer two years ago and has had several courses of radiotherapy but stopped as he was not tolerating the treatment. He has noticed worsening of his chronic shortness of breath and struggled to walk from his car to the hospital, a distance that he was managing well previously. On examination, he appears slightly pale, has dullness on right side of his chest with a few bronchial breaths. A chest X-ray demonstrates an effusion and an aspirate withdraws 40ml of blood. His observations show hypoxia with saturations at 91% but are otherwise normal, and blood tests, including a crossmatch, is requested.

What is the most appropriate management?.

A. Surgical chest drain
B. Seldinger chest drain
C. Repeat aspiration
D. Conservative management only
E. IV antibiotics

A. Surgical chest drain

The correct answer is a surgical chest drain. The key in this question is that blood, rather than blood-stained fluid, is aspirated. This means that a surgical chest drain rather than a narrow bore chest drain, a Seldinger chest drain, will be needed. Repeating the aspiration is unlikely to gain any further information, and would only be useful if it is suspected that the sample was due to aspiration of an artery. However, it is unlikely that 40ml could have been aspirated from an artery. There is no indication for antibiotics and treatment is clearly needed as the patient is hypoxic.

The correct answer is a surgical chest drain. The key in this question is that blood, rather than blood-stained fluid, is aspirated. This means that a surgical chest drain rather than a narrow bore chest drain, a Seldinger chest drain, will be needed. Repeating the aspiration is unlikely to gain any further information, and would only be useful if it is suspected that the sample was due to aspiration of an artery. However, it is unlikely that 40ml could have been aspirated from an artery. There is no indication for antibiotics and treatment is clearly needed as the patient is hypoxic.

Question 24 of 231

A 45-year-old man presents to the emergency department with reports of 8 days of fever, headache, epistaxis, diarrhoea and cough. He has no past medical history. He drinks 32 units of alcohol a week. He keeps parrots and enjoys open water swimming in rivers.

Observations:

Heart rate 88 beats per minute Blood pressure 115/74 mmHg Respiratory rate 21/minute Oxygen saturations 94% on room air

On examination, there is splenomegaly. Chest auscultation reveals crackles in both lungs. Blood tests:

Hb	136 g/L	Male: (135-180)Female: (115 - 160)
Platelets	189 * 10 /L 9	(150 - 400)
WBC	2.9 * 10 /L 9	(4.0 - 11.0)

Na +	136 mmol/L	(135 - 145)
K +	4.2 mmol/L	(3.5 - 5.0)
Urea	4.2 mmol/L	(2.0 - 7.0)
Creatinine	88 µmol/L	(55 - 120)
CRP	52 mg/L	(< 5)
Bilirubin	12 µmol/L	(3 - 17)
ALP	88 u/L	(30 - 100)
ALT	32 u/L	(3 - 40)

Chest radiography reveals bilateral patchy airspace opacification.

Which organism is most likely responsible for his illness?

A. Chlamydia psittaci
B. Klebsiella pneumoniae
C. Legionella pneumophilia
D. Leptospira alexanderi
E. Staphylococus aureus

A. Chlamydia psittaci

Chlamydia psittaci is a cause of atypical pneumonia. It is transmitted via aerosol inhalation of faecal and urinary products on bird feathers

Chlamydia psittaci is the correct answer. This organism can cause Psittacosis, which is a cause of atypical pneumonia. The prodrome can involve diarrhoea, epistaxis and high fevers. Splenomegaly and a low white blood cell count are common. This organism should be suspected in cases of respiratory infection together with splenomegaly and epistaxis, which is rare as a constellation of symptoms. The classic zoonotic exposure for this condition is birds (including parrots).

Klebsiella pneumoniae is incorrect. This is common in those people who drink to excess, as this patient does. However, it tends to cause cavitating pneumonia and is not associated with epistaxis.

Legionella pneumophilia is incorrect. This can cause atypical pneumonia and diarrhoea. However, the white cell count will be elevated and it tends not to cause epistaxis and splenomegaly is less common. Sodium may be low and liver function may be deranged. Patients are typically exposed to poorly ventilated air conditioning systems.

Leptospira alexanderi is incorrect. This causes Leptospirosis. This is typically characterized by jaundice, subconjunctival haemorrhages and deranged liver function. While freshwater swimming may expose the patient to this organism, it does not cause atypical pneumonia.

Staphylococcus aureus is incorrect. This causes typical cavitating pneumonia. Often this occurs as a secondary bacterial infection after an episode of influenza.

Question 25 of 231

A 26-year-old man is reviewed in the asthma clinic. Despite therapy with high dose fluticasone / salmeterol montelukast and oral theophylline, he has suffered a 4th exacerbation in the past year and is now unable to reduce his dose of oral prednisone below 10mg without a significant increase in shortness of breath and wheeze. On examination his blood pressure is 123/80 mmHg, his pulse is 80 beats per minute and regular. Auscultation of the chest reveals quiet wheeze. His peak flow is 429 ml/min, (550 predicted). Eosinophils are elevated at 3.9 x 10(9)/l. IgE is within the normal range.

Which of the following is the most appropriate next intervention?

A. Mycophenolate
B. Mepolizumab
C. Infliximab
D. Tiotropium
E. Regular nebulisers

B. Mepolizumab

Mepolizumab may be used for asthma with high eosinophils

Mepolizumab is an anti-IL5 monoclonal antibody and represents the optimal intervention here. It binds to IL5 and prevents it from binding to its receptor. IL5 is a cytokine which promotes growth in eosinophil numbers and activity. By blocking IL5 mepolizumab down-regulates eosinophil activity and has been shown to significantly improve symptoms in resistant asthma. Omalizumab is an anti-IgE monoclonal antibody, it is effective in treating resistant asthma with evidence of raised IgE and allergic symptoms and is often considered for similar patients to those treated with mepolizumab, although eosinophil count and IgE drive which of the two therapies are used.

Mycophenolate is not utilised as a disease modifying agent in resistant asthma, but is primarily used for the treatment of lupus nephritis and as an anti-rejection agent. Infliximab is an anti-TNF monoclonal antibody used in the treatment of inflammatory bowel disease and arthritides. Tiotropium is primarily used in the treatment of COPD, and is less effective than other options listed in the treatment of resistant asthma. Nebulisers only have a role in treating asthma where patients are unable to administer beta agonist therapy using a conventional inhaler and spacer device.

Question 26 of 231

A 64-year-old is referred to the respiratory clinic by his general practitioner. He has had four exacerbations of chronic obstructive pulmonary disease in the last 12 months, having previously been well controlled. His forced expiratory volume in one second (FEV1) is 44% of the predicted normal. His inhaled medications include a long-acting muscarinic antagonist, a long-acting beta-2 agonist and an inhaled corticosteroid. He quit smoking five years previously. His six-minute walk distance is 100m.

On examination, he is barrel-chested and there is mild wheeze heard bilaterally. The jugular venous pulse is not raised and there is no peripheral oedema.

What is the most appropriate next step in management?

A. Add furosemide
B. Add oral prednisolone
C. Add roflumilast
D. Refer for lung volume reduction surgery
E. Add zafirlukast

C. Add roflumilast

Roflumilast should be used in COPD for patients who are losing control on triple inhaled therapy

Add roflumilast is correct. This is a long-acting inhibitor of the enzyme phosphodiesterase-4. This is an option for treating severe COPD in cases where the FEV1 is < 50% and the person has had 2 or more exacerbations in the previous 12 months despite triple inhaled therapy with a long-acting muscarinic antagonist, a long-acting beta-2 agonist and an inhaled corticosteroid.

Add furosemide is incorrect. The patient presents with worsening symptoms of COPD rather than right heart failure. He is euvolemic and furosemide would not be of benefit in this case.

Refer for lung volume reduction surgery is incorrect. To be a candidate for lung volume reduction surgery, patients need to be able to complete a six-minute walk distance of at least 140m.

Add zafirlukast is incorrect. This is an orally administered leukotriene receptor antagonist used for the chronic treatment of asthma rather than COPD.

Add oral prednisolone is incorrect. This is typically used to treat acute exacerbations of COPD rather than prevent further exacerbations.

Question 27 of 231

A 25-year-old man attends the clinic. He is planning an expedition to the peak of Mount Everest. He is keen to take a medicine to prevent against acute mountain sickness. He has no past medical history and takes no regular medications.

What treatment will you prescribe?

A. Dexamethasone
B. Nifedipine
C. Sildenafil
D. Dorzolamide
E. Acetazolamide

E. Acetazolamide

Acetazolamide (a carbonic anhydrase inhibitor) can be used to prevent acute mountain sickness

If pharmacological prophylaxis of acute mountain sickness is required, then acetazolamide is first line. This drug is a carbonic anhydrase inhibitor which acts on the kidneys to generate a primary a metabolic acidosis. A compensatory respiratory alkalosis occurs in response, resulting in hyperventilation and improved oxygenation.

Dexamethasone, nifedipine and phosphodiesterase type V inhibitors (e.g. sildenafil) can be used both in the treatment and prophylaxis of acute mountain sickness, however for prophylaxis they are second line agents.

Dorzolamide is a topical carbonic anhydrase inhibitor used in glaucoma.

Question 28 of 231

A 27-year-old woman with brittle asthma presents to the Emergency department. She is managed with high dose salmeterol/fluticasone and montelukast but reports steadily worsening symptoms since starting with a cold some 48hrs earlier. She has been admitted to the intensive care unit on 3 previous occasions. On examination her blood pressure is 122/82 mmHg pulse rate is 88 beats per minute. On auscultation of her chest you hear quiet breath sounds with polyphonic wheeze, her respiratory rate is 30/min. She is given back to back salbutamol nebulisers, ipratropium nebulisers, and IV hydrocortisone. These interventions only lead to minimal improvement in her PEFR from 110 to 140, (330 predicted). Oxygen saturation is 92% measured by pulse oximeter, on 60% oxygen delivered via mask. You call the anaesthetist for possible intubation.

Which of the following is the most appropriate next step?

A. IV aminophylline
B. IV chlorpheniramine
C. IV magnesium
D. IV salbutamol
E. IV omalizumab

C. IV magnesium

This patient has acute severe asthma with little or no improvement from initial nebuliser therapy. As such she requires assessment for possible intubation. In the mean time however, guidelines support the use of IV magnesium. A dose of 2g is commonly given over a period of 20 mins, where it has been shown to improve pulmonary function when added to standard therapy.

Both IV aminophylline and salbutamol may be associated with significant tachyarrhythmias and hold limited benefit over and above inhaled beta agonists, they are therefore not recommended in this situation. Anti-histamines are of limited benefit, and IV omalizumab is used as a treatment for patients with allergic asthma which is resistant to other therapeutic options

Question 29 of 231

A 49-year-old man attends the emergency department after becoming unwell with symptoms of progressive shortness of breath. The man is a Saudi Arabian national, who arrived in the UK 3 days previously on a business trip. The patient reported his shortness of breath had gradually worsened over the previous 2 days to the point whereby even minor physical activity had become challenging. In addition, the patient had noticed a dry cough, runny nose, myalgia and fever over a similar period. The patient denied any history of a productive cough, haemoptysis or gastrointestinal symptoms.

The patient reported being in generally good health aside from the type 2 diabetes mellitus and hypertension. To control these conditions, the patient took regular metformin and captopril. The patient had no known allergies to medications. The patient worked as an international businessman and normally resided in Riyadh with his family. He denied any contact with domestic animals in recent months and had not knowingly spent time with any unwell contacts.

General examination of the patient revealed a slightly overweight middle aged man who appeared unwell and diaphoretic. Basic physical observations are recorded in the below table. Examination of the respiratory system revealed mild tachypnoea at rest but with unremarkable percussion and auscultation of both lung fields. Examinations of the cardiovascular and abdominal systems were unremarkable. No clinical evidence of deep vein thrombosis was identified. Please see below for results of initial investigations requested following assessment by the respiratory medicine team.

Heart rate	95 beats / minute
Respiratory rate	23 respirations / minute
Blood pressure	128 / 85 mmHg
Oxygen saturations (room air)	89 %
Oxygen saturations (O2 3 L / minute via nasal cannulae)	95 %
Glasgow coma scale score	15
Haemoglobin	145 g / dL
Mean cell volume	80.2 fl
White cell count	3.1 x 10>3 / microlitre
Lymphocytes	1.0 x 10>3 / microlitre (reference 1.5-4.0)
Platelets	115 x 10>3 / microlitre
Urea	6.4 mmol / L

Creatinine	85 micromol / L
eGFR	85 ml / min / 1.73 m2
Sodium	139 mmol / L
Potassium	4.0 mmol / L
C-reactive protein	74 mg / L (reference < 7)
Urinalysis	no abnormality detected
Chest x-ray	no pneumothorax; lung fields generally clear with no focal consolidation; no mass lesions
Electrocardiogram	normal sinus rhythm
Nasopharyngeal swab RTPCR	MERS-CoV RNA detected

What is appropriate medical management for the patient's condition?

A. Treatment in the community with anti-pyretic
B. Intravenous ganciclovir (5 mg / kg twice daily for 14 days)
C. In-patient supportive treatment to relieve symptoms and prevent or treat complications
D. Oral oseltamivir (75 mg twice daily for 5 days)
E. Inhaled zanamivir (10 mg twice daily for 5 days)

C. In-patient supportive treatment to relieve symptoms and prevent or treat complications

Middle East respiratory syndrome (MERS) is an acute viral respiratory tract infection caused by the betacoronavirus MERS-CoV. Infection with MERS-CoV is believed to originate from individuals coming in close contact with dromedary camels or consuming camel products (for example, unpasteurised camel milk). However, the majority of cases, are the result of human-to-human transmission via respiratory droplets, particularly secondary to outbreaks in the hospital setting. MERS is limited to the countries of the Arabian Peninsula, but individuals who have travelled from or through that region may present in other countries given the 2-14 day incubation period.

Most patients with MERS present with respiratory symptoms and fever. A cough secondary to MERS is typically dry, but may be productive in up to one-third of patients; haemoptysis is less common, occurring in around 10 % of patients. The clinical spectrum of MERS ranges from asymptomatic or minor respiratory symptoms through to severe pneumonia associated with multiorgan failure. Severe disease is more likely to occur in older individuals or in those with comorbidities.

Routine blood tests in MERS can show lymphocytopenia, thrombocytopenia, acute kidney injury or deranged liver function tests. MERS is confirmed via detection of viral RNA by reverse transcription polymerase chain reaction analysis of respiratory tract secretions.

Management of MERS requires intervention to minimise symptoms and prevent harm from complications such as respiratory failure or septic shock from superadded bacterial pneumonia. No specific drug treatment is available to treat MERS-CoV. The specific treatments given as possible answers are all anti-viral agents used in the treatment of other conditions: oseltamivir and zanamivir for influenza and ganciclovir for cytomegalovirus infections.

Given the patient's hypoxaemia, tachypnoea and comorbidities, it would not be appropriate to manage this patient in the community due to the high risk of clinical deterioration secondary to complications.

Question 30 of 231

A 50-year-old man is admitted under the respiratory team with a left lower lobe pneumonia. He has a history of chronic obstructive pulmonary disease and continues to smoke. He also suffers with with hypertension and high cholesterol.

He is treated with steroids, nebulisers and antibiotics. After 5 days he continues to be hypoxic with saturations of 86% on air, ongoing left basal crepitations and wheeze.

A CT scan of the chest is performed which shows emphysematous changes and resolving left basal consolidation. There is also a 7mm lung nodule in the right upper lobe with surrounding ground glass change.

Over the next 3 days his saturations improve to 93% on air and his symptoms resolve. He is discharged home.

What imaging does he require to follow up his pulmonary nodule?

A. PET CT 2 weeks
B. PET CT 4 weeks
C. Repeat chest x-ray 4 weeks
D. Repeat CT chest 1 year
E. Repeat CT chest 3 months

E. Repeat CT chest 3 months

Risk stratification of incidental pulmonary nodules is complex and needs to take into account the risk factors for lung cancer or metastases, as well as size and character of the nodule. Algorithms for surveillance are well defined in the British Thoracic Society Guidelines published in 2005.

Broadly:

- Nodules < 5 mm require no further surveillance.
- Nodules 5-6mm require CT at 1 year
- Nodules 6-8 mm require CT at 3 months

- Nodules > 8 mm require malignancy risk calculation using the Brock model and should then have CT or PET according to whether this risk is > 10%.

The Brock model (see McWilliams et al) takes into account age, gender, family history and features of the nodule

Question 31 of 231

A 51 year-old man is referred by her GP to the respiratory outpatient clinic. He has had several episodes of sinusitis over the past three years and is currently being referred to plastic surgery as the bridge of his nose has partially collapsed. A chest X-ray that was performed three weeks ago show five nodules and blood tests reveal a urea of 11.6 mmol/l and a creatinine of 198 µmol/l.

Which of the following is the most appropriate initial treatment for this condition?

A. Methotrexate
B. Cyclophosphamide
C. Azathioprine
D. Corticosteroids
E. Cyclophosphamide and corticosteroids

E. Cyclophosphamide and corticosteroids

This man has a likely diagnosis of granulomatosis with polyangiitis, as suggested by the recurrent sinusitis, collapsed nasal bridge and x-ray nodules. First-line treatment is with cyclophosphamide and corticosteroids in patients with moderate to severe disease.

Question 32 of 231

An 88-year-old man presents with increasing shortness of breath over the past three months. He lives with his wife and is usually independent, but is recovering after an admission 6 months ago for pneumonia.

He has lost 10kg in weight compared to before the admission. He has not had any fevers or night sweats. His chest feels tight at times.

He feels short of breath with minimal exertion and this is not worse at any time of day. He has a constant dry cough. He takes ramipril for hypertension and has never smoked. He had tuberculosis as a child.

On examination, he is frail and has bilateral crepitations in the left mid and upper zones and right middle and lower zones. The chest is resonant to percussion and his respiratory rate is 24 breaths/min. His saturation are 92% at rest but on standing drop to 86% and his lips turn purple. He has a palpable heave and his ankles are both swollen.

B-natriuretic peptide	upper limit of normal
CRP	13
FEV1 (% predicted)	87%
FVC (% predicted)	75%
ECG	right bundle branch block, sinus, rate 70/min
Chest X-Ray	reticular shadows bilaterally with reduced chest expansion
ABG in air	
pH	7.35
PaO2	7.9 kPa
PaCO2	6.8 kPa
HCO3	28 mmol/l

What is the most likely diagnosis?

A. Tuberculosis reactivation
B. Asthma
C. Pulmonary fibrosis
D. Congestive cardiac failure
E. Physiological ageing

C. Pulmonary fibrosis

Pulmonary fibrosis can occur following pneumonia

This gentleman has a compensated respiratory acidosis with hypoxia which suggests that he has been ill for at least a few weeks. He also has a restrictive picture on his lung function tests with a relatively preserved FEV1. These changes are abnormal and do not reflect ageing.

He has no sputum and no systemic features making TB unlikely.

He has no wheeze. Asthma would also have obstructive lung function tests.

His BNP is not supportive of cardiac failure.

Question 33 of 231

A 70-year-old woman presents to her GP with increasing shortness of breath over the last 4 months. She also reports an occasional productive cough. Her past medical history includes two previous heart attacks, both treated with percutaneous coronary intervention and hypercholesterolaemia that is treated with simvastatin. She has a 35 pack-year history and does not drink alcohol.

The GP refers her for lung function tests which reveal:

Forced expiratory volume in 1 second (FEV1)	0.78L
Forced vital capacity (FVC)	1.68L
Total lung capacity	5.08L

What is the most likely diagnosis?

A. Chronic obstructive pulmonary disease (COPD)
B. Congestive cardiac failure
C. Pulmonary embolism
D. Bronchiectasis
E. Asthma

A. Chronic obstructive pulmonary disease (COPD)

The most likely diagnosis in this case is COPD, based on the reduced FEV1 and and FEV1/FVC. The high pack-year history and the patient's age also points towards a diagnosis of COPD, as opposed to asthma.

Question 34 of 231

A 24-year-old woman is admitted to Accident and Emergency with acute shortness of breath. She is unable to detail a clinical history due to shortness of breath. Her medication includes;

Cetirizine 10mg Orally Once Daily Clobetasone butyrate (Eumovate) Topical

Her initial examination revealed bilateral widespread polyphonic wheezes. Her pulse rate was 102 beats per minute and blood pressure 128/75 mmHg. Her respiration rate was 33. Her oxygen saturations were 96% on 4L/min via face mask. She had a dry flaking rash in her elbow flexures with signs of excoriation.

The following blood tests were obtained;

Hb	11.2 g/dl
Platelets	204*10^9/l
WBC	10.3 *10^9/l
Eosinophil	0.56 *10^9/l
CRP	65
Na+	135 mmol/l
K+	4.4 mmol/l
Urea	7.6 mmol/l
Creatinine	101 µmol/l

Chest x-ray was clear. A peak expiratory flow was 200 L/min(expected 402). Initial arterial blood gas on 4L/min oxygen via face mask;

pH	7.46
pCO2	3.5 kPa
pO2	14 kPa
HCO3	19 mmol/l
Lactate	1.8 mmol/l

She is treated with nebulised salbutamol and ipratropium bromide and oral prednisolone 40mg and oxygen 4L/min.

You re-review the patient 30 minutes later on the Admissions Unit. Her examination reveals symmetrical quiet breath sounds and quiet bilateral wheezes. She has symmetrical chest wall movement. Her pulse rate is 80 beats per minute. Blood pressure 103/68 mmHg. Her respiratory was 18 with oxygen saturations 94% on 6L/min via face mask. Peak expiratory flow was 120 L/min.

Repeat arterial blood gas on 6L/min oxygen via face mask;

pH	7.33
pCO2	6.2kPa
pO2	13kPa
HCO3	20 mmol/l
Lactate	2.2 mmol/l

What is the most appropriate next step in management?

A. Intravenous Magnesium
B. Reduce oxygen levels
C. Intravenous Salbutamol
D. Referral to intensive care
E. Biphasic positive airway pressure

D. Referral to intensive care

According to British Guideline on Management of Asthma BTS/SIGN 2011.

This patient had acute severe asthma as highlighted by her first set of results and examination.

Acute Severe

- **PEF 33-50% best or predicted**
- **RR >25, HR >110**
- **Inability to complete sentances**

Her ABG was consistent with type one respiratory failure with hypocapnia due to hyperventilation. Her condition deteriorated to life threatening with raised PaCO2 suggesting of near fatal asthma

Life Threatening

- PEF <33%
- SpO2 <92% or PaO2 <8kPa
- Normal PaCO2
- Silent chest, cyanosis, poor respiratory effort, arrhythmia, exhaustion

Near Fatal

- Raised PaCO2
- Requiring mechanical ventilaion with raised inflation pressures

The patient had a deteriorating picture. Her respiratory rate was decreasing suggesting exhaustion, worsening hypoxia, hypercapnic acidotic ABG, deteriorating PEF indicating deterioration.

Intravenous Magnesium - This patient would certainly require consideration of IV Magnesium however as the picture is deteriorating they would require ITU referral immediately.

Reduce oxygen levels - Incorrect. Retention of CO2 is not secondary to her oxygen therapy, it is more likely due to exhaustion.

Intravenous Salbutamol - Not utilised in current guidance.

Referral to intensive care - Correct answer. The patient has near fatal asthma with deterioration despite nebulisers and steroids.

Biphasic positive airway pressure - No evidence to support NIV use in acute asthma.

Question 35 of 231

A 27-year-old woman presents for review to the respiratory clinic. She has a past medical history of cystic fibrosis and has been struggling at home. Her GP sent a sputum sample prior to her clinic appointment as he was concerned with the worsening nature of her cough and shortness of breath.

Which microorganism is associated with the worst prognosis?

A. Burkholderia
B. Klebsiella pneumoniae
C. Pseudomonas aeruginosa
D. Staphylococcus aureus
E. Haemophilus influenzae

A. Burkholderia

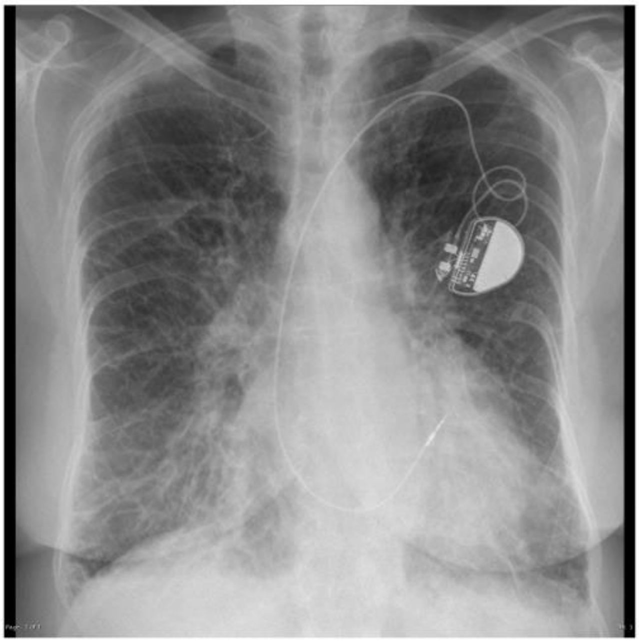
The correct answer is Burkholderia. All of the pathogens listed are potential pathogens which commonly cause exacerbation of cystic fibrosis, but Burkholderia and Pseudomonas are two pathogens which are very difficult to eradicate. Of those, Burkholderia has the worst prognosis.

Question 36 of 231

An 80-year-old woman presents with dyspnoea to the Emergency Department. According to her daughter she has been getting progressively worse over the past 4 weeks. Her past medical history includes a myocardial infarction 20 years ago following which she had a 'pacemaker' inserted. She still smokes 20 cigarettes/day.

On examination her pulse is 120/min irregularly irregular. Blood pressure is 102/72 mmHg, oxygen saturations are 94% on 40% oxygen via a Venturi mask.

Her chest x-ray is shown below:



What is the most appropriate initial treatment?

A. Oral beta-blocker
B. Low-molecular weight heparin
C. Intravenous loop diuretic
D. Reduce oxygen to 24% and nebulised bronchodilator
E. Intravenous broad-spectrum antibiotic

C. Intravenous loop diuretic

The chest x-ray is consistent with acute pulmonary oedema. Single lead PPM in situ. The heart is enlarged and there is marked prominence of the interstitial lines. Minimal blunting of the left costophrenic angle.

Question 37 of 231

You are caring for 54 year-old lady on the respiratory ward. She has community acquired pneumonia and large left sided pleural effusion. She is dyspnoeic at rest and hypoxic - oxygen saturations are 96% on 60% high flow oxygen.

A bedside ultrasound scan shows an anechoic effusion with no apparent loculations. You proceed to perform a diagnostic aspirate and the results are as follows:

pH	6.9
Glucose	2.9mmol/L
Protein	32g/dL
Gram stain	No organisms seen

What is the correct course of action?

A. 18Fr chest drain and intravenous antibiotics
B. Therapeutic aspiration and intravenous antibiotics
C. Referral to thoracic surgeons
D. Broad spectrum intravenous antibiotics
E. 18Fr chest drain, intravenous antibiotics and intrapleural fibrinolysis

A. 18Fr chest drain and intravenous antibiotics

Although the ultrasound appearances in this case are suggestive of a simple parapneumonic effusion the aspirate results suggest an empyema. Empyema must be treated promptly and aggressively with broad spectrum antibiotics and drainage*. Intra-pleural fibrinolysis is useful in loculated effusions and thoracic surgery should be considered if initial management fails

Question 38 of 231

A 56-year-old gentleman was admitted to the Emergency Department with a two-week history of progressively increasing shortness of breath, with frank haemoptysis for the last couple of days. He had recently returned from a holiday in Turkey a few weeks ago. By the point of admission, he was short of breath on minimal exertion and at rest and was unable to complete full sentences. He also complained of increasing orthopnoea for the last few weeks. He had a long history of recurrent epistaxis, rhinitis and sinusitis, as a well as a past medical history of diabetes, hypertension, hypercholesterolaemia and gout. His medication regimen comprised of mometasone nasal spray, allopurinol 100mg OD, ramipril 2.5 mg OD, amlodipine 5mg OD, simvastatin 40mg OD, metformin 500mg TDS and gliclazide 80mg OD. He smoked 20 cigarettes per day and did not consume alcohol.

On examination, he appeared very unwell with obvious respiratory distress. His respiratory rate was 28/min, his oxygen saturations were 90% on air, his blood pressure was 108/72 mmHg, his heart rate was 129 and his temperature was 37.9 degrees celsius. Examination of his cardiovascular system revealed normal heart sounds with a JVP of 3cm. Examination of the respiratory system revealed the use of accessory muscles with bibasal fine crackles. Examination of the gastrointestinal and neurological systems was unremarkable.

Initial investigations revealed the following:

Hb	82 g/l
Platelets	342 * 10 /l
WBC	14.2 * 10 /l
ESR	88 mm/hr
Na	128 mmol/l
K	6.1 mmol/l
Urea	28 mmol/l
Creatinine	762 µmol/l
CRP	42 mg/l
Glucose	5.6 mmol/l

Chest x-ray: bilateral patchy infiltration

ECG: heart rate 132 bpm normal sinus rhythm, no other changes Urinalysis blood ++++ protein +++ leuc/nit negative glucose negative

ABG on 15 l/min oxygen:

PaO2	16.9 kPa
PaCO2	2.8 kPa
HCO3	18.2 mmol/l
pH	7.49

He was catheterized, commenced on an intravenous infusion of normal saline, as well as coamoxiclav 625mg TDS and quickly transferred to the Intensive Care Unit. A decision was made to institute immediate haemodialysis, for which preparations were being made.

Further investigations revealed the following: Urine MCS: nil grown

Blood culture - interim results: nil grown

Transoesophageal echocardiogram: normal systolic function, valvular appearances and no vegetations seen

C3	1.22 (NR 0.65 1.65g/L)
C4	0.32 (NR 0.16 0.60 g/L)
ANA	negative
ENA	negative
dsDNA	negative
cANCA	positive
pANCA	negative
Rheumatoid factor	negative

Given the likely underlying diagnosis, which of the following interventions is the next best step whilst awaiting haemodialysis?

A. Stop IV augmentin, commence IV meropenem
B. Commence IV immunoglobulins
C. Commence high dose IV methylprednisolone and cyclophosphamide
D. Institute immediate plasma exchange
E. Commence intravenous activated protein C

C. Commence high dose IV methylprednisolone and cyclophosphamide

This patient has granulomatosis with polyangiitis, with severe pulmonary and renal involvement. He requires urgent immunosuppression. Of the available options, the next best step is therefore to institute cyclophosphamide and methylprednisolone. The options may serve a role, but this question is asking what the most appropriate next best step is. Therefore the best option is option 3

Question 39 of 231

An 83-year-old female presents to A&E with a four day history of increasing shortness of breath and worsening of a chronic productive cough. She speaks no English but you know from her medical records that she recently emigrated from rural India to live with her family two years ago. She has no diagnosed underlying lung condition. On examination, she has bilateral expiratory wheeze and hyperexpanded lungs with no clear inspiratory crackles. Heart sounds appear normal and mild bilateral pitting oedema. Her saturation measures 88% on air via pulse oxymeter, respiratory rate is 24 to 28 per minute. Her chest radiograph is hyperexpanded with mild bibasal fibrotic changes but demonstrates no focal signs of consolidation. She has no smoking or alcohol history. Her blood tests are as follows:

Hb	15.0 g/dl
Platelets	211 * 10 /l9
WBC	11.4 * 10 /l9
Neutrophils	9.5 * 10 /l9
Urea	8.4 mmol/l
Creatinine	112 µmol/l
CRP	37 mg/l

What is the most likely diagnosis?

A. Exacerbation of congestive cardiac failure
B. Infective exacerbation COPD
C. Community acquired pneumonia
D. Tuberculosis
E. Progression of pulmonary fibrosis

B. Infective exacerbation COPD

Although COPD is often associated as a disease of smokers, it is prudent to remember other causes of chronic emphysematous changes. In patients from the developing world, non-smokers from the Indian subcontinent and African countries often present with a COPD-like history from using open fires in their homes, particularly if burnt indoors for cooking or heating. The World Health Organisation (WHO) estimate that up to 3 billion people cook in this manner, accounting for up to 1 million deaths worldwide as a result of COPD without smoking history . The clinical findings are consistent with an obstructive picture and hyperexpansion of the lungs suggest a chronic element to an underlying lung condition, despite no formal previous diagnosis. Mild oedema may simply represent a degree of right heart failure from the underlying lung pathology. Bibasal fibrotic changes may be consistent with lung fibrosis but you would expect a restrictive picture instead. Community acquired pneumonia would be the main differential diagnosis but again, does not account for the chronic cough or hyperexpanded lungs.

Question 40 of 231

A 52-year-old man presents to the emergency department with shortness of breath. This started recently following a flight from Australia. He is morbidly obese and has a past medical history of type two diabetes mellitus, high cholesterol, hypertension and a previous cholecystectomy. He denies any leg pain or swelling and denies any chest pain. Observations are stable, and an ECG demonstrates sinus rhythm. A chest X-ray is clear. He undergoes a CT pulmonary angiogram (CTPA) to exclude a pulmonary embolus (PE). The CTPA is reported as finding no evidence of PE but notes a 7mm pulmonary nodule in the left lower lobe.

How should the pulmonary nodule be investigated or monitored?

A. Urgent CT-PET
B. Urgent image-guided biopsy
C. CT chest in three months
D. CT chest in one year
E. No further investigations needed

Not answered in the file

Question 41 of 231

A 40-year-old lady presents with dyspnoea. She describes a gradual onset of shortness of breath on exercise over several months. She has a past medical history of wheeze as an infant. She has recently commenced a intranasal steroid spray for rhinitis. She is a non-smoker.

On examination she has oxygen saturations of 91% on air. She has blood stained crusting around her anterior nares. On auscultation her chest has bilateral reduced air entry and coarse crepitations on both lung bases. Her temperature is 37.8 degrees centrigrade.

Hb	13.2 g/dL
WCC	10.3 *10^9/l
Neutrophil	7.8 *10^9/l
Eosinophil	0.07 *10^9/l
Sodium	135 mmol/l
Potassium	4.5 mmol/l
Creatinine	113 µmol/l
Urea	5.4 mmol/l
CRP	230 mg/L
Chest X - ray	Bilateral basal consolidation
Urine microscopy	Culture negative RBC +++ WCC +
c-ANCA	Positive
p-ANCA	Positive
Anti-GBM	Negative

What is the most likely diagnosis?

A. Pneumonia
B. Churg-Strauss Syndrome
C. Microscopic polyangiitis
D. Granulomatosis with polyangiitis
E. Goodpastures disease

D. Granulomatosis with polyangiitis

Consider granulomatosis with polyangiitis when a patient presents with ENT, respiratory and kidney involvement

Definitive diagnosis of vasculitis will require biopsy. However, granulomatosis with polyangiitisis the most likely as the presentation includes sinus disease, lung disease with likely renal involvement. Both ANCA tests can be positive but it is most associated with c-ANCA.

This patient had wheeze as a child, which is very common. Churg-Strauss is more associated with a progressive presentation of asthma, which may present later in life. The patient also does not have eosinophilia which is associated with this diagnosis.

Goodpasture's is a possibility but less likely with a negative anti-GBM. Pnemonia is less likely with such a prolonged presentation.

Microscopic polyangiitis can present very similar to granulomatosis with polyangiitis but is more associated with renal involvement, which is mild in this case. It is less associated with ENT disease.

Question 42 of 231

A 67-year-old man attends with reduced consciousness and shortness of breath. His wife accompanying him reports some diarrhoea over the past few days and he has taken himself to bed but was weak and unable to get up today. He is usually well but has been reporting morning headaches and is being referred to the sleep clinic. He takes no regular medications.

On examination, he has a respiratory rate of 11 breaths/minute and is saturating at 94% on 4 litres through a face mask. His chest clear is clear and his abdomen is soft and non tender. He is generally weak but is able to follow commands. It is difficult to understand him and you can only hear whispers when he speaks to you. His calves are soft with mild oedema in the ankles and he is overweight.

Na +	139mmol/l
K +	4.2 mmol/l
Urea	6.5 mmol/l
Creatinine	98 µmol/l
CRP	34
Hb	119 g/l
Platelets	298 * 10 /l9
WBC	5.6 * 10 /l9
ABG (in air)	
pH	7.30
pO2	7.9 kPa
pCO2	6.5 kPa
HCO3	24mmol/l
Lactate	2.4 mmol/l
Chest x-ray	poor inspiratory effort, clear lung fields, heart size within normal parameters
ECG	sinus tachycardia

What is the likely diagnosis?

A. Obstructive sleep apnoea
B. Myasthenia gravis
C. COPD exacerbation
D. Pneumonia
E. Pulmonary embolism

B. Myasthenia gravis

Type 2 respiratory failure with normal CXR -> neuromuscular weakness

This gentleman has presented with a type 2 respiratory failure. His bicarbonate is normal suggesting this is an acute deterioration and is less likely to be a COPD exacerbation or obstructive sleep apnoea where there is CO2 retainers and a raised bicarbonate. His chest x-ray shows no evidence of pneumonia. A pulmonary embolism is possible but usually presents with type 1 respiratory failure. Given that he has a poor respiratory effort and is not tachypnoeic, this should prompt thoughts of whether the ventilation is inadequate and this may reflect neuromuscular failure. These patients have shallow ventilation and spirometry shows a hugely reduced functional vital capacity.

Question 43 of 231

A 35-year-old woman is investigated for lethargy, fever, cough and weight loss. She has been treated by her GP with five courses of

antibiotics over the past six months for a combination of chest and sinus infections. Her symptoms, however, have continued unabated.

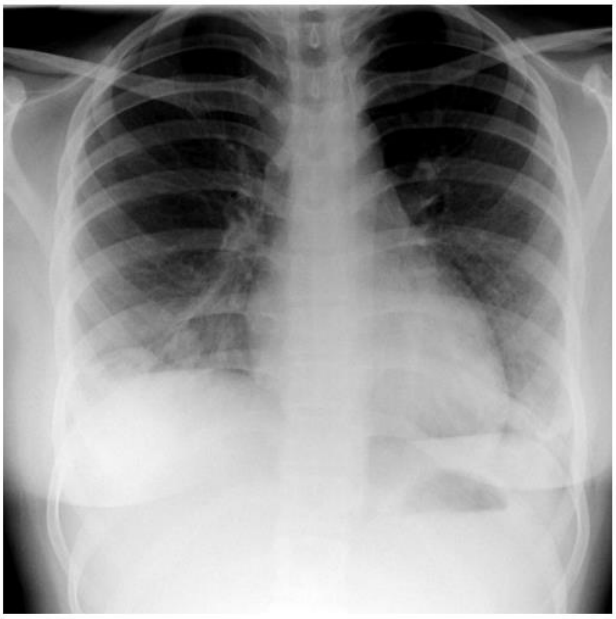
Clinical examination is unremarkable other than some scattered crackles in the right base. She is apyrexial. Urine dipstick: blood ++, protein +

Lung function tests and blood tests show the following

FEV1	4.3 L (99% of predicted)
FVC	5.1 L (101% of predicted)
FEV1/FVC	84%

Hb	13.1 g/dl	Na +	141 mmol/l
Platelets	459 * 10 /l 9	K +	3.6 mmol/l
WBC	6.9 * 10 /l 9	Urea	10.9 mmol/l
Creatinine	131 µmol/l		
CRP	78 mg/l		

A chest x-ray is requested:



What is the most likely diagnosis?

A. Lung cancer
B. Tuberculosis
C. Granulomatosis with polyangiitis
D. Cryptogenic organizing pneumonia
E. Churg-Strauss syndrome

C. Granulomatosis with polyangiitis

Whilst the changes are subtle the chest x-ray demonstrates a number of ill-defined nodules the largest of which projects over the dome of the right hemidiaphragm. This nodule appears to have a central lucency suggesting cavitation. Please see the accompanying CT scan from the same patient below.

Question 44 of 231

A 42-year-old lady presents to the Emergency Department with a 3-week history of a dry cough, fever and lethargy. She has no past medical history and takes no regular medications. There is no history of recent foreign travel. Clinical examination reveals inspiratory crepitations affecting the left upper zone and R lower zone. Saturations are 91% on room air. Chest x-ray shows bilateral peripheral pulmonary infiltrates.

Bloods show:

Hb	110 g/L
MCV	78 fL
WCC	13 x 10^9/L
Neutrophils	8 x 10^9/L
Lymphocytes	1 x 10^9/L
Eosinophils	3.1 x 10^9/L
Platelets	560 x 10^9/L
CRP	115mg/l

What is the most appropriate treatment given the likely diagnosis?

A. Oral prednisolone
B. High dose co-trimoxazole
C. Plasma exchange
D. Rifampicin, isoniazid, ethambutol and pyrazinamide
E. Amoxicillin and clarithromycin

A. Oral prednisolone

Eosinophilic pneumonia is the most likely diagnosis. It is characterised by sub acute respiratory symptoms, alveolar and/ or blood eosinophilia, and peripheral pulmonary infiltrates on imaging. Treatment with oral corticosteroids leads to a rapid improvement of symptoms and the prognosis is good. Tuberculosis is usually associated with upper lobe consolidation and eosinophilia is not typical. Pneumocystis jirovecii pneumonia and community-acquired pneumonia are not usually associated with eosinophilia and are less likely.

Question 45 of 231

An 18-year-old-female is brought into the emergency room after having a tonic-clonic seizure. On examination she has a Glasgow coma scale of 11 (M4, E3, V4). Her blood pressure is 85/65 mmHg. Her heart rate is 135bpm and an ECG demonstrates atrial fibrillation. She has a past medical history of asthma which she takes betamethasone, salbutamol, salmeterol, montelukast and theophylline. She has recently started erythromycin for a chest infection. She has been taking 6g of paracetamol per day for the last 3 days for fever and chest discomfort.

You commence fluid resuscitation with 0.9% saline and treat the supraventricular tachycardia with a beta blocker.

You suspect theophylline toxicity and administer repeated doses of activated charcoal. However the patient continues to deteriorate.

What treatment is indicated?

A. N-acetylcysteine (NAC)
B. Sodium bicarbonate
C. Flumazenil
D. Hydroxocobalamin
E. Intermittent haemodialysis or charcoal haemoperfusion

E. Intermittent haemodialysis or charcoal haemoperfusion

In cases of severe theophylline toxicity, charcoal haemoperfusion can be used

The patient has presented with several features of theophylline toxicity - seizures, reduced Glasgow coma score, and a tachyarrhythmia. The drug erythromycin which has recently been started, is an enzyme inhibitor which can precipitate theophylline toxicity.

The severity of symptoms warrants use of haemodialysis orcharcoal haemoperfusion.

Activated charcoal should also be considered. Since many products are slow release formulations, charcoal given more than 1 hour after ingestion may be of benefit. Repeated doses should also be administered.

Although the patient has been exceeding the maximum dose of paracetamol (4g / 24 hours), the clinical features are not in keeping with paracetamol toxicity.

Hydroxocobalamin is the antidote for cyanide poisoning.

Flumazenil is the antidote for benzodiazepine overdose.

Question 46 of 231

A 35 year old chronic alcoholic male is admitted with severe epigastric pain. Laboratory investigations confirmed a significantly raised amylase. He is started on treatment for severe pancreatitis but his condition deterioriated over the next 2 days and he developed acute kidney injury with severe respiratory distress to an extent needing mechanical ventilation. Initial chest radiographs revealed widespread bilateral infiltrates.

Which of the following therapies has been shown to most likely decrease overall mortality of ARDS?

A. Immediate initiation of glucocorticoid therapy to limit inflammatory progression
B. Placing patient in the prone position, allowing for greater recruitment of unaffected alveoliand improving PaO2/FiO2 ratio
C. Implementing a low tidal volume ventilation protocol
D. Changing from O2 to Nitric oxide at 5 ppm and titrating up until pt has an acceptablePaO2/FiO2 ratio
E. Aggressive diuresis to reduce pulmonary edema and vascular congestion

C. Implementing a low tidal volume ventilation protocol

Choice A - glucocorticoids to this point have not been shown to help patients in the acute phase of ARDS. There is some evidence that they may help in the fibroproliferative stage but not acutely

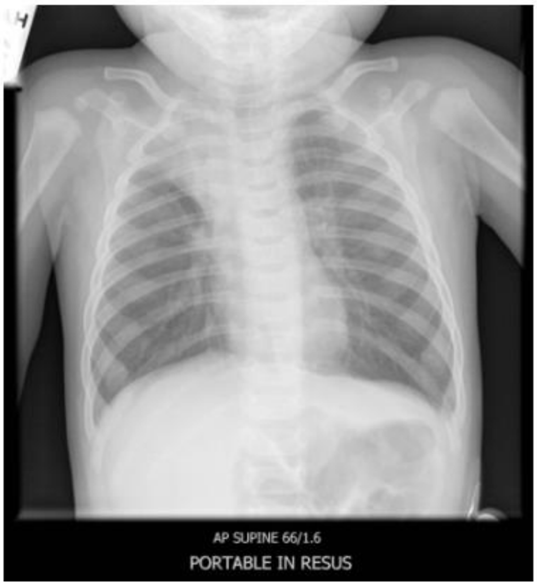
Choice B - While this is true, there is as of yet no good evidence showing improved overall mortality.

Choice C - ARDS causes severe hypoxaemia, almost always requiring invasive artificial ventilation. However, mechanical ventilation itself can injure the damaged lungs causing ventilation-induced lung injury. The main goal of treatment is to minimise any additional damage while maintaining adequate gas exchange. This has been effectively done by maintaining low tidal volume ventilation. A Cochrane Database review (1) and meta-analysis (2) suggest that a strategy of low tidal volume ventilation reduces the absolute mortality by about 7-9% as compared to using higher tidal volumes. This converts to a Number needed to treat (NNT) of between 11-15 people with ARDS to prevent one death by using low tidal volume ventilation.

Choice D - Nitric oxide does improve PaO2/FiO2 ratio but has not shown improved survival Choice E - Since the infiltrate of ARDS is primarily inflammatory, diuretics are not particularly effective. Overdiuresis also runs the risk of hemodynamic compromise, further decreasing the patient's already insufficient oxygen delivery to tissues

Question 47 of 231

The x-ray below was taken from a 7-year-old child who presented to the Emergency Department with respiratory symptoms:



What does the x-ray show?

A. Cystic fibrosis
B. Right upper lobe collapse
C. Lung cancer
D. Right-sided pneumothorax
E. Normal

B. Right upper lobe collapse

There is increased opacity in the right upper zone, The lateral / inferior border of the shadowing actually represents the horizontal fissure which has been 'dragged' upwards.

Question 48 of 231

A 56-year-old male presents with symptoms and signs consistent with exacerbation of chronic obstructive pulmonary disease (COPD). This is his fourth exacerbation in the last two months. His most recent FEV 1 is 40%. He is currently taking salbutamol as required once daily regimen.

On reviewing his peak flow diary you note a significant (> 20%) diurnal variation in his peak flow.

Which of the following drugs should be added next to his regular control of COPD?

A. Combined salmeterol and fluticasone inhaler
B. Salmeterol
C. Theophylline
D. Ipratropium bromide
E. Long term oxygen therapy

A. Combined salmeterol and fluticasone inhaler

COPD - still breathless despite using SABA/SAMA and **asthma/steroid responsive features** → add a LABA + ICS

As this patient has a feature suggestive of asthma/steroid responsiveness (significant diurnal variation) the next step in management is a long-acting beta agonist (e.g. salmeterol) + an inhaled corticosteroid. NICE now recommend the use of combined inhalers where possible.

Question 49 of 231

A 22-year-old agricultural student presents to the respiratory clinic with cough, breathlessness, fatigue and weight loss of 2 kg in 6 weeks. Examination reveals bilateral wheeze and crackles. His GP has tried 2 courses of antibiotics and a salbutamol inhaler with no real benefit; he had some relief from a short course of oral prednisolone. He denies any fever, haemoptysis, foreign travel or exposure to TB. He has never smoked.

His chest radiograph shows mid to upper zone alveolar fine nodular shadowing bilaterally. His lung function shows a FVC (forced vital capacity) of 70% and FEV1/FVC of 85% predicted. Bloods show:

CRP	3 mg/l
ESR	45mm/hr

What is the most likely diagnosis?

A. Pulmonary embolus (PE)
B. Community acquired pneumonia
C. Asthma
D. Extrinsic allergic alveolitis
E. Silicosis

D. Extrinsic allergic alveolitis

Extrinsic allergic alveolitis is associated with upper/mid zone lung fibrosis

Extrinsic allergic alveolitis is the most likely diagnosis in this agricultural student with a restrictive picture on spirometry. Extrinsic allergic alveolitis is caused by acute neutrophilic inflammation followed by lymphocytic infiltration & collagen deposition. Broncho alveolar lavage would show an excess of eosinophils. The presence of crepitations and wheeze make the diagnosis of PE less likely. Community acquired pneumonia would be associated with a higher CRP. Asthma would not cause a restrictive picture on spirometry. Silicosis is unlikely as there is no occupational exposure to silicon dioxide.

Question 50 of 231

A 53-year-old gentleman presents to the respiratory clinic for review. He has a history of chronic recurrent coughing episodes lasting for months. This has been worse over winters. He also has found that over the last two years his exercise tolerance has reduced and that he is more easily short of breath. He has no past medical history and takes no regular medications. He has a 20 pack-year history. He has undergone spirometry with shows an FEV1/FVC ratio of 63%, whilst FEV1 is 74% of predicted. His chest X-ray is unremarkable. Blood tests are normal as well. Previous sputum sample from during an exacerbation shows growth with pseudomonas.

What factor is not in keeping with a diagnosis of COPD?

A. FEV1/FVC ratio
B. Pseudomonas
C. FEV1
D. Smoking history
E. Clinical history

B. Pseudomonas

The correct answer is Pseudomonas. This is a patient with an otherwise straightforward diagnosis of COPD. COPD is an obstructive disease and therefore an FEV1/FVC ratio of 63% is matching, as does a low FEV1. Smoking is the main cause of COPD as well, and the history, describing winter bronchitis with a general decline in exercise tolerance fits COPD too. Pseudomonas is more common in bronchiectasis but may be a contaminant rather than a pathogen

Question 51 of 231

A 66-year-old retired farmer presents with a 4-week history of increasing shortness of breath, nonspecific non-pleuritic chest pain and weight loss. His past medical history includes previous angina, for which he was previously prescribed a GTN spray that he has not required for 3 years and hypertension. He is known to keep racing pigeons in a barn on his property. He keeps active and lives with his wife, drinks moderate amount of alcohol and is an active smoker with a 40 pack year history. He has no known allergies. On examination, you note bilateral clubbing and tar staining. Respiratory examination revealed a respiratory rate of 19 breaths per minute, sats 94% on air, reduced bilateral chest expansion and reduced air entry in both bases associated with dullness to percussion.

A chest radiograph demonstrates moderate right >left bilateral pleural effusions and patchy opacities across both lung fields in a non-lobar distribution. CT thorax with high-resolution slices demonstrates bilateral pleural effusion, thickened pleura with no lung parenchyma abnormalities.

Lung function testing demonstrates FEV1 1.7ls, FVC 55% of predicted.

What is the most appropriate next investigation?

A. CT chest/abdomen/pelvis with contrast
B. High resolution CT chest (HRCT)
C. Video assisted thoracoscopic biopsy (VATS)
D. Chest drain
E. Bronchoscopy with bronchoalveolar lavage (BAL)

C. Video assisted thoracoscopic biopsy (VATS)

An underlying pleural pathology should be strongly suspected in the case of thickened pleura, restrictive lung function and non-specific constitutional symptoms. The bilateral pleural effusions do not appear to be causing respiratory compromise and hence a chest drain is not indicated. In the absence of parenchymal lesions to target for tissue diagnosis, VATS is appropriate to differentiate pleural thickening described between organised empyema or mesothelioma, and also to obtain tissue to differentiate mesothelioma from other primary or metastatic cancers. VATS is optimally performed with a small amount of pleural fluid in situ.

A diagnosis of extrinsic allergic alveolitis (EAA) should also be considered as presentations with weight loss, dyspnoea and chest tightness can be similar to pleural pathology. BAL would be indicated if EAA is a significant differential. However, the lack of reticulonodular changes on chest radiograph and fibrotic changes on CT make hypersensitivity pneumonitis less likely. Be aware that only 20% of mesothelioma patients present with radiographic changes consistent with asbestosis, although the majority present with pleural abnormalities.

Question 52 of 231

A 72 year-old woman has been referred to the chest clinic by her General Practitioner (GP). The patient describes a 3-year history of cough and progressive shortness of breath. Over the past 3 years the GP had prescribed numerous courses of antibiotics and steroids but these had had little benefit. She was a non-smoker, did not drink alcohol and was on no regular medications. The cough was productive of purulent sputum with occasional streaks of blood. There was no history of weight loss or fever, but the patient did note lethargy. The rest of her family were well and the only medical history of note was an episode of pneumonia as a child.

Respiratory examination revealed bilateral crackles but no other added sounds. You note that the patient was not clubbed.

A chest x-ray, organised by the GP, shows minor bilateral atelectasis and opacities.

What is the most appropriate diagnostic test?

A. Bronchoscopy
B. Genetic testing for Primary Ciliary Dyskinesia
C. High resolution CT scan of chest
D. Serum α1-antitrypsin level
E. Tuberculin skin test

C. High resolution CT scan of chest

The history is highly suggestive of bronchiectasis, which often presents as a lower respiratory tract infection with a productive cough. Patents may also present with haemoptysis, chest pain and shortness of breath. Over time the condition can progress to respiratory failure.

The most common findings on examination are crackles (75%) and wheeze (22%). Clubbing is only found in 2%.

A chest x-ray should be performed in all suspected cases and, indeed, anyone presenting with a chronic cough. In some cases a suggestive history and chest x-ray will be enough to diagnose bronchiectasis. However, the best diagnostic test for bronchiectasis is high-resolution CT scan (HRCT). A HRCT will show the classic features of bronchial wall thickening and dilatation, it may also demonstrate features that suggest a cause for the underlying cause, e.g. allergic bronchopulmonary aspergillosis (ABPA) of cystic fibrosis (CF).

The routine work up for patients with suspected bronchiectasis should include bloods for serum immunoglobulins and protein electrophoresis, total IgE and skin prick testing or serum IgE testing to Aspergillus species. Bronchoscopy is not performed routinely unless there is a suspicion of a lesion or foreign body. α1-Antitrypsin levels are not routinely requested but may be of help if the HRCT shows evidence of emphysema. A tuberculin skin test is very unlikely to be helpful in a patient of this age and is not the test of choice. Tuberculin skin tests can identify cases of latent tuberculosis but must be interpreted with the patients immunisation history.

Genetic testing, although potentially diagnostic, would not be appropriate in this case. Primary Ciliary Dyskinesia is an autosomal recessive disorder that can cause bronchiesctasis but patients usually present as newborns or in childhood and not in the elderly.

Question 53 of 231

A 30 year old gentleman returns from a camping holiday in the Nile Delta with nocturnal wheeziness and dry cough, weight loss, and fever. He recalls multiple mosquito bites. Chest X-ray demonstrates pulmonary infiltrates. His blood results are shown below.

Hb	14 g/dl
Platelets	385 * 10 /l9
WBC	14 * 10 /l9
Neutrophils	7.5 * 10^9/l (reference range 2.0-7.5 * 10^9/l)
Eosinophils	1.2 * 10^9/l (reference range 0.04-0.44 * 10^9/l)
Serum IgG	90 mg/dl (reference range 80 - 350 mg/dl)
Serum IgM	200 mg/dl (reference range 45 - 250 mg/dl)
Serum IgE	6.0 mg/dl (reference range 0.002 - 0.2 mg/dl)

Given the likely diagnosis which of the following treatments will be most effective?

A. Antiretrovirals
B. Tazocin
C. Steroids
D. Diethylcarbamazine
E. Quinine

D. Diethylcarbamazine

The diagnosis here is tropical pulmonary eosinophilia. HIV and indeed plasmodium falcipirum malaria could present with fever and weight loss however the blood picture along with wheeze is suggestive of an eosinophilic lung disease. The eosinophilic lung diseases are allergic reactions to some environmental toxin and all present with cough, wheeze, pulmonary infiltrates on chest Xray, raised serum IgE, and a raised eosinophil count. They are a differential for asthma, which can present in a similar fashion clinically and on bloods, and therefore a careful history (particularly travel and medications) is important. It is important to recognise them as the management is different to asthma. Here are some examples worth knowing for the exam:

Allergic bronchopulmonary aspergillosis: an allergic reaction to the fungus aspergillus fumigates. The course is characterised by recurrent episodes of eosinophilic pneumonia, especially in the autumn and winter. Test for precipitating antibodies to Aspergillus species, which will be present in 90% of cases. Also order sputum microscopy and culture, which can show fungal spores or hyphae. A skin-prick test can be ordered and almost always positive to Aspergillus fumigates. Management is with steroids. Simple pulmonary eosinophilia, aka Loeffler's syndrome. This is caused by Ascaris lumbricoides, Strongyloides, or Ancylostoma. Infection with these worms is prevalent worldwide, especially in tropical and subtropical countries. Their eggs are passed in the stool of an infected person and then ingestion in food that has had contact with human sewage (they are surprising resilient eggs). The ingested eggs mature into larval worms in the gut and seed into the blood stream via the duodenal walls, from where they circulate around in the blood to eventually enter the lungs were they are coughed up (the pulmonary eosinophilia phase with wheeze and cough for around 2 weeks) and then swallowed back down and settle into mature worms in the small intestine, resulting in a chronic malabsorption condition. The pulmonary condition is self-limmiting although mebendazole given for 3 days can help.

Tropical pulmonary eosinophilia: caused by the roundworms Wuchereria bancrofti or Brugia malayi, common in the Asian Subcontinent and Africa. The worms are spread via a mosquito vector. It takes a human blood meal and thereby ingests microfilariae (worm-like sheathed eggs) from an infected patient and deposits them into a new host. They spread through the blood to settle in the lymphatics where they mature into adults and reside there for 15 or more years, reproducing sexually, and causing elephantiasis. The pulmonary involvement happens as there is intense inflammation of lung parenchyma to microfilariae which are periodically released into the circulation by those adult worms. There is therefore a history of paroxysmal nocturnal cough, wheeze, and breathlessness. Diagnosis is aided by a clinical response to diethylcarbamazine, which is also the main treatment. Churg-Strauss syndrome: a pANCA positive small to medium vessel vasculitis that presents with a refractory asthma type of picture, mononeurtitis multiplex (look out for e.g foot drop in a stem that sounds like asthma), and sinusitis. Biopsy of affected tissue is helpful diagnostically. Management is largely with steroids but can progress to the need for steroid-sparing agents such as cyclophosphamide/azathioprine. Drugs-induced pulmonary eosinophilia: sulphonamides (e.g. sulfasalazine), nitrofurantoin, penicillin, tetracycline. Stop the offending drug.

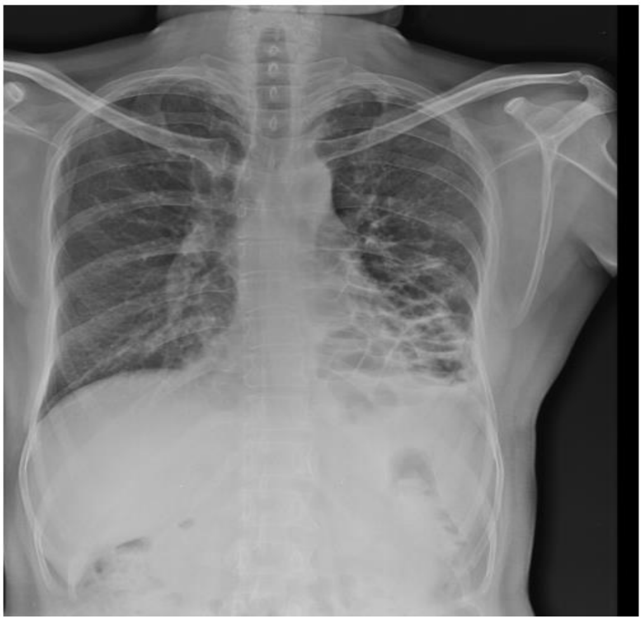
Question 54 of 231

A 46-year-old man presents with a persistent cough. This has been present for around 2 months and is productive of yellow-green sputum. He feels more short-of-breath on exertion but is experiencing chest pain. He has not lost any weight and has not coughed up blood.

He has a 25-pack-year history of smoking although is trying to give up. He does not drink alcohol.

On examination his respiratory rate is 18/min, heart rate 84/min, blood pressure 114/72 mmHg and temperature 37.3ºC. His oxygen saturations on room air are 96%. Scattered crackles are heard in both bases.

A chest x-ray is requested:



What is the most likely diagnosis?

A. Pneumonia
B. Sarcoidosis
C. Chronic obstructive pulmonary disease
D. Bronchiectasis
E. Pulmonary fibrosis

D. Bronchiectasis

The chest x-ray has classical changes of bronchiectasis - tramlines are seen, most prominently in the left lower zone.

Question 55 of 231

A 66 year old gentleman, who is a known heavy smoker, presents with a non infective exacerbation of chronic obstructive pulmonary disease (COPD). Upon admission despite maximal medical therapy his blood gasses worsen and he is commenced on non invasive ventilation and transferred to the high dependency unit. The nurses call you in the middle of the night to come and urgently review the patient. They have been titrating up the patient's non invasive ventilation (NIV) as they felt his blood gasses were not improving on standard settings. On arrival the patient is diaphoretic and complaining of chest pain. He is unable to tolerate his face mask. Observations are recorded as a pulse of 110/min regular, respiratory rate 32/min, Sats 75% on air, Blood pressure 100/82 mmHg.

What is the most likely cause for this patient's acute deterioration?

A. Ventilator associated pneumonia
B. Acute myocardial infarction

C. Ventilator associated pneumothorax
D. Worsening of his COPD
E. Panic attack

C. Ventilator associated pneumothorax

This question assesses candidates ability to recognise some of the complications of NIV. This patient has developed a pneumothorax secondary to the increasing pressures used as part of his NIV. Although less common these days due to nurse training, ventilation associated pneumothorax is an important complication of NIV to be aware of and therefore, prompt assessment and treatment will be essential in the management of this patient.

Ventilator associated pneumonia tends to present in patients who have been ventilated for long a long period of time and would not present so acutely.

Acute myocardial infarction would be unlikely as the question makes a point of mentioning the increasing pressures that are needed.

Candidates should also familiarise themselves with some of the contraindications to NIV:

- Recent facial or upper airway surgery or facial injuries.
- Recent upper gastrointestinal surgery.
- Confusion/agitation.
- Bowel obstruction.
- Upper airway obstruction.
- Excessive upper airway secretions.
- Patient actively vomiting

Question 56 of 231

A 66-year-old man, presented with two episodes of mild haemoptysis in the past 3 months. He also had lost 5 kilograms of weight in the past 3 months. He was a smoker with 50 pack years history and a social drinker. He was actively mobile and had no significant past medical history.

Clinical examination revealed no abnormality except clubbing.

A contrast enhanced CT chest showed a 4 cm mass in the right upper lobe periphery close to the chest wall and enlarged right hilar (2 cm) and subcarinal (2 cm) lymph nodes. A positron emission tomography (PET) scan was done which showed a standard uptake value (SUV) max of 20 for the lung mass. The ipsilateral hilar and subcarinal lymph nodes were also found to be FDG (fluorodeoxyglucose) avid. There was no evidence of distant metastasis.

What is the next appropriate step in the management?

A. CT guided biopsy of the lung mass
B. Ultrasound guided biopsy of the lung mass
C. Flexible bronchoscopic biopsy of the lung mass
D. Endobronchial ultrasound (EBUS) guided mediastinal lymph node sampling
E. Video-Assisted Thoracoscopic Surgery (VATS) biopsy of the mediastinal lymph nodes

D. Endobronchial ultrasound (EBUS) guided mediastinal lymph node sampling

This is probably a stage IIIA (T2aN2M0) lung cancer. Diagnosis and staging has to be confirmed by tissue sampling. EBUS guided mediastinal lymph node sampling provides diagnosis and staging in a single step whereas CT guided or ultrasound guided or bronchoscopic biopsy of the lung mass yields only tissue diagnosis (not staging). Wherever possible minimally invasive procedures should be undertaken first for mediastinal node sampling (e.g., EBUS) before embarking into more invasive procedures like VATS.

Question 57 of 231

A 17-year-old male is admitted to the resuscitation area of the emergency department with an acute exacerbation of asthma. It is his 3rd hospital attendance this winter. He has widespread wheeze, globally poor air entry and looks tired. He cannot complete full sentences. His arterial blood gases reveal:

pH	7.4
PaO2	9.3 kPa
PaCO2	3.3 kPa
HCO3-	26 mmol/L

You treat him with 100% oxygen, high dose steroids, back to back salbutamol nebulisers and commence intravenous magnesium sulphate infusion. 1 hour later his arterial blood gases reveal:

pH	7.35
PaO2	15.4 kPa
PaCO2	4.7 kPa
HC03-	22 mmol/L

What is the next management step?

A. Start BIPAP
B. Intensive care review
C. Move to medical ward with close monitoring
D. Intravenous adrenaline
E. Start CPAP

B. Intensive care review

Rising CO2 is an indication that the patient is becoming tired. Oxygenation has only mildly improved despite 100% oxygen. Patients with life-threatening features of asthma do not always appear distressed. The patient will likely require mechanical ventilation.

Question 58 of 231

An 83-year-old male presents with his third admission of right lower zone community-acquired pneumonia in 5 months. A CT thorax demonstrates a 2.2cm mass in right lower lobar bronchus with no regional lymph nodes. Bronchoscopy reveals non-small cell lung Ca 4cm from the carina, CT staging reveals no other metastases. A final staging diagnosis of T1b N0 M0 is made, at stage 1A. The patient undergoes lung function testing as follows:

FVC	2.2l
FEV1	1.7l/s
TLCO	42% of predicted

What is the most appropriate treatment?

A. Right pneumonectomy
B. Right lower lobectomy
C. Chemotherapy
D. Radiotherapy
E. Palliative care

B. Right lower lobectomy

The patient has been diagnosed with surgically resectable non-small cell lung carcinoma (NSCLC). All NSCLC with stage I or II disease should undergo resection surgery where possible, stage III disease generally is focused on chemotherapy while stage IV disease is managed with symptomatic palliative treatments. However, while the disease burden may be amenable to surgery, the patient's lung function testing may restrict management options. Multiple studies have demonstrated FEV1 and transfer coefficients to be the most accurate predictors of postoperative dyspnoea, with the British Thoracic Society recommending the patient to demonstrate FEV1 >2l/s for pneumonectomy and 1.5l/s for lobectomy. TLCO is similarly recommended to be greater than 40% of predicted for resective surgery1. Forced vital capacity is not demonstrated to be a significant predictor of post-operative outcomes.

Question 59 of 231

A 39-year-old lady presented to the emergency department acutely unwell with fever, weight loss and malaise. She had been finding it increasingly difficult to mobilise in recent weeks due to weakness in her right ankle. She had diarrhoea and was passing loose brown stool up to five times a day. She had a previous medical history of asthma and sinusitis. Her medications included a salbutamol inhaler, salmeterol inhaler, montelukast and intermittent courses of prednisolone and antibiotics as required.

On examination, she appeared unwell, had petechial purpura in the nail beds of her fingers and on her feet, expiratory wheeze on auscultation of her chest, a soft abdomen with no palpable masses and weakness of dorsiflexion of her right ankle. Her temperature was 37.4 degrees Celsius, her pulse was 115 beats per minute and regular, her blood pressure was 140/95 mmHg, her respiratory rate was 24 breaths per minute and her oxygen saturations were 94% on room air.

Investigations:

Urine dip: blood++, protein+

Haemoglobin	95 g/L
White cell count	23.6 x10^9 /L
Platelet Count	181x10^9/L
Neutrophils	12.3 x10^9/L
Eosinophils	9.6 x10^9/L
Basophils	0.2 x10^9/L
Lymphocytes	1 x10^9/L
Monocytes	0.5 x10^9/L
Erythrocyte sedimentation rate	65mm/hr
INR	0.9

Serum sodium	138mmol/L
Serum potassium	4.6mmol/L
Serum urea	15.4mmol/L
Serum creatinine	220micromol/L

Antinuclear antibody	negative
C-terminus antineutrophil cytoplasmic antibodies	negative
Myeloperoxidase anti-neutrophil cytoplasmic antibodies	positive

What is the most likely diagnosis?

A. Infective endocarditis
B. Wegener's granulomatosis
C. E.Coli gastroenteritis
D. Churg-Strauss syndrome
E. Crohn's disease

D. Churg-Strauss syndrome

This lady with a history of asthma and sinusitis has presented systemically unwell with diarrhoea, a right common peroneal nerve palsy, vasculitic rash and has evidence of a nephritis. Investigations show a significant eosinophilia and immunological screen has shown she is P-ANCA positive. The most likely diagnosis is, therefore, Churg-Strauss syndrome. This is a small vessel vasculitis with eosinophilic infiltration and granulomatous formation that is characterised by chronic sinusitis, asthma and eosinophilia. Systemic features of malaise, myalgia, fever and weight loss are common. Other symptoms and signs depend on other organ systems involved which most commonly are the lung (asthma, pneumonitis) and skin (purpura, subcutaneous nodules); however, any organ system can be affected including the cardiovascular (heart failure, myocarditis), gastrointestinal (gastroenteritis, ischaemia, bleeding), renal (glomerulonephritis, hypertension), and central nervous systems (mononeuritis multiplex). ANCA is positive in approximately 60% of cases and is predominantly P-ANCA.

Infective endocarditis could account for some of her symptoms however the absence of a heart murmur and the multisystem involvement, in this case, makes it less likely. Wegener's granulomatosis is another granulomatous small vessel vasculitis which most commonly presents with lesions in the upper respiratory tract (rhinorrhoea, sinusitis, saddle nose deformity), lungs (infiltrates and pulmonary haemorrhage) and kidneys (rapidly progressive glomerulonephritis). It is classically C-ANCA positive. The history of immunological screen of this case is, therefore, more typical of Churg-Strauss syndrome. E.coli gastroenteritis would be unlikely to present in this fashion.

Crohn's disease is a chronic inflammatory bowel disease that classically presents with abdominal pain, weight loss and change in bowel habit. There are often extra-intestinal manifestations involving the mouth, skin, eyes, joints and episodes of perianal abscess or anal fissure. Crohn's disease could account for some of this lady's symptoms but again is less likely than Churg-Strauss syndrome.

Question 60 of 231

A 70-year-old gentleman is admitted with three days of shortness of breath and arrives to the emergency department where he is started on non invasive ventilation for a suspected infective exacerbation of COPD.

On examination, he is in respiratory distress with cyanosis and a retention flap. He has widespread wheeze throughout and crepitation at the right base. There is no oedema and he has a capillary refill time of 3 seconds. Blood pressure 90/65mmHg, respiratory rate 24 breaths per minute, oxygen saturations are 84% on arrival and rise to 92% with 28% oxygen.

He is treated with full maximal therapy but fails to improve. He appears comfortable with the mask on and there is hissing heard with each inflation breath. The machine is set to 12cmH2O IPAP and 4cmH2O EPAP but the pressures recorded are 8cmH2O IPAP and 3cm H2O EPAP.

ABG	on arrival
pH	7.32
pCO2	8.2kPa
pO2	7.1 kPa
HCO3	26mmol/l
Lactate	3.02 mmol/l
ABG	current
pH	7.31
pCO2	8.3kPa
pO2	8kPa (on FiO2 0.28)
HCO3	26mmol/l
Lactate	2.78 mmol/l
Chest x-ray	hyperexpanded lung fields, consolidation on right middle zone

What is the most likely explanation of the failure to improve?

A. Carbon dioxide narcosis
B. Dyssynchrony
C. Mucus plugging
D. Mask leak
E. Pneumothorax

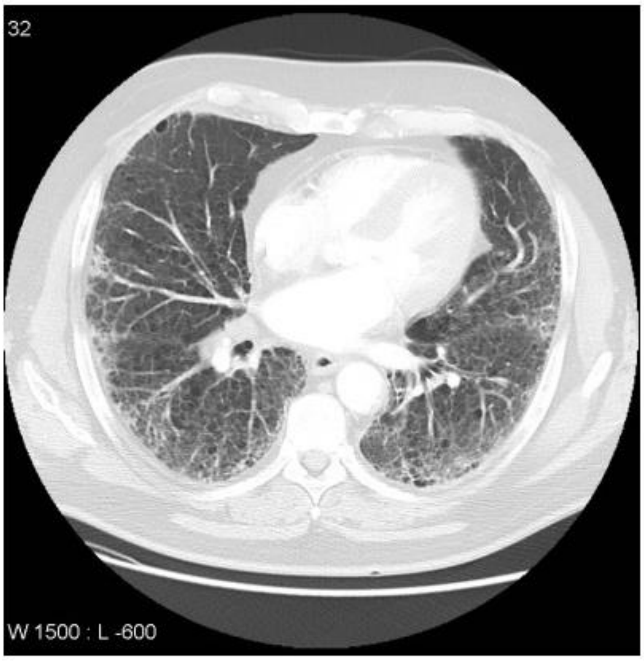
D. Mask leak

Significant air leak from the mask will prevent adequate pressures being achieved with noninvasive ventilation

The patient has a clear COPD exacerbation and starts non invasive ventilation but fails to improve. The clues are the audible hiss of air that can be heard from the mask and the failure of the machine to achieve the pressures set. Poor mask application or facial dysmorphia can lead to a poor seal to the face. Air will leak and the pressure will fall with this. The patient is unlikely to have deteriorated due to reducing consciousness secondary to CO2 narcosis as he continues to breathe at an elevated respiratory rate. There is no suggestion of a pneumothorax and this usually presents as a deterioration rather than a failure to improve. The patient is comfortable and does not appear to be fighting the machine as in dyssynchrony. A large mucus plug usually presents with sudden desaturation which is not seen here.

Question 61 of 231

A 59-year-old man is investigated for dyspnoea. Over the past 12 months he has become more breathless on exertion and has been troubled by a cough. He stopped smoking 10 years ago but prior to this had a 30-pack-year history. There is no history of chest pain, fever, night sweats or asbestos exposure. A CT chest is requested and shown below:



What does the CT scan show?

A. Lung cancer
B. Idiopathic pulmonary fibrosis
C. Multiple pulmonary embolisms
D. Bronchiectasis
E. Emphysema

B. Idiopathic pulmonary fibrosis

The CT scan shows advanced pulmonary fibrosis including 'honeycombing'.

Question 62 of 231

A 75-year-old man with a background of chronic kidney disease, hypertension and type 2 diabetes presents to the Emergency Department with cough and breathlessness on minimal exertion. On examination, he has bilateral inspiratory crepitations and bilateral pitting oedema up to his sacrum. His chest radiograph shows a moderate right-sided effusion and smaller left-sided effusion.

What single management would be most appropriate?

A. Fluid restriction to 1L daily
B. IV furosemide
C. Oral spironolactone
D. Referral for dialysis
E. Therapeutic aspiration of right-sided effusion

B. IV furosemide

In the assessment of a pleural effusions, if the clinical picture suggests a transudate, then you should treat the cause initially. If this does not resolve the effusion then perform a diagnostic aspiration

This patient presents with signs and symptoms of fluid overload, possibly due to worsening chronic kidney disease. While he has bilateral pleural effusions, they are most likely transudative and initial management should be to offload with **IV diuretics such as furosemide**. This may lead to the resolution of his fluid overload and pleural effusions.

Fluid restriction to 1-1.5L daily would be appropriate as part of medical management. It should be recommended alongside diuresis with medication as on its own it would not lead to significant offloading in a patient who is significantly fluid overloaded.

Oral spironolactone is a diuretic that would help to offload the patient. However, it is not commonly used as a first-line diuretic in a patient who is significantly fluid overloaded. It also cannot be given intravenously. Intravenous diuresis may be clinically more effective as patients who are significantly fluid overloaded may also have gut oedema and thus have poor absorption of oral diuretics.

The patient may need to eventually be **referred for dialysis** if they have end-stage renal disease. However, many patients with chronic kidney disease may be safely treated with diuretics without their renal function significantly worsening. IV diuresis should thus be attempted first. Should this fail, a renal physician could be consulted for advice, including regarding dialysis.

Therapeutic aspiration of the larger right-sided effusion involves aspirating up to 1.5L of fluid or inserting a chest drain to reduce the size of the effusion. This may relieve symptoms but would be unlikely to lead to the resolution of the contralateral effusion or improvement of this patient's peripheral oedema. If there are atypical features, or the patient has failed to respond to treatment such as with IV diuretics, he can be referred for therapeutic aspiration.

Question 63 of 231

A 66-year-old gentleman presented with a 4 day history of cough associated with green sputum. He has also been feeling more breathless, wheezy, feverish at times and having some right sided pleuritic chest pains. His past medical history includes chronic obstructive pulmonary disease, hypertension and diabetes. He has a 40 pack year history of smoking but he stopped 3 years ago.

On examination, he looks unwell, flushed and breathless at rest. His heart sounds are normal and he has right basal crackles with bronchial breathing and wheeze. There is no leg oedema or tenderness. His vital signs show pulse of 120 beats per minute, blood pressure of 120/70 mmHg, SaO2 = 89% on 24% oxygen, respiratory rate of 32 breaths per minute and T=38.5oC.

His Electrocardiogram reveals sinus tachycardia and the chest X-ray confirms right basal consolidation.

The following bloods tests have been obtained:

Hb	13.1 g/dl
Platelets	180 * 10 /l9
WBC	15.4 * 10 /l9
Na +	135 mmol/l
K +	4.9 mmol/l
Urea	10 mmol/l
Creatinine	120 µmol/l
CRP	180 mg/l
pH	7.28
pCO2	5.0 kPa
pO2	8.5 kPa
HCO3-	15 mEq/l

What is the best management?

A. Intravenous fluids, intravenous antibiotics, steroids, nebulisers and non-invasive ventilation
B. Intravenous fluids, intravenous antibiotics, nebulisers and steroids
C. Intravenous fluids, oral antibiotics, nebulisers and steroids
D. Oral fluids, intravenous antibiotics, nebulisers and non-invasive ventilation
E. Oral fluids, oral antibiotics, nebulisers and steroids

B. Intravenous fluids, intravenous antibiotics, nebulisers and steroids

The aim of this question aims to increase awareness about the use of non-invasive ventilation (NIV) in patients with chronic obstructive pulmonary disease (COPD).

The guidelines mention that NIV should be considered in patients with COPD exacerbation in whom a respiratory acidosis persists despite immediate maximum standard medical treatment on controlled oxygen for no more than one hour. Patients with COPD can develop community acquired pneumonia (CAP) with metabolic acidosis due to the sepsis, but in these cases NIV is not indicated.

This patient presented with CAP and he had metabolic acidosis. His CURB65 score is 3 and he also has acute kidney injury. He should be treated with intravenous fluids, intravenous antibiotics, nebulisers and steroids but not NIV.

Question 64 of 231

A 69-year-old man with a history of rheumatoid arthritis well controlled on methotrexate presents with gradual pain and swelling in his wrists and ankles.

The pain is described as a dull ache that is intermittent, often worse in the evenings. There is associated swelling which can sometimes feel warm. He has tried regular paracetamol but this has had limited effect.

He also describes a chronic cough and shortness of breath on exertion for the past 6 months that he has not mentioned to his GP. There has been no haemoptysis and he denies any fevers. His wife has noticed that he has been losing weight recently.

He has a past medical history of rheumatoid arthritis, hypertension, hypercholesterolaemia and type 2 diabetes mellitus. He currently takes methotrexate weekly, folic acid 5mg weekly, ramipril 5mg, simvastatin 20mg at night, metformin 500mg three times a day and paracetamol 1g four times daily.

He is a retired accountant and a current smoker with a 50 pack year smoking history. He drinks approximately 30 units of beer a week. He denies any recent foreign travel.

On examination, he is cachectic and short of breath on exertion. His pulse is 80/min and regular, blood pressure 140/93 mmHg, oxygen saturations of 93% on air. He has marked fingernail clubbing. Examination of his wrists reveals slightly swollen and tender joints.

Swan neck and ulnar deviation deformities are noted in both hands. Other than his wrists, no other joint abnormalities are detected. Examination of the peripheral nervous system is normal.

Examination of his chest is normal with no focal consolidation.

Initial bloods are as follows:

Na+	134 mmol/L
K+	3.9 mmol/L
Urea	7.8 mmol/L
Creatinine	105 µmol/L
Hb	100 g/L
WBC	6.0x10^9/L
Platelets	200x10^9/L

Chest X-ray reveals a discrete opacification at the periphery of the right middle lobe.

What is the most likely underlying diagnosis?

A. Methotrexate induced pulmonary fibrosis
B. Bronchogenic carcinoma
C. Caplan's syndrome
D. Chronic obstructive pulmonary disease
E. Pneumonia

B. Bronchogenic carcinoma

This man has a strong smoking history, chronic cough, weight loss and an abnormal chest x-ray. He also has symptoms suggestive of hypertrophic pulmonary osteoarthropathy which is associated with bronchogenic carcinoma in 90% of cases. His case would warrant urgent investigation.

Pneumonia is unlikely in the context of normal inflammatory markers

Methotrexate-induced fibrosis is unlikely in view of the chest x-ray findings and normal auscultation of the chest.

COPD may be a concomitant diagnosis in a man with such an extensive smoking history, but the presence of weight loss and hypertrophic pulmonary osteoarthropathy should raise suspicions of the more pressing diagnosis of malignancy.

Caplan's syndrome is an interstitial lung disease described in coal miners with rheumatoid arthritis. Chest x-ray usually demonstrates multiple nodules often at the lung peripheries.

Question 65 of 231

You are asked to review the results of a long term oxygen therapy (LTOT) assessment for a 74- year-old female. She has a past medical history of COPD. She has developed dyspnoea at rest.

Results of the LTOT assessment are as follows: ABG on air:

PaO2	7.1 kPa
PCO2	5.1 kPa
pH	7.41
HCO3	26

ABG after 1 litre of O2 via a nasal cannula:

PaO2	9.3 kPa
PCO2	6.5 kPa
pH	7.33
HCO3	26

The patient feels less short of breath at the end of the trial.

You decide that further medical optimisation and reassessment of arterial blood gas in 4 weeks is required.

She returns to the clinic in 4 weeks time. Her COPD appears to be stable and the medical management is optimised with salbutamol, ipratropium and carbocysteine.

A repeat LTOT assessment is as follows:

ABG on air:

LFTs	Normal
Serum uric acid	410 µmol/L
CRP	12 mg/L
PaO2	6.9 kPa
PCO2	5.0 kPa
pH	7.42
HCO3	26

ABG after 1 litre of O2 via a nasal cannula:

PaO2	9.5 kPa
PCO2	6.6 kPa
pH	7.32
HCO3	26

How will you manage this patient?

A. LTOT with nocturnal BiPAP
B. LTOT
C. LTOT with nocturnal CPAP
D. Decline LTOT
E. Palliation of breathlessness with morphine

A. LTOT with nocturnal BiPAP

Patients who develop a respiratory acidosis and/or a rise in PaCO2 of >1 kPa (7.5 mmHg) during an LTOT assessment on two repeated occasions, while apparently clinically stable, should only have domiciliary oxygen ordered in conjunction with nocturnal ventilatory support

The ABG on room air meets the criteria for LTOT as the PaO2 is < 7.3 kPa. After a 1 hour trial of low flow oxygen, the patients symptoms have improved, and his PaO2 has significantly improved. However it is important to note that his PCO2 has significantly increased resulting in a respiratory acidosis.

Patients who develop a respiratory acidosis and/or a rise in PaCO2 of >1 kPa (7.5 mmHg) during an LTOT assessment may have clinically unstable disease. These patients should undergo further medical optimisation and be reassessed after 4 weeks.

The patient is medically optimised and returns after 4 weeks for further LTOT assessment. This again demonstrates a respiratory acidosis with oxygen therapy. Patients who develop a respiratory acidosis and/or a rise in PaCO2 of >1 kPa (7.5 mmHg) during an LTOT assessment on two repeated occasions, while apparently clinically stable, should only have domiciliary oxygen ordered in conjunction with nocturnal ventilatory support. BiPAP is the modality of choice for treating CO2 retention.

Question 66 of 231

A 67-year-old man presents to the emergency department with a 24-hour history of shortness of breath. Also, he complains of a worsening cough with green sputum. He has a 20 pack-year smoking history and drinks 10 units of alcohol a week. He has a past medical history of COPD, for which he takes inhaled corticosteroids, formoterol and salbutamol.

On auscultation of his chest, there are crackles at the right base, no wheezes. He has a respiratory rate of 23 breaths per minute, a heart rate of 98bpm, BP is 115/62mmHg, SpO2 is 91% on 4L through a 28% Venturi mask, and he is afebrile.

A chest X-ray shows right lower zone consolidation. An arterial blood gas shows the following:

HCO3	28	(22-26 meq/L)
pH	7.37	(7.35 - 7.45)
pCO2	6.2	(4.5 - 6.0 kPa)
pO2	9.8	(10 - 14 kPa)

You have prescribed appropriate IV fluids and administered IV co-amoxiclav and clarithromycin.

Which of the following is the most appropriate additional treatment?

A. BIPAP
B. Decrease oxygen
C. Increase oxygen
D. Prednisolone
E. Prednisolone and increase oxygen

D. Prednisolone

Patients diagnosed with pneumonia who have COPD should be given corticosteroids even if no evidence of the COPD being exacerbated

This man is suffering from community-acquired pneumonia (CAP). Patients diagnosed with pneumonia who have COPD should be given corticosteroids even if no evidence of the COPD being exacerbated.

We do not routinely give non-invasive ventilation (NIV) or continuous positive airways pressure (CPAP) support in patients with respiratory failure due to CAP, though it ought to be considered if there is evidence of type 2 respiratory failure.

If not critically ill and history of moderate-severe COPD or other condition predisposing to hypercapnia (e.g. neuromuscular disease) then aim for 88-92% with a 28% Venturi mask initially. Then do a blood gas:

- If pH<7.35 and pCO2 >6KPa then consider NIV
- If pH>7.35 and pCO2 >6KPa then continue with 88-92% with a repeat in 30-60mins
- If pH>7.35 and pCO2 <6KPa titrate O2 to sats 94-98% with a repeat in 30-60mins

Decreasing his oxygen supply would not be appropriate as the single best answer, as his oxygen sats are in an appropriate range and he also requires corticosteroids.

Essentially, this man likely has a degree of chronic CO2 retention, as indicated by the fact that he has a compensatory metabolic alkalosis which has managed to maintain his pH within a normal range. Thus increasing his oxygen at this point is not necessary, as he may well be relying on his low O2 to drive his respiratory rate, and a target of 88-92% is satisfactory. As he is not acidotic NIV is not indicated at present.

Question 67 of 231

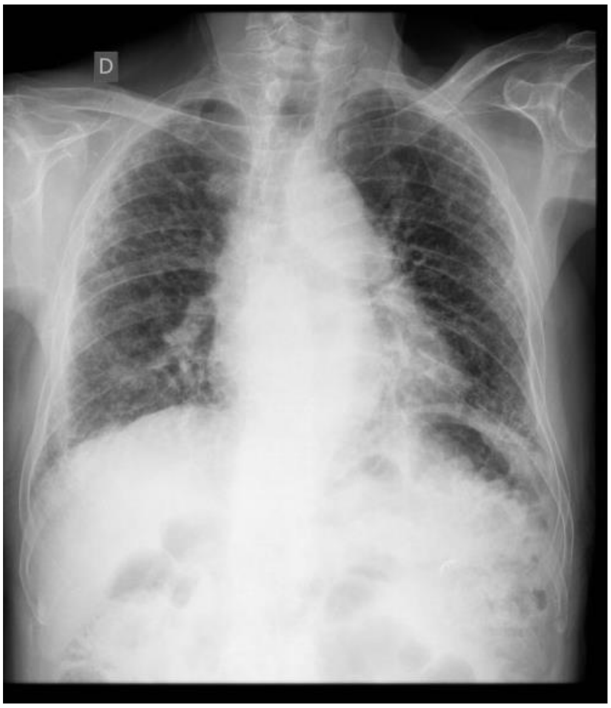
A 59-year-old man is investigated for increasing shortness-of-breath. This has been getting progressively worse for the past 6 months and is associated with a dry cough. He has received two courses of antibiotics from his GP which only temporarily improved his symptoms. He was diagnosed with asthma as a child but has not had to use an inhaler since he was around 10-yearsold. He stopped smoking 20 years ago.

On examination he is noted to have scattered crackles bilaterally, predominantly in the bases. Oxygen saturations are 96% on room air. Pulse, blood pressure and temperature are normal.

Spirometry is arranged:

FEV1	2.2 L (predicted 3.8 L)
FVC	2.7 L (predicted 4.5 L)
FEV1/FVC	81% (normal > 75%)

A chest x-ray is arranged:



Which one of the following interventions is most likely to be beneficial?

A. Bosentan
B. Rotation of prophylactic antibiotic + postural drainage
C. Bronchodilator therapy
D. Prednisolone
E. Pirfenidone

E. Pirfenidone

The spirometry shows a restrictive picture consistent with pulmonary fibrosis. The chest x-ray is also consistent with this diagnosis, showing sub-pleural reticular opacities that increase from the apex to the bases of the lungs.

Pirfenidone is currently the only drug recommended by NICE for the treatment of idiopathic pulmonary fibrosis.

Question 68 of 231

A 45-year-old man presents to the emergency department with a two-week history of a dry cough, fever and retrosternal discomfort. He has a past medical history of HIV and is poorly compliant with anti-retroviral medications. He has recently returned from a holiday in Mississippi.

Observations:

- Heart rate 95 beats per minute
- Blood pressure 101/65 mmHg
- Respiratory rate 24/minute
- Spo2 93% on room air
- Temperature 37.3C

The examination is unremarkable.

A chest x-ray demonstrates multifocal consolidation that extends to the periphery of the lungs. Sputum microscopy reveals yeasts.

What organism is most likely responsible for this presentation?

A. Coccidioides posadasii
B. Histoplasma capsulatum
C. Legionella pneumophila
D. Pseudomonas aeruginosa
E. Streptococcus pneumoniae

B. Histoplasma capsulatum

Mississippi and Ohio River valleys → ?histoplasmosis

Histoplasma capsulatum is the correct answer. The patient presents with a dry cough, retrosternal discomfort, fever and chest x-ray evidence of multifocal pneumonia after travelling from a region where histoplasmosis is endemic. Yeasts identified on sputum microscopy are also uggestive of a fungal rather than bacterial cause. Severe histoplasmosis is more common in immunocompromised patients. His history of poor compliance with HIV medications suggests he is at further risk of developing severe fungal infections. Histoplasmosis is often a self-limiting illness in an immunocompetent individual.

Coccidioides posadasii is incorrect. This causes coccidioidomycosis, another endemic mycosis. While infection with this fungal organism can cause respiratory failure and multifocal consolidation in patients with HIV, it is not endemic to Mississippi. It is found in more arid regions including southern California, southern Nevada, Arizona, New Mexico, Utah and western Texas. it is also found in parts of South America and Central America.

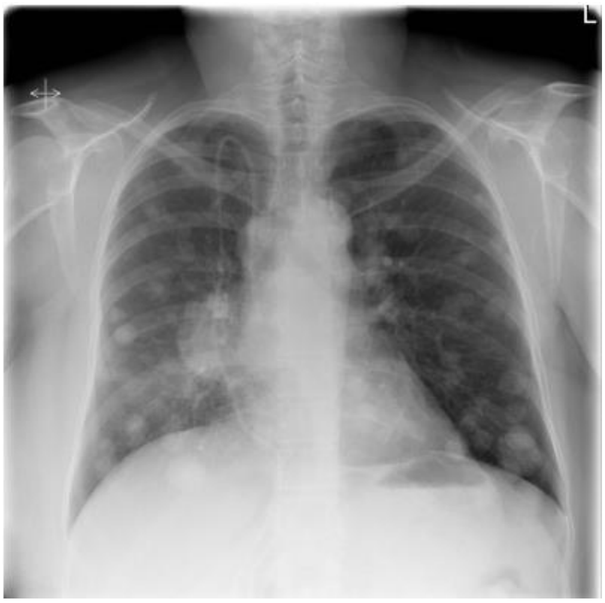
Legionella pneumophila is incorrect. This is a cause of a bacterial atypical pneumonia. It is typically transmitted by inhaling contaminated water droplets and so outbreaks have been linked to swimming pools and air conditioning systems. It is not necessarily endemic to Mississippi and sputum microscopy would not demonstrate a yeast, which implies a fungal infection.

Pseudomonas aeruginosa is incorrect. This is another potential cause of pneumonia in a patient with HIV and probable low CD4 count. However, yeasts would not be seen on sputum microscopy and this indicates a fungal infection. *Pseudomonas* infections are also more common in patients with pre-existing lung disease and indwelling vascular access.

Streptococcus pneumoniae is incorrect. This is one of the most common causes of pneumonia. However, this typically causes lobar rather than multifocal pneumonia. Additionally, the sputum sample may confirm that organism rather than grow a yeast.

Question 69 of 231

A 68-year-old woman is admitted to the Emergency Department with shortness of breath. A chest x-ray is taken as part of the initial investigations.



Based on the chest x-ray, what is the most likely diagnosis?

A. Multiple pulmonary emboli
B. Tuberculosis
C. Multiple lung abscesses
D. Bronchiectasis
E. Lung metastases

E. Lung metastases

The multiple rounded lesions are highly suggestive of lung metastases.

Question 70 of 231

A 50-year-old female patient presents to the respiratory outpatient clinic for her asthma review.

Unfortunately, she has been admitted to hospital three times in the last year with asthma exacerbations, requiring 2-3 days in hospital but never requiring intubation or intensive care admission. This is on a background of multiple admissions to hospitals in previous years.

On review, she reports wheeze associated with dyspnoea on most days and this is usually triggered by exertion. There is a non-productive cough most mornings which settles throughout the course of the day. There is no history of allergic rhinitis, eczema or other medical problems. She is a lifelong non-smoker.

Current asthma therapy is high dose inhaled fluticasone propionate plus salmeterol 2 puffs twice daily, prednisolone 10mg once daily and inhaled salbutamol as required. She has been taking regular prednisolone 10mg for the last eighteen months.

On examination, observations revealed a respiratory rate of 14/min, oxygen saturation 98% on room air, heart rate 80/min regular, blood pressure 130/70 mmHg and a temperature of 36.8ºC. There is no clubbing, cervical lymphadenopathy or elevation of the jugular venous pressure. Auscultation of the chest revealed dual heart sounds with no murmurs and some mild expiratory wheeze in the upper zones. The calves were soft and non-tender, with no pedal oedema.

You had reviewed this patient during her most recent exacerbation and had arranged some outpatient tests, the results of which are shown below:

Hb	140 g/l	Na +	138 mmol/l
Platelets	350 * 10 /l 9	K +	3.4 mmol/l
WBC	8 * 10 /l 9	Urea	5 mmol/l
Neuts	4.5 * 10 /l 9	Creatinine	70 µmol/l
Lymphs	1.0 * 10 /l 9	CRP	7 mg/l
Eosin	2.5 * 10 /l9		
Fraction of exhaled nitric oxide	65 parts per billion (upper limit of normal 50 ppb)		

Which of the following would be the most appropriate management for this patient?

A. Add in mepolizumab
B. Start a course of voriconazole
C. Increase dose of prednisolone to 20mg once daily
D. Start home nebulisers
E. High resolution computed tomography of the thorax (HRCT)

A. Add in mepolizumab

Mepolizumab and other anti-IL-5 therapies can be used in adults with eosinophilic asthma

This patient has inadequately controlled asthma as evidenced by the three admissions to hospital and ongoing cough, wheeze and dyspnoea. There is a serum eosinophilia and elevated fraction of exhaled nitric oxide (FENO), both suggesting eosinophilic inflammation as an important driver of this patient's asthma. Such patients are eligible for anti-interleukin-5 therapy such as mepolizumab.

Mepolizumab administered either intravenously or subcutaneously has been shown to significantly reduce asthma exacerbations and is particularly useful in patients on long-term steroid therapy.

There are several inflammatory phenotypes in asthma most commonly described as eosinophilic, neutrophilic, mixed, and paucigranulocytic. The presence of eosinophilic inflammation can be demonstrated by one or more of the following:

- Sputum Eosinophils 3% or more
- FENO of 50 ppb or more
- Eosinophil count of 0.3X109/L or higher
- Prompt deterioration of asthma control after a 25% or less reduction in inhaled corticosteroid dose

Studies have shown an association between higher levels of FENO and eosinophilic airway inflammation, although the mechanism by which this occurs is still unclear.

Option A - the correct answer

Option B - there is insufficient evidence to support a diagnosis of allergic bronchopulmonary aspergillosis (ABPA) and therefore a course of voriconazole is not indicated

Option C - Another advantage of mepolizumab if that is has been shown to improve asthma symptoms to such an extent that steroid-dependent patients can actually cease their oral corticosteroid therapy. Given the significant side effects long-term corticosteroid therapy can have, increasing the steroid dose further is not the most appropriate management step, especially when there is a treatment that specifically targets this patient's asthma phenotype

Option D - home nebulisers would not treat the underlying problem of eosinophilic asthma Option E - There is no indication for HRCT in this case.

Question 71 of 231

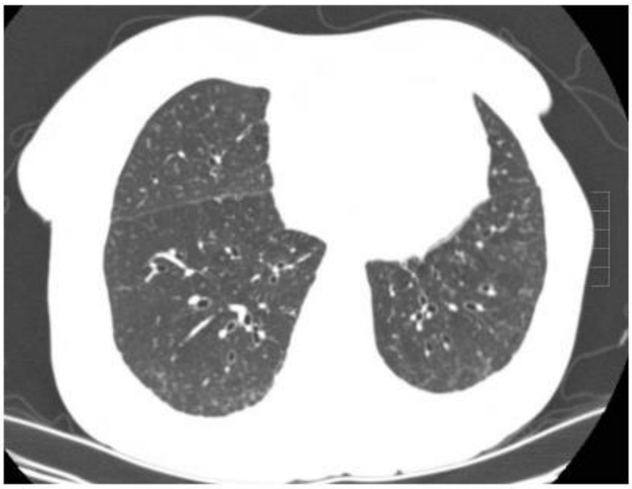
A 41-year-old woman is seen in the respiratory clinic with a 4-month history of a productive cough and shortness of breath on exertion. There is no history of fever or weight loss although she does describe feeling more fatigued. Her past medical history includes coeliac disease and she smokes 10 cigarettes/day.

Her observations are recorded as follows:

- Temperature 36.1ºC
- Heart rate 87bpm
- Blood pressure 114/67mmHg
- Respiratory rate 15 breaths/min
- Saturations 94% on room air

On examination, there is evidence of finger clubbing. On auscultation, bilateral coarse crackles are heard. Heart sounds are normal and her abdominal examination is unremarkable.

A high resolution computed tomography (HRCT) chest is requested:



What is the most likely diagnosis?

A. Mycobacterium tuberculosis
B. Bronchiectasis
C. Chronic obstructive pulmonary disease
D. Pneumocystis jiroveci pneumonia
E. Pulmonary fibrosis

B. Bronchiectasis

The diagnosis here is bronchiectasis. Bronchiectasis is the term given to a respiratory condition characterised by the permanent dilatation of the airways. This transverse slice of a CT chest shows multiple dilated bronchi, in keeping with the diagnosis. Causes of bronchiectasis include chronic infection and inflammation for example cystic fibrosis or following treatment for mycobacterium tuberculosis. This patient has had recurrent respiratory tract infections which may be involved in the pathogenesis of bronchiectasis in this patient. Symptoms include a persistent productive cough, shortness of breath and haemoptysis. Coarse crackles may be heard on auscultation and finger clubbing may be present.

Mycobacterium tuberculosis is a bacterial infection characterised by fever, lethargy and a productive cough. Constitutional symptoms such as weight loss and night sweats may also be seen. Whilst chronic inflammation secondary to *Mycobacterium tuberculosis* is a cause of bronchiectasis it is unlikely to be the current diagnosis as this patient has no history of fever or constitutional symptoms.

Chronic obstructive pulmonary disease (COPD) is most commonly secondary to tobacco use and a history of regular smoking can increase the likelihood of the disease. Examination findings tend to include wheeze rather than coarse crackles and COPD is not a cause of finger clubbing, suggesting an alternative diagnosis. Findings in keeping with COPD on a CT scan vary from person to person and include features such as bronchial wall thickening, air-space enlargement and bullae formation.

Pneumocystis jiroveci pneumonia is a serious fungal infection characterised by fever, cough, shortness of breath, chest pain and fatigue. It is most commonly seen in patients with immunosuppression. Autoimmune conditions are associated with coeliac disease, but coeliac disease itself should not give rise to significant immunosuppression.

Pulmonary fibrosis presents as progressive shortness of breath and a dry cough. It is also a cause of finger clubbing. Pulmonary fibrosis can be diagnosed on a CT scan by the presence of the characteristic honeycomb appearance which is not seen here.

Question 72 of 231

A 65-year-old man is seen in the respiratory clinic for a follow-up appointment of his chronic obstructive pulmonary disease (COPD). At his last appointment, his medications were escalated to regular inhaled Spiriva (tiotropium bromide), Symbicort (budesonide and formoterol), and salbutamol, as required. He reports ongoing shortness of breath at rest and on exertion that is limiting his activities of daily living. He has had 2 exacerbations in the last year and he has been an ex-smoker for 6 months.

On examination, he is tachypnoeic with saturations of 92% on air. On auscultation, there is an audible wheeze bilaterally and normal heart sounds. His calves are soft and non-tender with no evidence of oedema.

Arterial blood gas:

pH	7.35	(7.35 - 7.45)
PaO2	8.2 kPa	(11 - 13)
PaCO2	5.1 kPa	(4.7 - 6.0)
Haemoglobin	135 g/L	(135 - 180)
FEV1	< 50% predicted	

What is the most appropriate addition to the long-term management of this patient?

A. Long-term oxygen therapy
B. Montelukast
C. Nebulised salbutamol
D. Omalizumab
E. Roflumilast

E. Roflumilast

Roflumilast should be used in COPD for patients who are losing control on triple inhaled therapy

Important for me Less im

This patient has poorly controlled COPD, as seen by his persistent symptoms of shortness of breath despite increasing his regular medication regime. In patients who continue to have persistent symptoms despite a long-acting muscarinic antagonist, beta-agonist and inhaled corticosteroid, roflumilast can be tried. **Roflumilast** is a phosphodiesterase inhibitor recommended by NICE as an option for treating severe chronic obstructive pulmonary disease if the following criteria are met:

- Severe disease is defined as a forced expiratory volume in 1 second (FEV1) after a bronchodilator of less than 50% of predicted normal.

AND

- The person has had 2 or more exacerbations in the previous 12 months despite triple inhaled therapy with a long-acting muscarinic antagonist, a long-acting beta-2 agonist and an inhaled corticosteroid.

Long-term oxygen therapy is indicated for patients with COPD if the following criteria are met:

- Resting PaO2 ≤7.3-kPa

OR

- Resting PaO2 ≤8-kPa with evidence of peripheral oedema, polycythaemia (haematocrit ≥55%) or pulmonary hypertension.

This patient has a PaO2 of 8.2kPa, making long-term oxygen therapy currently inappropriate.

Montelukast is a leukotriene receptor antagonist that is used in the management of asthma. It is not routinely used in the management of COPD.

Nebulised salbutamol may help alleviate symptoms in patients with COPD. However, salbutamol is a short-acting beta-agonist and nebulised salbutamol will only provide short-term symptomatic relief and does not help with the long-term management of symptoms.

Omalizumab is a monoclonal antibody used in the treatment of persistent allergic asthma. It works by reducing the levels of IgE and subsequent inflammation. It is not used in the treatment of severe COPD.

Question 73 of 231

A 75-year-old man presents to the emergency department with hemoptysis. He has a past medical history of hepatitis C. He declined anti-viral therapy.

On examination, he has a saddle nose deformity. There is a non-blanching rash on his lower limbs. Blood tests:

Hb	88 g/L	Male: (135-180)Female: (115 - 160)
Platelets	489 * 10 /L 9	(150 - 400)
WBC	8.2 * 10 /L 9	(4.0 - 11.0)
Na +	138 mmol/L	(135 - 145)
K +	4.2 mmol/L	(3.5 - 5.0)
Urea	18.2 mmol/L	(2.0 - 7.0)
Creatinine	155 µmol/L	(55 - 120)
CRP	151 mg/L	(< 5)

Urinalysis:

Blood	+
Protein	+++

Plain radiography of the chest demonstrates bilateral infiltrates.

Given the probable diagnosis, what test is most likely to be positive?

A. Anti glomerular basement membrane antibody (anti-GBM)
B. C-ANCA
C. Cryoglobulins
D. Double-stranded DNA (dsDNA)
E. P-ANCA

B. C-ANCA

Granulomatosis with polyangiitis - cANCA positive

C-ANCA is correct. The patient has a multisystem disease characterized by haemoptysis, saddle nose deformity, likely pulmonary haemorrhage (infiltrates, haemoptysis, anaemia) and an acute kidney injury with haematoproteinuria. The likely diagnosis is granulomatosis with polyangiitis (GPA). GPA is an ANCA-associated vasculitis that is most commonly associated with cANCA.

Anti glomerular basement membrane antibody (anti-GBM) is incorrect. This can also cause a pulmonary-renal syndrome with pulmonary haemorrhage and rapidly progressive glomerulonephritis. However, it is not associated with the ENT features that are seen in GPA i.e. saddle nose deformity, as seen in this case. Rarely, the two disorders can co-exist but the more likely scenario is that cANCA will be positive rather than an anti-GBM antibody.

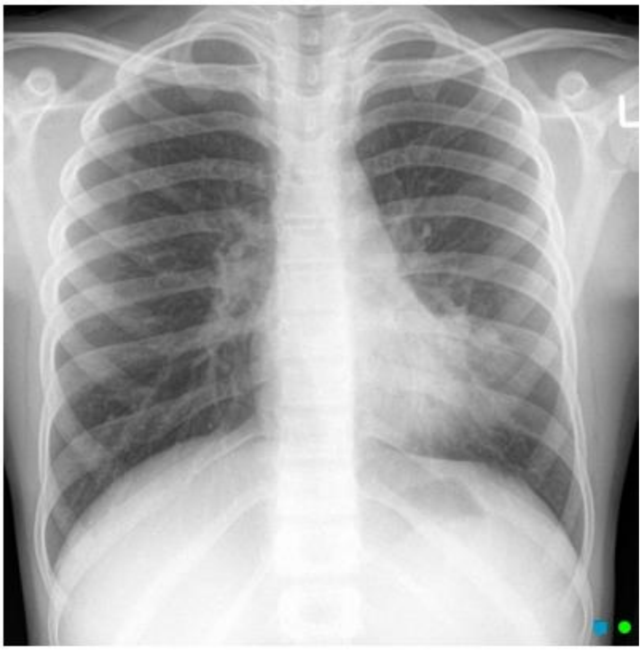
Cryoglobulins are incorrect. Hepatitis C infection would put the patient at risk of cryoglobulinaemia, which can cause rash and renal impairment. However, it is less likely to cause pulmonary haemorrhage and is not associated with ENT involvement, wich is present in this case.

Double-stranded DNA (dsDNA) is incorrect. This is a specific antibody associated with systemic lupus erythematosus. SLE is typically associated with younger women rather than older men. Additionally, ENT involvement is not typical and pulmonary haemorrhage is rarer than in ANCA vasculitis.

pANCA is incorrect. This is more commonly associated with microscopic polyangiitis. This typically caused pulmonary and renal vasculitis but is discriminated from GPA by lack of ENT involvement in most cases. It may also be positive in a variety of other conditions including inflammatory bowel disease and primary sclerosing cholangitis. In general, a positive pANCA with negative MPO/PR3 has a poor positive predictive value for vasculitis.

Question 74 of 231

An 11-year-old girl presents with a productive cough and fever. A chest x-ray is taken:



What is the main finding on the x-ray?

A. Bilateral pneumothoraces
B. Left lingual consolidation
C. Dilated cardiomyopathy with pulmonary oedema
D. Left humeral head fracture
E. Left middle lobe consolidation

B. Left lingual consolidation

The loss of the left heart border is a classic sign of left lingual consolidation. There is no left middle lobe

Question 75 of 231

A 56-year-old man presents to the Medical Admissions unit with a two-week history of a productive cough. His past medical history is significant for type 2 diabetes mellitus. He reports that he developed a cough productive of green sputum shortly after returning from a business trip to the United States. He also complains of persistent retrosternal pain for the last ten days. He has completed a course of doxycycline prescribed by his GP, to little symptomatic benefit.

Alongside routine blood tests, he provides a sputum sample for culture, with provisional results as follows:

Sputum culture	Fungal spores seen - full speciation awaited

Based on the likely diagnosis, what is the most appropriate management option?

A. Co-trimoxazole
B. Co-trimoxazole + prednisolone
C. Dapsone
D. Itraconazole
E. Voriconazole

D. Itraconazole

Amphotericin or itraconazole are the pharmacological agents of choice for histoplasmosis

The correct answer is **itraconazole**. This patient has a likely diagnosis of histoplasmosis. This is based on the presence of retrosternal pain alongside symptoms of a respiratory tract infection, combined with a history of recent travel to the United States, where the causative pathogen *Histoplasma capsulatum* is endemic, in parts. Further weight is given to the diagnosis of histoplasmosis by the presence of fungal spores in sputum culture. Of the options provided, only itraconazole is a typical first-line treatment of histoplasmosis.

Co-trimoxazole is incorrect. This would be an appropriate initial management option for *Pneumocystis jirovecii* pneumonia (PJP). This would tend to appear in question stems as a cause for hypoxia with exertional desaturation in immunosuppressed patients, rather than with a productive cough and retrosternal pain. Diagnosis tends to be confirmed on bronchoalveolar lavage rather than sputum culture.

Co-trimoxazole + prednisolone is incorrect as this would tend to be a regime used for PJP with severe hypoxia rather than histoplasmosis.

Dapsone is incorrect, as this tends to be used as a second-line agent for PJP if co-trimoxazole is ineffective or contraindicated, rather than treatment for histoplasmosis.

Voriconazole is incorrect. Although from the same class of drugs as itraconazole, voriconazole is more conventionally used for the treatment of invasive aspergillosis, or its prevention, for example in allogeneic stem cell transplant recipients. It is not typically used as a first-line treatment option for histoplasmosis.

Question 76 of 231

A 21-year-old man is admitted to the specialist burns unit with severe burns following a house fire. He is haemodynamically stable and receives IV fluids and oxygen, although does not require intubation. Three days after his admission, he develops acute-onset shortness of breath. The only past medical history reported was hay-fever as a child and his mother has relapsing-remitting multiple sclerosis. He smoked 1-2 roll-up cigarettes per week and did not drink any alcohol.

His observations include a respiratory rate of 28 breaths per minute, oxygen saturations of 91% on 12L of oxygen, a heart rate of 112 beats per minute and blood pressure of 114/78 mmHg. His temperature was 36.9ºC. Examination revealed widespread crackles over both lung fields.

Chest x-ray reveals bilateral infiltrates.

What is the most likely diagnosis?

A. Hospital-acquired pneumonia
B. Pneumonitis
C. Acute respiratory distress syndrome
D. Renal failure
E. Asthma attack

C. Acute respiratory distress syndrome

The correct answer is **Acute respiratory distress syndrome (ARDS)**. This patient presents with the classical features of ARDS following a major burn injury, which is a well-known precipitating factor. The diagnosis is supported by the acute onset of respiratory symptoms, bilateral infiltrates on chest x-ray, and hypoxaemia (requiring high-flow oxygen to maintain saturations above 90%) occurring within a week of the initial insult. The patient meets the Berlin criteria for ARDS, which includes timing within 7 days of a known clinical insult, bilateral opacities not fully explained by effusions or collapse, and respiratory failure not fully explained by cardiac failure or fluid overload.

Hospital-acquired pneumonia is incorrect because although this can occur in burns patients due to immunosuppression, the bilateral nature of the infiltrates and the timing (3 days) would be unusual. Hospital-acquired pneumonia typically presents after 48 hours of admission with unilateral changes initially, and often includes pyrexia and purulent sputum, which are not present here.

Pneumonitis is incorrect as this term typically refers to inflammation of lung tissue from noninfectious causes such as chemical injury or autoimmune conditions. While smoke inhalation can cause chemical pneumonitis, this would have been evident from admission rather than developing after three days.

Renal failure is incorrect as while this can occur in burns patients due to fluid loss and myoglobinuria, it would not explain the respiratory symptoms and chest x-ray findings. Whilst pulmonary oedema can occur in renal failure, the acute onset and bilateral infiltrates are more consistent with ARDS.

Asthma attack is incorrect because despite the patient's history of atopy (hay fever), the presentation is not consistent with acute asthma. Asthma typically presents with wheeze rather than crackles, and the chest x-ray would not show bilateral infiltrates. Additionally, asthma would be unlikely to develop acutely in this setting without a clear trigger.

Question 77 of 231

A 52-year-old gentleman presents to the respiratory clinic with shortness of breath and a dry cough. He has noticed his symptoms getting progressively worse over months. He has a past medical history of hypertension and depression. He takes only ramipril and has no allergies. On examination, he has bilateral inspiratory crackles at both lung bases. A chest X-ray demonstrates extensive pleural plaques. He has worked as an electrician 20 years ago and believes that he may have had asbestos exposure. A diagnosis of asbestosis with extensive pleural plaques is suspected.

What is his pulmonary function tests likely to show?

A. FEV1 to FVC ratio >0.7
B. FEV1 to FVC ratio <0.7
C. Normal ratio of FEV1 to FVC and normal FEV1
D. FEV1 to FVC ratio <0.7 but improves with inhaled steroids
E. FEV1 to FVC ratio <0.7 but improves with inhaled salbutamol

A. FEV1 to FVC ratio >0.7

The correct answer is FEV1 to FVC ratio >0.7, which would be in keeping with a restrictive pattern if the FEV1 compared to expected was also reduced. In asbestosis, there is restriction of lung expansion due to pleural disease, limiting expansion of lung volume as the plaques are not as expansile as pulmonary tissue. This stops the lungs from fully expanding leading to a restrictive picture on spirometry. Obstructive patterns, with a decreased ratio of FEV1 to FVC would be associated with COPD and asthma.

Question 78 of 231

A 24-year-old woman attends the respiratory clinic for a review of her asthma treatment. The patient reported that overall, her symptoms of asthma had been well controlled in the 6 months since her last review. She had not suffered any significant exacerbations of her asthma in that time and felt she was required to use her salbutamol inhaler only around once per week (which represented a significant improvement over her normal control). The patient also reported that she was now 3 months pregnant, and had just had an unremarkable 12-week ultrasound. While she had not been planning to have a baby, the patient reported being very happy about it, and she was keen to maximise her health during this time.

The patient had a long-standing diagnosis of asthma, with her first presentations during early childhood. While the illness had only caused mild symptoms between the ages of 10 and 15 years, the patient had suffered several severe exacerbations of asthma in her late teenage years, coinciding with the time when the patient had become a regular smoker. The patient did not report any other significant on-going health problems or past medical history. In particular, she denied a history of seizures, high blood pressure or mental health problems.

The patient's current asthma treatment was a salmeterol-fluticasone combination inhaler (Seretide Accuhaler 250), one puff twice daily. She was also prescribed a metered-dose salbutamol inhaler for use as required. The patient had no history of drug allergies.

The patient reported that she continued to be a regular cigarette smoker. Since finding out she was pregnant, she had managed to reduce her regular intake to 12 cigarettes per day, reduced from her previous typical intake of 20 cigarettes per day. The patient was motivated to use her pregnancy as a motivating factor to stop smoking permanently, and during the clinic asked about medication to assist her with quitting.

What is the appropriate drug treatment strategy to assist this patient to stop smoking?

A. Nicotine replacement patch with short-acting nicotine replacement
B. Varenicline
C. No drug treatment options available
D. Bupropion
E. Nicotine replacement patch only

Not answered in the file

Question 79 of 231

A 33-year-old woman with a three-year history of extremely difficult to control asthma and allergic rhinitis was admitted to the intensive

care unit having been deteriorating at home for several weeks Blood tests from a GP consultation last week were available:

Hb	121 g/l(115 - 165)
Platelets	135 * 10 /l(150 - 400)9
WBC	11.8 * 10 /l(4.0 - 11.0)9
Neuts	7.6 * 10 /l(1.5 - 7.0)9
Lymphs	0.9 * 10 /l(1.5 - 4.0)9
Eosin	3.03 * 10 /l(0.04 - 0.4)9
Serum CRP	103 mg/l(<10)
c-ANCA	negative
p-ANCA	positive
PR3	5U/ml(<10)
MPO	100U/ml(<10)

What is the most likely diagnosis?

A. Anti-glomerular basement antibody (anti-GBM) disease
B. Legionnaires' disease
C. Eosinophilic granulomatosis with polyangiitis
D. Microscopic polyangiitis
E. Granulomatosis with polyangiitis

C. Eosinophilic granulomatosis with polyangiitis

Eosinophilic granulomatosis with polyangiitis is a necrotizing small vessel vasculitis

The most likely diagnosis is eosinophilic granulomatosis with polyangiitis (EGPA), which tends to progress through three phases: atopy (asthma, rhinitis, sinusitis), eosinophilia >10% (often with pulmonary infiltrates), and necrotizing multi-system small vessel vasculitis. pANCA is positive in 40-70% of patients with eosinophilic granulomatosis with polyangiitis.

In contrast, patients with microscopic polyangiitis tend to have diffuse alveolar haemorrhage rather than nasal or asthma symptoms as the scenario depicted. Similarly, patients with granulomatosis with polyangiitis (Wegener's syndrome) tend not to have asthma as a feature and have predominantly positive cANCA. Patients with anti-glomerular basement antibody (anti-GBM) disease present with symptoms of pulmonary/renal involvement include haematuria, shortness of breath, cough, and expectoration of material that ranges from blood-streaked sputum to massive haemoptysis. The presence of anti-GBM antibodies is pathognomonic. Legionnaires disease is a form of pneumonia caused by *Legionella pneumophila* and negative to both cANCA and pANCA.

Question 80 of 231

A 60-year-old man is admitted with an infective exacerbation of COPD. He normally has a productive cough but has been bringing up large quantities of sputum and estimates about 1 cup of yellow sputum with streaks of blood being produced a day.

He takes steroid nose drops and had endoscopic surgery for recurrent sinusitis 5 years ago. He lives alone independently and drives a car. He can usually walk 50-100 metres on the flat.

He arrives to the emergency department in extremis. There is accessory muscle use and widespread coarse crepitations in both lungs. His heart sounds are normal with no signs of cardiac failure. He is started on antibiotics, steroids, aminophylline and back to back nebulisers. He continues to produce lots of phlegm and the nurses suction to clear a yellow mucoid substance from his mouth. He responds to commands, his eyes open to voice. and he is talking in normal words but his thoughts are incoherent. He is started on non invsaive ventilation and tolerates the mask. He continues to cough producing a 20ml plug of sputum with dried clot whilst given his first nebuliser. The respiratory registrar decides that NIV should be stopped.

Na +	139 mmol/l
K +	4.2 mmol/l
Urea	4.3 mmol/l
Creatinine	76 µmol/l
CRP	189 mmol mg/l
Hb	90 g/l
Platelets	178 * 10 /l9
WBC	23 * 10 /l9
ABG	(in air)
pH	7.39
pCO2	7.37 kPa
pO2	6.9 kPa
HCO3	25 mmol/l
Lactate	2.3 mmol/l
Chest x-ray	bilateral alveolar shadowing

Which contraindication to non invasive ventilation is present in this case?

A. Copious secretions
B. Not on maximal medical therapy
C. Endoscopic sinus surgery
D. Level of consciousness
E. Lack of consent

A. Copious secretions

Copious secretions are a contraindication to non-invasive ventilation

There are many contraindications to non invasive ventilation that must be carefully considered. Patients must be awake and able to protect their airway which this man although drowsy should be able to achieve with a Glasgow Coma Score of 13. The patient is on maximal medical therapy and there are few additional agents that would be helpful. His ENT surgery is important but some years ago and there is no head or neck trauma present which would be a contraindication. The gentleman is coughing up large volumes of sputum/secretions and this could pose a compromise to his airway. It is difficult to understand if he has capacity from the question but given his good premorbid function, active treatment would likely be in his best interests.

Question 81 of 231

A 52-year-old gentleman is admitted to hospital with a cough and fever. He has been unwell for a week with high temperatures, coughing rusty coloured sputum as well as small amounts of blood. He has no significant past medical history but smokes ten cigarettes per day. A chest X-ray shows right sided lower lobe consolidation with a pleural effusion. An aspirate demonstrates low pH raising the suspicion of empyema and a chest drain is requested under ultrasound guidance.

What factor may help predict drainage success or failure?

A. Protein level of pleural fluid
B. Echogenicity on US
C. pH of pleural fluid
D. Presence of associated consolidation
E. Septation density on ultrasound

E. Septation density on ultrasound

The correct answer is septation density on ultrasound. This is a patient with a possible empyema or parapneumonic effusion. The presence of low pH in the effusion makes the former more likely. The presence of septation density indicates various pockets within the effusion indicating that even if a drain accesses any pocket in particular, it may fail in draining all of the effusion. Protein level helps determine if an effusion is a transudate or an exudate, whilst a low pH is most useful in determining the risk of empyema. Consolidation may imply ongoing pneumonia and may suggest a cause of the effusion.

Question 82 of 231

You arrange a long term oxygen therapy (LTOT) assessment for a 69-year-old man. He has a past medical history of COPD. He has developed dyspnoea at rest.

Results of the LTOT assessment are as follows: ABG on air:

PaO2	7.1 kPa
PCO2	5.1 kPa
pH	7.41
HCO3	26 mmol/l

ABG after 1 litre of O2 via a nasal cannula:

PaO2	9.3 kPa
PCO2	6.5 kPa
pH	7.33
HCO3	26 mmol/l

The patient feels less short of breath at the end of the trial.

How will you manage this patient?

A. Provide LTOT
B. Decline LTOT
C. Further medical optimisation and reassess after 4 weeks
D. BiPAP
E. CPAP

C. Further medical optimisation and reassess after 4 weeks

Patients who develop a respiratory acidosis and/or a rise in PaCO2 of >1 kPa (7.5 mmHg) during an LTOT assessment may have clinically unstable disease. These patients should undergo further medical optimisation and be reassessed after 4 weeks

The ABG on room air meets the criteria for LTOT as the PaO2 is < 7.3 kPa. After a 1 hour trial of low flow oxygen, the patients symptoms have improved, and his PaO2 has significantly improved. However it is important to note that his PCO2 has significantly increased resulting in a respiratory acidosis.

Patients who develop a respiratory acidosis and/or a rise in PaCO2 of >1 kPa (7.5 mmHg) during an LTOT assessment may have clinically unstable disease. These patients should undergo further medical optimisation and be reassessed after 4 weeks.

Question 83 of 231

A 55-year-old male presents to the emergency department with worsening cough and breathlessness over the past three weeks. He reports a productive cough with haemoptysis and admits to fevers and weight loss over this time.

Past medical history is significant for type 2 diabetes mellitus. He currently smokes 20 cigarettes per day and drinks approximately 60 units per week.

Examination reveals on unkempt, cachectic man with bronchial breath sounds in the right upper zone.

Chest X-Ray is reported as having right upper lobe consolidation with a 'bulging fissure sign'

What is the most likely causative organism?

A. Klebsiella pneumonia
B. Mycobacterium tuberculosis
C. Aspergillus fumigatus
D. Staphylococcus aureus
E. Streptococcus pneumoniae

A. Klebsiella pneumonia

The bulging fissure sign is classical of Klebsiella pneumonia

The bulging fissure sign is an increasingly rare but classical feature of *Klebsiella pneumonia*. It is due to large volumes of consolidation (typically in the right upper lobe) causing displacement of the adjacent fissure.

Other features in the question to support a diagnosis of *Klebsiella pneumonia* include a history of alcohol excess and haemoptysis, often described as 'red currant jelly sputum'.

Whilst tuberculosis is an important differential to consider but seldom causes such dense consolidation to cause this sign. Radiologically, tuberculosis and aspergillosis usually appear as cavities. Klebsiella can also cause an isolated cavitating pneumonia.

Both *Streptococcal pneumoniae* and *Staphylococcus aureus* appear radiologically as the bulging fissure sign, other features in the question would be present to point you towards this diagnosis. Questions relating to *Streptococcal pneumoniae* will often describe a cough productive of rustycoloured sputum and often a proceeding herpes labialis. Questions relating to *Staphylococcus aureus* will commonly report a proceeding flu-like illness with subsequent rapid deterioration into a bacterial pneumonia.

Question 84 of 231

A 60-year-old gentleman is seen by his general practitioner 3 weeks after a recent infective exacerbation of COPD. He is currently well but concerned that this is his second exacerbation during this winter season.

He is currently managed on fostair, tiotropium and as required salbutamol. He currently has a mild cough but is well and has attended his recent COPD review.

Today, his chest is clear with no wheeze or crepitations. The JVP is not raised and there is no oedema. His oxygen saturations are 94% in air and he has a respiratory rate of 22 breaths per minute.

He is currently well and able to do his gardening and walks the dog up to half a mile each day. He does not get breathlessness at night and has been discharged from chest physio who are happy with his current performance.

FEV1 (% predicted)	40%
FVC (% predicted)	80%
Chest x-ray	several bullae noted, hyperexpanded chest with no consolidation
ECG	sinus rhythm, right bundle branch block

What intervention should be added to his current management?

A. Furosemide
B. Long term home oxygen
C. Home nebulisers
D. Roflumilast
E. Pulmonary rehabilitation

D. Roflumilast

Oral PDE-4 inhibitors such as roflumilast reduce the risk of COPD exacerbations in patients with severe COPD and a history of frequent COPD exacerbations

This is a gentleman with COPD and two recent exacerbations. He shows no signs of cor pulmonale or fluid overload and would therefore not benefit from a loop diuretic. Home nebulisers and pulmonary rehabilitation can be beneficial in severe symptomatic COPD but this patient is currently well and maintaining regular exertion without difficulty. Likewise, he is not hypoxic and there are no signs of cor pulmonale so he would not be a candidate for long term home oxygen. This leaves roflumilast as the answer, this is a phosphodiesterase-4 inhibitor that is recommended in COPD patients on maximal medical therapy with FEV1 <50% and had more than one exacerbation as per recent GOLD guidance.

Question 85 of 231

A 73-year-old gentleman presents to oncology clinic with a two-week history of shortness of breath. He has a background of mesothelioma diagnosed two years ago. He has noticed that he has been finding it more difficult to walk distances without developing shortness of breath, and today struggled to make it into the clinic from the car-park, 200 yards away. He denies having a cough or fever but has been feeling more tired lately. Saturations are 95% whilst breathing room and his blood pressure, temperature, heart rate and respiratory rate are all within normal parameters.

A chest X-ray is done which demonstrates a right-sided pleural effusion. He is admitted to hospital and a chest drain is inserted, draining three litres of blood-stained fluid in two hours. He becomes increasingly short of breath and starts to cough. He denies any chest pain. On examination, he is uncomfortable and breathing 4L of oxygen via a non-rebreather mask. There are bilateral crepitations in his chest.

25/11/16 16:31	25/11/16 14:18	
Saturations	91%	95%
Respiratory rate	22/min	18/min
Blood pressure	131/81mmHg	139/85mmHg
Heart rate	101/min	71/min
Temperature	37.0°C	37.1°C

What is the likely diagnosis?

A. Empyema
B. Iatrogenic chest infection
C. Re-expansion pulmonary oedema
D. Pneumothorax
E. PE

C. Re-expansion pulmonary oedema

The correct answer is re-expansion pulmonary oedema. This is a potentially life-threatening condition which can occur when a large volume of fluid or air is rapidly drained, as has happened with this patient. It is suggested by sudden onset of shortness of breath, cough and hypoxaemia following chest drain insertion. Empyema is unlikely given the absence of fever and no evidence of it on the original chest X-ray. Again, the absence of a fever makes iatrogenic infection less likely, and the timing is too early for infection. Pneumothorax is possible, but the examination demonstrates crepitations rather than any clinical evidence of pneumothorax. A PE is also possible given the history of malignancy and tachycardia and cannot be excluded clinically, but given the timing of the chest drain and the volume taken, re-expansion pulmonary oedema is more likely.

Question 86 of 231

A 26 year old asthmatic woman is admitted to hospital following a deliberate overdose of her usual theophylline tablets.

Which one of the following may be a complication of theophylline toxicity?

A. Hypoglycaemia
B. Atrioventricular nodal blockade
C. Bronchoconstriction
D. Increased myocardial contractility
E. Hyperkalaemia

D. Increased myocardial contractility

Theophylline poisoning presents with hypokalaemia, hyperglycaemia, tachycardia and increased myocardial contractility

Theophylline is a methylxanthine derivative drug which is structurally and clinically similar to caffeine. Aminophylline is the name given to theophylline when it is ligand bound to the chemical ethylenediamine which increases water solubility and hence aminophylline tends to be the intravenous form. Methylxanthine derivatives are used clinically for the treatment of bronchoconstriction in asthma, chronic obstructive pulmonary disease and in neonatal apnoea. They work in a two-fold fashion; firstly they inhibit the family of metabolic enzymes, the phosphodiesterases which increase levels of cyclic adenosine monophosphate and dampens the inflammatory cytokine cascade, and secondly they are competitive antagonists at the endogenous adenosine receptors located in the central nervous system, heart and lungs.

Inhibition of various phosphodiesterases causes a reduction in the circulating population of inflammatory cytokines and also increases cardiovascular and peripheral blood flow. Due to increased metabolism, hypokalaemia is common and gluconeogenesis and glycogenolysis is stimulated causing a rise in the plasma glucose load.

Competitive antagonism at the adenosine receptor causes roughly the opposite effects of adenosine. Whereas this drug is used clinically to slow the heart rate and block transmission at the AV node, theophyllines cause tachycardia and an increase in AV conduction, cardiac automaticity and an increase in myocardial contractility, ultimately leading to an increased cardiac output with hypertension and flushing. Inhibition of adenosine receptors in bronchioles causes relaxation and improved airflow, and in the central nervous system leads to agitation, headache, anxiety, tremor and insomnia.

Other symptoms of methylxanthine toxicity include extreme nausea and vomiting, pancreatitis, gastroesophageal irritation, psychiatric symptoms and seizures. Tachyarrhythmias may deteriorate into ventricular rhythms quickly which may be fatal. Competing metabolic acidosis and respiratory alkalosis may cause a mixed arterial blood gas picture and many electrolyte disturbances can occur but an unexplained hyperglycaemia with a hypokalaemia is very suggestive of theophylline toxicity. Theophylline has a narrow window of therapeutic efficacy and patients may become toxic rapidly if drugs which inhibit their metabolism are given concurrently, particularly antibiotics such as macrolides or fluoroquinolones.

Management of theophylline toxicity is largely supportive with seizures controlled by benzodiazepines or intubation. Phenytoin is best avoided as it can worsen arrhythmia. Electrolyte abnormalities must be corrected, particularly hypokalaemia and hypomagnesaemia. Hypertension and tachycardia may respond to beta-blockers but beware of making the situation worse in an asthmatic patient. Verapamil is an alternative. Ventricular rhythms should be electrically cardioverted.

Theophyllines are one of the few drugs which are highly adsorbed by activated charcoal and this should be considered even beyond an hour after ingestion due to the extended release nature of most preparations and the drugs slowing of gastric emptying

Question 87 of 231

A 53-year-old man with known interstitial lung disease presents with worsening of his shortness of breath, fevers and night sweats. He is an immigrant from Pakistan and has previously worked on quarry sites extracting silica from sand. He stopped due to shortness of breath and moved to the UK five years ago.

He does not work and can usually walk 100 metres before he becomes breathless but this has reduced to 10 metres over the past two weeks. He has a cough with phlegm but no haemoptysis and feels weak. He was well 1-2 months prior when he visited his home town in Pakistan and he does not recall meeting any ill contacts but reports that he took a number of long train journeys on public trains there.

On examination, he has crepitations in both lungs which are best heard at the apices. He is saturating at 92% on 2 litres of nasal cannulae. He is thin with a body mass index of 21. His JVP is not visible and there is no oedema in the calves.

Na +	139 mmol/l
K +	3.9 mmol/l
Urea	7.6 mmol/l
Creatinine	132 mmol/l
Hb	115 g/l
Platelets	378 * 10 /l9
WBC	11.9 * 10 /l9
Neuts	9.8 * 10 /l9
Eosin	0.1 * 10 /l9
D-dimer	elevated
Interferon-gamma release assay	positive
Sputum	gram stain negative, cultures awaited
ECG	sinus, p-pulmonale
Blood cultures	awaited
CXR	cavitating disease with reticulonodular pattern predominantly upper lobe

High-resolution CT chest	ground glass changes and honeycomb lung appearance, 2cm cavity seen in the left upper lobe with background emphysematous change

What is the likely diagnosis?

A. Cor pulmonale
B. Influenza
C. Pulmonary embolism
D. Tuberculosis
E. Lower respiratory tract infection

D. Tuberculosis

Silicosis increases susceptibility to tuberculosis

This gentleman with previous silica exposure likely has silicosis. He has recent travel on long distance train journeys and pulmonary embolism is possible. However, he has more systemic symptoms with weight loss and night sweats and has been to the Indian subcontinent where tuberculosis is prevalent. His IGRA test is positive suggesting exposure and he has cavitating disease on his CXR/CT scan. He could have a lower respiratory tract infection or influenza but the presentation is usually more acute. Cor pulmonale is the development of right-sided heart failure on the background of chronic respiratory distress and this is usually more gradual. Patients with silicosis are at higher risk of tuberculosis infection that is more severe and extensive than for other people and is termed silicotuberculosis.

Question 88 of 231

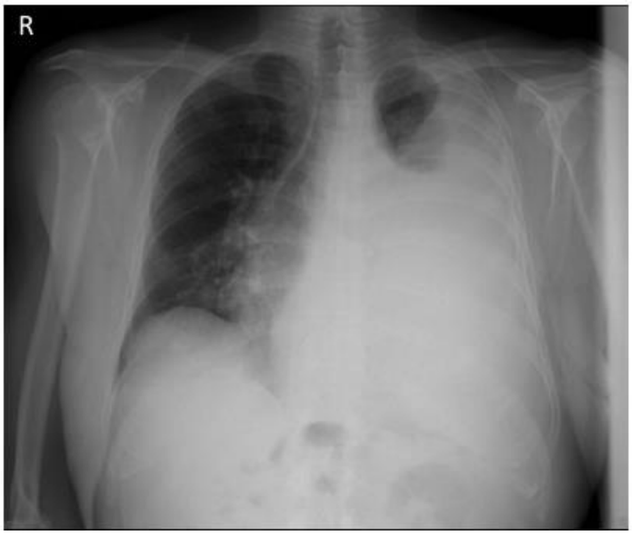
A 74-year-old man is admitted to the hospital with a 2-month history of progressive dyspnoea and weight loss. He has also noted a cough productive of purulent phlegm, with occasional flecks of dark red blood. His only medical history is of type 2 diabetes for which he takes oral therapies. He is an ex-smoker of 40 pack years.

His observations reveal a heart rate of 80/min, blood pressure of 130/85 mmHg, temperature 37.4ºC, respiratory rate of 24 breaths per minute and oxygen saturation of 94% whilst breathing 4 litres/min via nasal cannula.

Laboratory results are shown below:

Hb	120 g/L	Male: (135-180)Female: (115 - 160)
Platelets	410 * 10 /L 9	(150 - 400)
WBC	12.5 * 10 /L 9	(4.0 - 11.0)
Na +	130 mmol/L	(135 - 145)
K +	3.9 mmol/L	(3.5 - 5.0)
Urea	6.7 mmol/L	(2.0 - 7.0)
Creatinine	78 µmol/L	(55 - 120)
Calcium	2.75 mmol/L	(2.1-2.6)
Phosphate	0.81 mmol/L	(0.8-1.4)
Parathyroid hormone	1.0 pmol/L	(1.6 - 7.2)
CRP	90 mg/L	(< 5)

His chest radiograph is shown below:



What is the next most appropriate test for this patient?

A. Bronchoscopy
B. CT chest
C. Ultrasound-guided pleural aspiration
D. Sputum microscopy, sensitivity and culture (MC&S)
E. Technetium sesta-methoxyisobutylisonitrile (sestamibi) imaging

C. Ultrasound-guided pleural aspiration

This man's chest x-ray shows a unilateral left-sided pleural effusion. The history of weight loss and haemoptysis in a smoker raises concern for an underlying malignancy. Infection is a possibility given his elevated CRP and borderline pyrexia however the hypercalcemia makes malignancy more likely. The most appropriate next test for this man is an ultrasound-guided aspiration of the pleural fluid to both ascertain a diagnosis and to alleviate his symptoms of dyspnoea. The pleural fluid should be tested for biochemistry (including pH, protein, glucose and LDH), microbiology and cytology.

A CT chest will form part of this man's diagnostic tests however a pleural aspiration will both add diagnostic value and alleviate his symptoms in the first instance.

If the cytology yielded from a pleural aspiration is non-diagnostic or insufficient, then a bronchoscopy could be considered.

A CT scan of the chest would be required in advance of pursuing a bronchoscopy.

Technetium sestamibi scan is a nuclear medicine imaging technique that helps to localise parathyroid adenomas. Although this man is hypercalcemic, his parathyroid hormone levels are appropriately suppressed thus there is no evidence of hyperparathyroidism.

Sputum microscopy, culture and sensitivity (MC&S) would be a reasonable test to perform however the concern based on the imaging and clinical syndrome is of malignancy. Sputum MC&S is an essential test when diagnosing respiratory infection but will not diagnose the pleural effusion found on this man's chest radiograph.

Question 89 of 231

A 58-year-old man presents to the emergency department with a three-day history of sharp chest pain. He denies any shortness of breath. His pain is worse on deep inspiration and has not improved with paracetamol. He has no other symptoms. He has a past medical history of COPD and uses regular combination inhalers with salbutamol inhalers when he becomes short of breath. He has not recently had an exacerbation and he has never been admitted to ITU or had invasive ventilation. Examination demonstrates no abnormalities; the chest is clear on auscultation and vital parameters are within normal range. A chest X-ray demonstrates a <1cm right-sided pneumothorax. He is treated with high flow oxygen.

What is the benefit of oxygen In the treatment of a pneumothorax?

A. Maintaining pressure inside alveoli to prevent further expansion of pneumothorax
B. Exchange of nitrogen for oxygen allowing quicker resorption of the pneumothorax
C. Prophylactic treatment if suddenly becomes hypoxic
D. There is no indication for oxygen
E. Symptomatic relief

B. Exchange of nitrogen for oxygen allowing quicker resorption of the pneumothorax

The correct answer is the exchange of nitrogen for oxygen allowing quicker resorption. Patients who are diagnosed with a pneumothorax should be given high-flow oxygen as it allows the exchange of nitrogen from the air trapped forming the pneumothorax with the additionally inspired oxygen from given to the patient. This exchange increases the oxygen content of the pneumothorax whilst decreasing the nitrogen concentration. Oxygen is more readily absorbed, and therefore the pneumothorax resolves more quickly.

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A 35-year-old man presents with a chronic productive cough which has been affecting him for several years. He has no other past medical history apart from recurrent otitis media as a child. He takes no regular medications. He has never smoked and has no history of passive smoking and works as a solicitor. He has no pets at home and has no mould that he is aware of. On examination there is finger clubbing, he appears underweight, there are coarse late-inspiratory crepitations and a mild wheeze. Also, his heart sounds are louder on the right side, and his apex beat is also only present on the right.

What further investigation would most likely confirm the diagnosis?

A. Immunological testing for Aspergillus fumigatus specific IgE
B. CFTR gene testing
C. Sputum MCS
D. Sputum Zeil-Neilsen staining and microscopy.
E. Ciliary function tests

E. Ciliary function tests

The correct answer is ciliary function tests. This would confirm a diagnosis of primary ciliary dyskinesia. Ciliary function is commonly tested by the use of saccharin. Saccharin is placed 1cm behind the inferior turbinate and the test is positive if the patient does not taste sweetness within 20min. This is because if cilia work normally they will sweep the sweet saccharin inwards, allowing the patient to taste it.

Cilia line the upper respiratory tract, including the eustachian tubes, and moves normally in a coordinated fashion to help clear mucous, secretions and debris. In ciliary dyskinesis there is less movement or it is less coordinated. For this patient there is first a clear history and signs of bronchiectasis with chronic productive cough, bilateral late inspiratory crepitations and clubbing. But the history of otitis media, young patient and indications dextrocardia are all suggestive of primary ciliary dyskinesis. Cystic fibrosis is less likely due to the absence of gastrointestinal symptoms and signs, there is no travel history, heamoptysis or other factors to suggest TB, and there is no history of asthma making fungal infection and immunological response unlikely.

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A 32-year-old woman is reviewed in the respiratory clinic. She was admitted some 6 weeks earlier with acute severe asthma which required a period of ventilation. She is already treated with maximum dose salmeterol / fluticasone combination inhaler, montelukast and low dose oral corticosteroids, and a decision is made to start her on omalizumab.

Which of the following reflects the mode of action of omalizumab?

A. Anti-IgE
B. Anti-TNF
C. Anti-IL6
D. Anti-IL13
E. Anti-IL22

A. Anti-IgE

Omalizumab is an anti-IgE monoclonal antibody recommended by NICE for patients with severe allergic asthma which is refractory to other treatments including multiple courses of oral corticosteroids per year. The manufacturer recommends that the drug is only used in patients who have a positive skin test to a recognised respiratory allergen.

Anti-TNF antibodies such as etanercept are used in the treatment of rheumatoid arthritis. Anti-IL6 antibodies such as tocilizumab are also used in the treatment of arthritis. Anti-IL13 and anti IL22 antibodies are both thought to target cytokines responsible for the development of asthma, and may play a role in combination therapy for the disease.

Question 92 of 231

A 51-year-old lady is admitted to the Medical Admissions Unit with a 2-week history of fever, breathlessness, lethargy and fatigue.

She has a history of poorly controlled asthma which has caused her to attend the Emergency Department five times over the past 2 years. She was reviewed by the respiratory team during her last admission and started on reducing dose oral prednisolone. However, she was unable to reduce her dose below 10mg per day due to recurrent breathlessness and wheeze.

She was reviewed again in the Respiratory Clinic 4 months ago and started on montelukast 10mg per day as a steroid-sparing therapy. Her prednisolone was subsequently reduced to 5mg per day and she reports that she had been well since then.

Her past medical history is otherwise remarkable for sinusitis and nasal polyposis. She has had several surgeries for the latter. Her current medications include Seretide 500/50 1 puff twice daily, montelukast 10mg once daily, prednisolone 5mg once daily and salbutamol via MDI as required. She is allergic to aspirin.

On examination, her temperature is 37.6ºC, her pulse is 98bpm and her blood pressure is 164/91mmHg. Her respiratory rate is 22/min and her oxygen saturations are 93% breathing room air. Her JVP is elevated at 5cm. Auscultation of the chest reveals diffuse polyphonic wheeze and bibasal crackles extending to the mid-zones. There is mild ankle oedema. Her abdomen is soft and non-tender.

Her initial investigations are as follows:

Hb	112 g/l	Na +	136 mmol/l
Platelets	215 * 10 /l 9	K +	5.9 mmol/l
WBC	14.2 * 10 /l 9	Urea	22.3 mmol/l
Neuts	8.4 * 10 /l 9	Creatinine	484 µmol/l
Lymphs	2.1 * 10 /l 9	CRP	107 mg/l
Eosin	3.4 * 10 /l9		
Urine dipstick	Blood +++ protein ++		

What would be the most likely finding on renal biopsy?

A. Mesangial hypercellularity
B. Pauci-immune crescentic glomerulonephritis

C. Podocyte fusion
D. Thickened capillary walls with subepithelial deposits
E. Diffuse proliferative glomerulonephritis

B. Pauci-immune crescentic glomerulonephritis

The patient has presented with worsening asthma, prominent peripheral blood eosinophilia, and acute kidney injury. This, in combination with the history of paranasal sinusitis, points towards a diagnosis of eosinophilic granulomatosis with polyangiitis (EGPA) or Churg-Strauss syndrome.

EGPA is a rare systemic necrotising vasculitis that affects small to medium-sized blood vessels. The natural history of the disease progresses through three distinct phases: a prodromal phase, characterised by asthma and paranasal sinusitis; an eosinophilic phase, with prominent peripheral blood eosinophilia and eosinophilic infiltration of various bodily tissues, and a final vasculitic phase. Vasculitic phenomena include skin rashes, peripheral neuropathy, and renal involvement; the latter is most commonly due to rapidly progressive glomerulonephritis (RPGN). RPGN is characterised histologically by the formation of epithelial crescents in the majority of glomeruli.

There is an association between the introduction of leukotriene receptor antagonists (LTRAs) and the onset of EGPA. The reason for this is unclear and various theories have been proposed. It has been suggested that some patients with difficult-to-control asthma may actually have a subclinical form of EGPA that is suppressed by repeated courses of corticosteroids. Instituting therapy with LTRAs allows tapering of corticosteroids and precipitates clinical disease.

The patient in the vignette may have had aspirin-sensitive asthma, in the which LTRAs have a prominent therapeutic role. There may also be a direct link between aspirin-sensitive asthma and EGPA.

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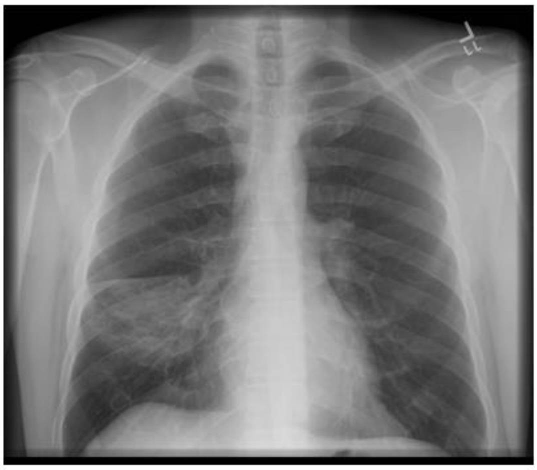
A 25-year-old man presents to the emergency department with a one-week history of cough, fever and shortness of breath. He has no past medical history and has never previously attended the hospital. He is orientated and responds to questioning appropriately. His respiratory rate is 14 breaths per minute, oxygen saturations are 98% on room air and his blood pressure is 115/65 mmHg.

He reports that he had a previous reaction to pivmecillinam aged 20, with the development of a widespread raised itchy rash.

Blood tests are taken with the following results:

Hb	123 g/L	(135-180)
Platelets	478 * 10 /L 9	(150 - 400)
WBC	17 * 10 /L 9	(4.0 - 11.0)
Na +	137 mmol/L	(135 - 145)
K +	4.2 mmol/L	(3.5 - 5.0)
Urea	4.5 mmol/L	(2.0 - 7.0)
Creatinine	85 µmol/L	(55 - 120)

A chest x-ray is taken, as shown below:



What is the most appropriate management for this patient?

A. Admit for IV amoxicillin and clarithromycin
B. Admit for IV co-trimoxazole
C. Admit for respiratory review
D. Discharge with oral doxycycline
E. Discharge with oral doxycycline and clarithromycin

D. Discharge with oral doxycycline

The chest x-ray shows an area of dense consolidation in the right mid-zone, typical of pneumonia, which is community-acquired in this case. The patient scores 0 on the CURB-65 score, which means that he is in a low-risk group with a 0.6% 30-day mortality, and can be managed as an outpatient with oral antibiotics given that there are no other concerning features.

The patient has had a previous urticarial reaction to pivmecillinam (which is a type of penicillin usually given for urinary tract infections), suggesting he is likely to have a type 1 allergic reaction to penicillin, and should not receive penicillin in the future.

Discharge with oral doxycycline is correct. NICE recommends that patients with penicillin allergies should be treated with either a macrolide (clarithromycin or azithromycin) or doxycycline.

Admit for IV amoxicillin and clarithromycin is incorrect. A patient might be admitted if they scored 2 or more on the CURB-65 score, or if there were other features giving cause for concern, which is not the case in this scenario. The patient should also not be given amoxicillin, which is a type of penicillin.

Admit for IV co-trimoxazole is incorrect. This antibiotic choice would be safe for this patient as it is not penicillin based but, as above, the patient does not require hospital admission. Cotrimoxazole is sometimes used for hospital-acquired pneumonia as it has a broader spectrum of activity but is not routinely used for community-acquired pneumonia.

Admit for respiratory review is incorrect. Although there is a significant area of consolidation on the chest x-ray, the patient is not compromised and does not require specialist respiratory input at this point. He would require a follow-up chest x-ray at 4-6 weeks to ensure that the consolidation has resolved.

Discharge with oral doxycycline and clarithromycin is incorrect. It would be reasonable to give the patient doxycycline alone, as per NICE guidelines for patients with penicillin allergies.

Clarithromycin is often added to provide cover for atypical organisms, such as *Legionella pneumophila* and *Mycoplasma pneumoniae* which are not treated by penicillin-based antibiotics. However, NICE recommends that atypical cover should only be prescribed for patients with moderate or severe community-acquired pneumonia, so it is not required in this patient.

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You are the admitting medical registrar and receive the following referral from your emergency department colleague.

A 30-year-old woman with asthma has presented with three days of increasing wheeze, dyspnea, productive cough and fevers. You are informed there is a leukocytosis and consolidation on the chest x-ray. The emergency department team have commenced antibiotics and referred for inpatient admission.

You obtain a further history. This patient was diagnosed with asthma in childhood but as an adult, she rarely required any use of her salbutamol inhaler and did not require follow up with a respiratory physician or general practitioner. However, over the last 18 months, she has been experiencing increasing wheezing and cough. Initially, this was a dry cough but has now become productive of brown sputum. There is no history of haemoptysis. This has been associated with malaise and weight loss. She was admitted to hospital with an asthma exacerbation two weeks ago and was discharged on a short course of prednisolone and started on a salmeterol-fluticasone combination inhaler. Following discharge, she initially felt improved but feels her symptoms have returned over the last few days. She denies missing any doses of her inhaled medication and uses a spacer. There is no history of chest pain or travel. She is a lifelong non-smoker and works full time as a computer programmer.

Past medical history includes allergic rhinitis, eczema and endometriosis.

Examination revealed a thin woman, with respiratory rate of 22/min, oxygen saturation 95% on air, blood pressure 100/60 mmHg, heart 90/min and temperature of 37.8ºC. There was diffuse expiratory wheeze and reduced breath sounds in the right upper zone of the anterior chest. Heart sounds were dual with no murmurs. The jugular venous pressure was not elevated. There was palpable cervical lymphadenopathy and no clubbing. Calves were soft, non-tender and there was no pedal oedema.

Hb	140 g/l	Na +	138 mmol/l
Platelets	380 * 10 /1 9	K +	3.6 mmol/l
WBC	11 * 10 /1 9	Urea	5 mmol/l
Neuts	9 * 10 /1 9	Creatinine	70 µmol/l
Lymphs	1 * 10 /1 9	CRP	20 mg/l
Eosin	1 * 10 /l9		

A chest x-ray shows a homogeneous triangular shaped opacity extending from the mediastinum towards the right upper zone.

Which of the following are you most likely to find this patient?

A. Elevated serum IgE levels
B. Upper lobe bronchiectasis on high resolution computed tomography of the thorax
C. A positive urinary Streptococcus pneumoniae antigen
D. Sputum analysis staining positive for acid-fast bacilli
E. No change in symptoms with a repeat course of prednisolone

A. Elevated serum IgE levels

Consider allergic bronchopulmonary aspergillosis in asthma patients who have developed difficult to control symptoms and/or have become dependent on oral steroids

This patient has allergic bronchopulmonary Aspergillosis (ABPA) as evidenced by the development of inadequately controlled asthma, cough productive of brown sputum, malaise, weight loss and a homogeneous opacity on chest x-ray. Typically there is a serum eosinophilia but given the recent course of prednisolone, the eosinophil count may still be suppressed.

ABPA is a chronic hypersensitivity reaction that occurs in response to colonization of the lower airways with Aspergillus species. It occurs most commonly in patients with atopic asthma or cystic fibrosis (CF). The resulting inflammation can lead to the destruction of pulmonary parenchyma and bronchiectasis.

A diagnosis of ABPA is supported by:

- Symptoms as described above
- Serum eosinophilia in steroid naÃ¯ve patients and/or no history of recent steroid use
- Elevated total serum IgE level (>1000 iU/ml), positive serum Aspergillus precipitins (IgM, IgG and IgE antibodies to Aspergillus species)
- Sputum showing elevated eosinophil count and/or fungal mycelia with healthy cytoplasm (cf. dead mycelia devoid of cytoplasm seen in patients with Aspergilloma)
- Immediate skin test reactivity to Aspergillus antigens
- A massive homogeneous shadow without fissure displacement is the most common abnormality seen on a chest radiograph with ABPA
- The bronchiectasis seen in ABPA is typically central

Management is usually with prednisolone +/- azole class antifungals (e.g. Itra or Voriconazole)

Option A - the correct answer

Option B - central bronchiectasis is typically seen, rather than lobe specific

Option C - community acquired pneumonia is a valid differential but the history and investigations are in favour of ABPA. If there was a pneumonic process that has such a dense consolidation to give homogeneous opacity on chest x-ray, you would expect worse hypoxia and respiratory distress as seen in this patient.

Option D - *Mycobacterium tuberculosis* infection is unlikely given the absence of risk factors and the history/investigations makes the pre-test probability of ABPA much higher than

Mycobacterium tuberculosis

Option E - prednisolone is the first line treatment for ABPA and patients may require multiple and/or prolonged courses. Azole anti-fungal therapy may also be added in order to decrease steroid burden and/or if there is a poor response to corticosteroid therapy.

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A 67-year-old gentleman presents with a three-day history of a productive cough. He complains that over the last two days he has been progressively more short of breath, He complains of feeling very weak and lethargic and on further questioning reports fevers and rigors. His wife brought him to the emergency department as she was concerned as he appeared to be deteriorating rapidly.

Observations are: heart rate 125 beats per minute, respiratory rate 32 breaths per minute, S 0 90% on room air, temperature 38.9º, blood pressure is 130/84 mmHg.

He appears distressed but is not confused. Initial investigations show:

Hb	134 g/l
Platelets	550 * 10 /l9
WBC	18 * 10 /l9
Na +	141 mmol/l
K +	3.7 mmol/l
Urea	9.2 mmol/l
Creatinine	130 µmol/l

Chest x-ray shows left lower zone consolidation.

From the above information, what is his CURB-65 score?

A. 1
B. 2
C. 3
D. 4
E. 5

C. 3

This patient has a community-acquired pneumonia which is a common presentation on the acute medical take (Incidence: 5-11/1000). Patients typically present with a productive cough, fevers, shortness of breath, anorexia, rigors and may report pleuritic chest pain and haemoptysis. Community-acquired pneumonia may be primary or secondary to underlying disease.

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A 49-year-old woman presents with a one-year history of joint pain and an intermittent purpuric rash. For the last 3 months, she feels more fatigued than usual with malaise, fever and intermittent abdominal pain. She had an appendectomy 10 years ago. Besides that, there is no past medical history of significance.

On clinical examination, the only abnormality is a purpuric rash involving her calves and thighs.

The results of initial investigations showed a normal full blood count, but impaired renal function with a plasma creatinine of 160 µmol/l.

Another set of investigations were ordered to reach a diagnosis:

Na+	135 mmol/l
K+	4 mmol/l
Creatinine	165 µmol/l
Urea	11 mmol/l
CRP	50 mg/l
ESR	70 mm/hr
Urine analysis	Protein +,RBCs ++
Rheumatoid factor	negative
C3 and C4 levels	normal
cANCA	positive
pANCA	positive
ANA	negative

Renal biopsy showed focal necrosis, crescent formation, and absence of immunoglobulin deposits on immunofluorescence.

What is the most likely diagnosis?

A. Rheumatoid vasculitis
B. Mixed cryoglobulinaemia
C. Wegener's granulomatosis (WG)
D. Microscopic polyangiitis (MPA)
E. Systemic lupus erythematosus (SLE)

D. Microscopic polyangiitis (MPA)

MPA is a systemic vasculitis affecting the small vessels including arterioles, capillaries, and venules. It usually presents with a long history of constitutional symptoms with multisystem involvement. Note that cANCA and pANCA could be useful in differentiating WG from MPA but not always.

SLE would be unlikely with a negative ANA, and pauci-immune glomerulonephritis.

WG is unlikely with the absence of upper and lower airway involvement. Also, the absence of granuloma formation in the kidneys would not favour it.

Usually, rheumatoid vasculitis would be clinically apparent because it develops on a background of severe rheumatoid disease. Also, the rheumatoid factor would be very high, with low C3 and C4.

Mixed cryoglobulinaemia characteristically would have a positive rheumatoid factor. Glomerulonephritis has distinctive features, being characterized by marked deposition of immunoglobulin and complement. C4 would be markedly low in 90% of patients due to classical pathway activation by cryoglobulins. This is in contrast to MPA where the complement levels would be normal or even elevated as an acute phase response.

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A 40-year-old man attends the emergency department with a 3-day history of worsening respiratory symptoms. The patient reported that his initial symptoms had included a runny nose, sore throat and myalgia. Subsequently, he developed a persistent dry cough and had experienced mild shortness of breath on exertion, for example after climbing a flight of stairs. The patient had not measured his temperature at home but recalled that, at times, he had felt extremely hot and sweaty. The patient denied any other symptoms, including a cough productive of sputum or blood, abdominal symptoms or chest pain.

The patient reported having just returned from a 2 week holiday in Egypt. The majority of the patient's trip had been based in large towns and cities; however, the patient had also participated in a 4-day 'camel trek' through the desert. On direct questioning, the patient reported having drunk camel milk offered to him by his guides, which he did not think had been pasteurised or otherwise processed prior to consumption. The patient had not knowingly spent time around any unwell individuals and had not sought medical advice while overseas. Prior to this holiday, the patient had not travelled outside of the UK for several years.

The patient was in generally good health, with no active chronic medical problems. The patient's only previous attendance at the emergency department was 5 years previously when he had treated for a broken ankle sustained when playing football. The patient took no regular medications and had no known allergies to medications. The patient did not regularly consume alcohol or recreational drugs but did smoke 5-10 cigarettes per day.

General examination of the patient showed a middle-aged man who appeared flushed in the face and unwell. Examination of the respiratory system demonstrated normal air entry and resonance to both lung fields, with no significant added sounds on auscultation. With the patient at rest, there were no signs of respiratory distress. Examination of the cardiovascular and abdominal systems demonstrated no significant abnormality. There was no notable rashes and no signs of peripheral deep vein thrombosis.

Heart rate	92 beats / minute
Respiratory rate	18 respirations / minute
Blood pressure	125 / 76 mmHg
Oxygen saturations	95 %
Glasgow coma scale score	15
Temperature	38.2 Co

Haemoglobin	135 g / dL
White cell count	3.7 x 10>3 / microlitre
Neutrophils	2.5 x 10>3 / microlitre (reference 1.8-7.8)
Lymphocytes	1.0 x 10>3 / microlitre (reference 1.5-4.0)
Platelets	123 x 10>3 / microlitre
Urea	6.1 mmol / L
Creatinine	82 micromol / L
Alkaline phosphatase	154 U / L (reference 35-100)
ALT	72 U / L (reference 3-36)
Bilirubin	23 micromol / L (reference < 26)
Chest x-ray	clear lung fields, no pneumothorax

What is the likely pathogen responsible for the patient's illness?

A. MERS coronavirus
B. Dengue virus
C. West Nile virus
D. Junin virus
E. Ebola virus

A. MERS coronavirus

The patient's presentation is consistent with Middle East respiratory syndrome (MERS), caused by the betacoronavirus MERS-CoV. Dromedary camels are the primary animal host for MERS-CoV, with human transmission occurring secondary to close contact with camels, or the consumption of unpasteurised camel milk or undercooked camel meat. However, human-to-human transition via respiratory droplets is responsible for the majority of cases of MERS, particularly in the healthcare setting. At the present time, MERS-CoV is limited to the Arabian Peninsula and its surrounding countries, although due to an incubation period of 2-14 days travellers returning from this region may present with MERS in other parts of the world.

The clinical syndrome of MERS is varied, ranging from very only mild symptoms through to lifethreatening multi-organ failure. The patient in the question has typical blood results for MERS, including lymphocytopenia, thrombocytopenia and deranged liver function tests. The diagnosis of MERS can be confirmed by reverse transcriptase polymerase chain reaction analysis of respiratory tract secretions to identify MERS-CoV RNA. No specific treatment for MERS-CoV infection is available. Individuals developing significant complications (or those at high-risk of doing so) require admission to hospital for supportive care.

The pathogens included as incorrect answers to the question either are not endemic in the region the patient had travelled or have clinical presentations dissimilar to that described in the question.

Question 98 of 231

A 73-year-old is referred to the pulmonary hypertension team after undergoing an echocardiogram. She had initially been admitted after a 4-day history of exertional dyspnoea. A chest x-ray revealed bilateral pulmonary oedema. She demonstrated acute ischaemic changes on her ECG. Her past medical history includes type 2 diabetes mellitus, hypertension, hypercholesterolaemia and a previous NSTEMI in 2010. An echocardiogram demonstrated moderate to severe LV dysfunction, with a pulmonary arterial pressure of 83 mmHg (normal <25 mmHg).

What is the most appropriate next diagnostic investigation?

A. No further investigations for pulmonary hypertension
B. V/Q scan
C. CTPA
D. Right and left heart catheterisation
E. Lung function tests

A. No further investigations for pulmonary hypertension

Pulmonary hypertension is divided according to the World Health Organisation (WHO) into five classes based on aetiology:

- I pulmonary arterial hypertension
- II pulmonary hypertension secondary to left heart disease
- III pulmonary hypertension secondary to lung disease
- IV pulmonary hypertension secondary to chronic thromboembolic disease
- V pulmonary hypertension with unclear causes

This patient has sufficient left-sided heart disease to explain her pulmonary hypertension and is hence a group 2 pulmonary hypertension patient. No further investigations are required. Treatment of pulmonary hypertension, in this case, is dependent on optimising left ventricular failure.

Question 99 of 231

A 67-year-old female patient presents to respiratory outpatients having been referred by her GP with increasing breathlessness. The referral letter mentions she has received 2 courses of antibiotics for chest infections in the last year and a 50 pack year smoking history. The patient complains of a productive cough with occasional streaks of blood but no history of weight loss. Her past medical history includes cardiac failure treated with enalapril, bisoprolol and furosemide, recurrent urinary tract infections treated with long-term nitrofurantoin and osteoarthritis treated with paracetamol and meptazinol. On examination, there is bilateral scattered coarse crepitations and finger clubbing.

Spirometry in clinic is as follows:

Forced vital capacity (FVC)	1.99L (72% predicted)
Forced expiratory volume in 1 second (FEV1)	1.20L (52% predicted)
FEV1/FVC ratio	0.60

What is the likely diagnosis?

A. Bronchiectasis
B. Chronic obstructive pulmonary disease
C. Pulmonary fibrosis
D. Squamous cell carcinoma
E. Congestive cardiac failure

A. Bronchiectasis

The spirometry results suggest an obstructive lung disease as FEV1/FVC ratio is < 0.7.

This patient is likely to have bronchiectasis given the obstructive spirometry, productive cough, coarse crepitations and history of recurrent chest infections.

Suspicion of pulmonary fibrosis is raised by long-term nitrofurantoin use but the history, examination and spirometry findings do not fit this diagnosis. Congestive cardiac failure may cause bilateral crepitations but does not cause finger clubbing. A malignant diagnosis is less likely given the absence of weight loss and bilateral signs. One would expect to find wheeze to support a diagnosis of chronic obstructive pulmonary disease.

Question 100 of 231

A 30-year-old man presents to the Emergency Department with severe shortness of breath, cough and ongoing weight loss. He has a past medical history of asthma and nasal polyps.

He has seen his general practitioner (GP) 3 times in the last 6 months for chest infections and been given antibiotics.

Given the likely underlying diagnosis, what would you expect to see on his blood results?

A. Low lymphocyte count
B. Iron deficiency anaemia
C. Raised neutrophil count
D. Raised eosinophil count
E. Low platelet count

D. Raised eosinophil count

Churg-Strauss syndrome - eosinophilia

Although a raised neutrophil count is a potential here, a raised eosinophil count is classical of Churg-Strauss, and is required for diagnosis. Nasal involvement is more commonly associated with Wegener's granulomatosis, but it is also seen in a variety of other vasculitides.

Churg-Strauss syndrome (also known as eosinophilic granulomatosis with polyangiitis) is a vasculitis that affects small and medium-sized blood vessels.

It is commonly seen in patients with a history of atopy. It is often described to occur in three stages - the allergic 'prodromal' stage, the eosinophilic stage and the final vasculitic stage.

The mainstay of treatment includes steroids and other immunosuppressant drugs.

Question 1 of 133

A 22-year-old man presents to the emergency department with a worsening cough and shortness of breath over the past few days. He has no significant past medical history except for a known allergy to wasp stings and is not on any regular medication. Although he does not smoke himself, he works in an environment where others are often smoking around him.

On examination, he looks to be in respiratory distress, with tachypnoea, tachycardia and low-grade pyrexia. Peripheral oxygen saturations are 87% on air. Blood tests are performed:

Hb	112 g/L	Male: (135-180)Female: (115 - 160)
Platelets	185 * 10 /L 9	(150 - 400)
WBC	13.3 * 10 /L (22% eosinophils) 9	(4.0 - 11.0)
Urea	6.9 mmol/L	(2.0 - 7.0)
Creatinine	87 µmol/L	(55 - 120)

A chest X-ray is also performed, which demonstrates patchy mixed alveolar-interstitial infiltrates. The medical team are asked to review the patient and a bronchoalveolar lavage is conducted which demonstrates no signs of infection, but 56% eosinophils.

Given the likely diagnosis, which intervention is most appropriate initially?

A. Albendazole
B. Cyclophosphamide
C. Itraconazole
D. Methylprednisolone
E. Plasma exchange

D. Methylprednisolone

Acute eosinophilic pneumonia is highly responsive to steroids

The diagnosis here is the rare condition acute eosinophilic pneumonia. It is one of several conditions that cause pulmonary eosinophilia and is characterised by a cough, shortness of breath and sometimes a history of allergy. Being a smoker, or being exposed to second-hand smoke, is also thought to be a trigger. The key to diagnosis here is the patient's chest x-ray demonstrating patchy infiltrates, but crucially the bronchiolar lavage which shows a high eosinophil count. This condition is highly responsive to steroids and so methylprednisolone is the correct answer.

Albendazole is an antiparasitic medication. This may be indicated in Loeffler's syndrome - chest xray shadowing and blood eosinophilia sometimes due to parasites such as *Strongyloides stercoralis*. The history would have included more focus on a fever and night sweats, and parasites would have been identified during diagnosis.

Cyclophosphamide is an immunosuppressant, used in the management of cancers and autoimmune conditions. This would be indicated if the diagnosis were a rheumatological condition such as eosinophilic granulomatosis with polyangiitis. Similar features would be seen, but a history of asthma would be noted, as well as renal failure; this patient's renal function bloods were normal.

Itraconazole is an antifungal medication and may be used in the management of a differential, allergic bronchopulmonary aspergillosis (ABPA), characterised by asthma-like symptoms that do not respond to initial treatment. Eosinophilia is also seen, but a positive radioallergosorbent (RAST) test to *Aspergillus* should be demonstrated, which is not seen here. Oral steroids are actually used first-line, with itraconazole sometimes being used as an adjunct.

Plasma exchange does not play a role here. It may be used in the management of severe granulomatosis with polyangiitis, as well as a number of other conditions.

Question 2 of 133

A 62-year-old gentleman comes to the respiratory clinic for review. He was seen by his GP after presenting with pleuritic chest pain and slight breathlessness. The GP was concerned and arranged for a chest X-ray which shows pleural plaques and a slight right-sided pleural effusion. He has a past medical history of type 2 diabetes, hypertension and angina. He takes ramipril, amlodipine, metformin and has a GTN spray for when he needs it. He worked as a plumber over 30 years ago. On examination he has clubbing and slightly reduced air entry in the right base of his lungs, with some dullness on percussion. A diagnosis of asbestosis is suspected.

What feature of his history or examination is evidence towards poor prognosis?

A. Comorbidity with ischaemic heart disease
B. Shortness of breath
C. Pleuritic pain
D. Clubbing
E. Male sex

D. Clubbing

The correct answer is clubbing. This is a patient who presents with pleuritic chest pain and shortness of breath with a history of possible asbestos exposure (plumbing). These are common findings on diagnosis and are not associated with a difference in prognosis. Clubbing, however, has been shown to be a poor prognostic indicator as it has been associated with progression to mesothelioma.

Question 3 of 133

A 24-year-old woman attends the respiratory clinic for a review of her asthma treatment. The patient reported that overall, her symptoms of asthma had been well controlled in the 6 months since her last review. She had not suffered any significant exacerbations of her asthma in that time and felt she was required to use her salbutamol inhaler only around once per week (which represented a significant improvement over her normal control). The patient also reported that she was now 3 months pregnant, and had just had an unremarkable 12-week ultrasound. While she had not been planning to have a baby, the patient reported being very happy about it, and she was keen to maximise her health during this time.

The patient had a long-standing diagnosis of asthma, with her first presentations during early childhood. While the illness had only caused mild symptoms between the ages of 10 and 15 years, the patient had suffered several severe exacerbations of asthma in her late teenage years, coinciding with the time when the patient had become a regular smoker. The patient did not report any other significant on-going health problems or past medical history. In particular, she denied a history of seizures, high blood pressure or mental health problems.

The patient's current asthma treatment was a salmeterol-fluticasone combination inhaler (Seretide Accuhaler 250), one puff twice daily. She was also prescribed a metered-dose salbutamol inhaler for use as required. The patient had no history of drug allergies.

The patient reported that she continued to be a regular cigarette smoker. Since finding out she was pregnant, she had managed to reduce her regular intake to 12 cigarettes per day, reduced from her previous typical intake of 20 cigarettes per day. The patient was motivated to use her pregnancy as a motivating factor to stop smoking permanently, and during the clinic asked about medication to assist her with quitting.

What is the appropriate drug treatment strategy to assist this patient to stop smoking?

A. Nicotine replacement patch with short-acting nicotine replacement
B. Varenicline
C. No drug treatment options available
D. Bupropion
E. Nicotine replacement patch only

A. Nicotine replacement patch with short-acting nicotine replacement

The patient is motivated to stop smoking and this can be expected to have significant benefits, both for her own long-term health and for the health of her unborn fetus. The best drug treatment option available to her in pregnancy is combination nicotine replacement therapy, consisting of a long-acting nicotine replacement patch with a short-acting nicotine formulation (either oral or nasal spray) to help manage acute cravings. When used in individuals attempting to quit smoking, combination nicotine therapy has an absolute abstinence rate of 32 %, compared to 25 % for noncombination nicotine replacement.

Nicotine replacement therapy can be prescribed in pregnancy as systematic reviews do not show significant differences in serious adverse events between treatment and control groups in pregnancy. It is also clearly safer than tobacco smoke, which contains many toxins. The expert recommendation is for use of 16-hour nicotine patches in pregnancy, rather than 24-hour patches, to reduce unnecessary exposure to nicotine.

Varenicline is an oral nicotinic receptor partial agonist that binds less effectively than nicotine. The most significant unwanted effects of

varenicline are nausea and a possible increased risk of suicidal thoughts, especially in individuals with mental health problems. Bupropion is an oral nicotinic receptor antagonist with dopaminergic and adrenergic actions. It is contraindicated in individuals with a history of seizures. The absolute abstinence rates of varenicline and bupropion are 34 % and 25 % respectively. Unwanted effects include increased blood pressure and psychological effects. The safety of both varenicline and bupropion in pregnancy is unknown; therefore, the use of these medications in pregnancy should be avoided.

Question 4 of 133

A 45-year-old male with long standing asthma presents to his specialist doctor complaining of recurrent chest infections over the last year. He has a productive cough most mornings of thick, brownish sputum for most days of the last year and he feels it is getting worse. He has been to his general practitioner on a number of occasions and been treated with oral antibiotics and steroids but with no significant impact on symptoms. His general practitioner has also stepped up his asthma treatment to regular use of a long-acting beta-2 adrenergic agonist and an inhaled steroid, which he feels may have made a slight difference. He denies any fevers or night sweats but has lost approximately two kilograms in weight over the last year. He was diagnosed with asthma as a teenager, takes his inhalers regularly and has never had any hospital admissions. He has no other medical conditions and takes no other medications other than over the counter vitamin supplements. He drinks a moderate amount of alcohol and does not smoke. He works as an economist and travels regularly to the middle east and china.

On examination, he is comfortable at rest. His respiratory rate is 16 breaths/min, his oxygen saturation 95% breathing room air and his chest is clear to auscultation. His heart rate is 65 beats/min and his heart sounds are normal. There is no palpable lymphadenopathy in the axillae or groin.

What is the most likely diagnosis?

A. Cystic fibrosis
B. Allergic broncho-pulmonary aspergillosis
C. Tuberculosis
D. Lung cancer
E. Pseudomonas infection

B. Allergic broncho-pulmonary aspergillosis

This man's symptoms are suggestive of the development of bronchiectasis - thick sputum over a long period of time. There are many causes of bronchiectasis, most of which are unlikely in this case. The lack of risk factors or constitutional symptoms makes tuberculosis less likely. The worsening in asthma control (he has had to step up on his treatment) raises the possibility of ABPA (allergic bronchopulmonary aspergillosis), a diagnosis not often found in non-asthmatics. Investigation is with chest imaging and serum aspergillus precipitins levels. Treatment is with steroids and antifungals.

Question 5 of 133

A 53 year-old businessman presents to the neurology clinic complaining of weakness and numbness affecting his left hand. One month ago he was irritated when he noticed that the little finger of his left hand was often getting caught when he tried to put his hand in his pocket. Since then he has noticed progressive difficulty using the left hand, associated with an unpleasant tingling sensation.

In the last two weeks he has also noticed difficulty walking, and has tripped over on several occasions. When driving he finds that his right foot often becomes stuck behind the accelerator pedal and he struggles to lift it out.

On examination, in the left hand sensation to pin-prick is diminished over the little finger and medial side of the ring finger, as well as the medial side of the palm. There is weakness of finger abduction and adduction, but thumb abduction is normal. On examination of the legs, you note diminished sensation over the lateral aspect of the right calf, as well as the dorsum of the right foot. When asked to walk on his heels, he finds it difficult to do so, and trips over the right foot.

Investigations are as follows:

Haemoglobin	14.2 g/dl
WCC	7.1 x10^9/l
Platelets	420 x10^9/l
ESR	65 mm/hr
Na +	139 mmol/l
K +	4.3 mmol/l
Urea	13.2 mmol/l
Creatinine	171 µmol/l
Corrected calcium	2.26 mmol/l
ANCA	Positive, with perinuclear staining pattern
PR3 antibodies	Negative
MPO antibodies	Positive
Urine dipstick	+++ blood, +++ protein

Urine microscopy	Red cell casts
Chest radiograph	Clear

What is the most likely diagnosis?

A. Microscopic polyangiitis
B. Polyarteritis nodosa
C. Wegener's granulomatosis
D. Diabetes mellitus
E. Entrapment neuropathy

A. Microscopic polyangiitis

This is mononeuritis multiplex with ulnar and common peroneal neuropathy. The causes of mononeuritis multiplex include vasculitis, diabetes, AIDS, amyloidosis, and rheumatoid arthritis.

The elevated urea and creatinine, haematuria with red cell casts, and proteinuria, all suggest glomerulonephritis. The combination of mononeuritis multiplex with glomerulonephritis strongly suggests systemic vasculitis. This is supported by the raised ESR.

Microscopic polyangiitis is a small-vessel vasculitis which is typically positive for p-ANCA, with antibodies against MPO (myeloperoxidase). Other features may include systemic symptoms such as fever, weight loss, and fatigue, as well as rash.

ANCA is negative in classic polyarteritis nodosa.

Wegeners granulomatosis often features upper airway disease, and is typically positive for cANCA, with antibodies against PR3.

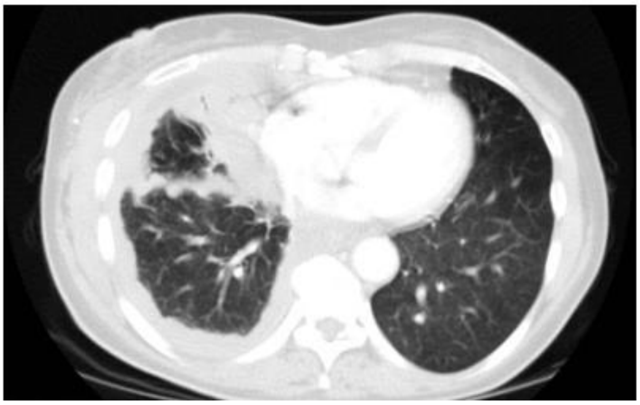
Diabetes mellitus is a common cause of many peripheral neuropathies including mononeuritis multiplex, however there is nothing in the history to suggest this. Haematuria and red cell casts are not a typical feature of diabetic nephropathy, and neither is ANCA positivity.

Entrapment is a common cause of both ulnar and common peroneal neuropathy, but for both to occur simultaneously would be unusual.

Question 6 of 133

A 75-year-old retired electrician presents with a 3-month history of progressive exertional dyspnoea, with associated chest pain. The chest pain is right-sided and is not worse on deep inspiration. He also describes intermittent haemoptysis and has noted 7kg of weight loss since the chest pain started.

A CT scan of his thorax is carried out, the images for which are given below:



What is the most likely cause of this imaging appearance?

A. Large cell carcinoma
B. Mesothelioma
C. Sarcoidosis
D. Small cell lung cancer
E. Tuberculosis

B. Mesothelioma

The CT shows concentric, nodular, pleural thickening. There also appears to be interlobular fissure pleural involvement. Pleural thickening can be due to a number of causes, one of which is **mesothelioma**. Other causes include pleural metastases, pleural lymphoma, chronic inflammation following empyema or recurrent pneumonia, and asbestos or silicosis-related lung disease. Pleural thickening can be differentiated from the appearance of a pleural effusion which is usually gravity dependent and crescent shaped.]

Mesothelioma is a cancer of the mesothelial layer of the pleural cavity that is strongly associated with asbestos exposure, which may have occurred due to this man's previous occupation as an electrician.

Small cell lung cancer is typically seen as a solid nodule within the lung, often located centrally in the hilar or peri-hilar region. They readily metastasise but do not generally involve the pleura.

Large cell lung carcinoma would usually manifest as a large peripheral mass of solid attenuation and irregular margin. There is not usually pleural involvement.

Sarcoidosis may show peri-lymphatic nodular thickening primarily affecting the upper/mid zones, ground glass opacities, cysts, or evidence of pulmonary fibrosis. It would not cause pleural thickening.

CT findings in pulmonary **tuberculosis** often show similar patterns of disease as seen with chest xrays, which would include fibronodular opacities in upper lobes with or without cavitation. Tuberculosis can involve the pleura but would present with a pleural effusion rather than pleural thickening as seen here.

Question 7 of 133

A 43-year-old Afro-Caribbean male presents with sudden onset palpitations and feeling generally unwell for 2 hours. An admission ECG in the emergency department demonstrates ventricular tachycardia. His heart rate is 80 beats/ minute with a blood pressure of 140/75mmHg. The patient is chemically cardioverted back to sinus rhythm with a single intravenous bolus of amiodarone. The patient was commenced on haemodialysis 9 months ago after developing end-stage renal failure over a course of 16 months with no conclusive underlying cause found for the deteriorating renal function. He also complains of a new dry cough over the past 18 months, weight loss of at least one and a half stone and general malaise, which he attributes to his deteriorating kidneys.

On examination, the patient has normal heart sounds with no additional murmurs. Auscultation of his chest demonstrates biapical find inspiratory crackles with no wheeze. Abdominal examination reveals a mild 2cm hepatomegaly with no splenomegaly. An arteriovenous fistula is noted in the left brachiocephalic region. No skin rashes are noted. An admission chest X-ray demonstrates no clear consolidation, reticular opacities in both apices and prominent bilateral hilar, with no cardiomegaly or tramlining.

Blood tests are as follows:

Hb	134 g/l
Platelets	292 * 10 /l9
WBC	12.5 * 10 /l9
Na +	131 mmol/l
K +	5.9 mmol/l
Urea	22.6 mmol/l
Creatinine	540 µmol/l
Bilirubin	17 µmol/l
ALP	55 u/l
ALT	70 u/l
CRP	12 mg/l
ACE (angiotensin converting enzyme)	negative

Pulmonary function tests: FVC 60% predicted FEV1 92% predicted

A bronchoalveolar lavage is performed, demonstrating lymphocytosis of 25%, CD4:CD8 ratio of 5:1, a transbronchial biopsy demonstrates non-caseating granulomas.

What is the underlying diagnosis?

A. Human immunodeficiency virus (HIV)
B. Miliary tuberculosis

C. Amyloidosis
D. Sarcoidosis
E. Lymphoma

D. Sarcoidosis

The patient has presented with multi-system disease with renal, cardiac, hepatic manifestations. In addition, the bihilar changes with apical interstitial changes, lymphocytosis, increased CD4:CD8 ratio and non-caseating granulomas on histopathology are strongly suggestive of sarcoidosis. A negative ACE is present in 25% of all patients with sarcoidosis, it has a poor negative predictive value. The most diagnostic test in differentiating against other multisystem disorders is histopathology in this case: Tb would produce caseating granulomas, lymphomas atypical lymphocytes and amyloid crossed beta sheets.

Question 8 of 133

A 78-year-old man presents to the acute medical unit with severe back pain. He described the pain as sudden onset sharp pain which reached maximum severity within seconds.

Investigation results are as follows:

Chest x-ray	Blunting of the left costophrenic angle
Pleural sample	Bloody in appearance

What is the most useful pleural fluid marker to make a diagnosis of a haemothorax?

A. pH
B. Lactate
C. Haematocrit
D. Lactate dehydrogenase
E. Triglyceride

C. Haematocrit

If the pleural fluid appears bloody, a haematocrit can be obtained if there is doubt as to whether it is a haemothorax. If the haematocrit of the pleural fluid is more than half of the patient's peripheral blood haematocrit, the patient has a haemothorax

The clinical features are suggestive of an aortic dissection which is a well known cause of a haemothorax. If the pleural fluid appears bloody, a haematocrit can be obtained if there is doubt as to whether it is a haemothorax. If the haematocrit of the pleural fluid is more than half of the patient's peripheral blood haematocrit, the patient has a haemothorax.

Question 9 of 133

A 72-year-old man presented with shortness of breath and haemoptysis. His past medical history includes polymyalgia rheumatica and Paget's disease. He is very fit and active and is independent of activities of daily living. He drives and does his own shopping, and attends the gym twice a week alongside tai chi lessons.

A chest x-ray revealed a left-sided mass.

CT thorax, abdomen, pelvis: There is a 4cm solid mass left-sided left upper lobe lung mass causing airway compression and partial lobe collapse. There is no evidence of distant metastases.

A biopsy of the lesion confirms adenocarcinoma.

What would be the most appropriate management option for this patient?

A. Chemotherapy
B. Complete resection with lobectomy and adjuvant chemotherapy
C. Complete resection with lobectomy and adjuvant radiotherapy
D. Pneumonectomy with adjuvant chemo-radiotherapy
E. Radiotherapy

C. Complete resection with lobectomy and adjuvant radiotherapy

Patients with stage I or II non-small cell lung cancer should be offered surgical resection if they are fit for surgery

Complete resection with lobectomy and adjuvant chemotherapy is the correct answer. This patient has stage II lung cancer (i.e. it is between 4-5cm, there is no distant spread, but there is blockage of the airway causing partial lobe collapse). All patients with stage I or II lung cancers should be offered surgical resection and adjuvant chemotherapy. This is done with curative intent.

Complete resection with lobectomy and adjuvant radiotherapy is incorrect. Radiotherapy can be offered as a sole treatment for patients with stage I or II NSLC who are not fit for surgery, and this is offered with curative intent. It is generally not used as an adjuvant treatment alongside surgical resection unless the resection is incomplete.

Pneumonectomy with adjuvant chemo-radiotherapy is incorrect. A pneumonectomy would not be the best option here as the mass is confined to the left upper lobe, a lobectomy would be preferable to preserve as much lung tissue as possible for the patient.

Radiotherapy is incorrect. This could be offered with curative intent if the patient was not fit for surgery, but here we have an active and well patient who should be given the opportunity to discuss surgical resection.

Chemotherapy is incorrect. Chemotherapy could be offered to patients with stage III or IV disease. Our patient here has stage II disease and so should be offered surgery. Stage III disease may include spread to mediastinal lymph nodes, spread to another lobe in the same lung, and invasion of the oesophagus, aorta, and mediastinum, for example.

Question 10 of 133

A 52-year-old man presents to the emergency department with shortness of breath. This started recently following a flight from Australia. He is morbidly obese and has a past medical history of type two diabetes mellitus, high cholesterol, hypertension and a previous cholecystectomy. He denies any leg pain or swelling and denies any chest pain. Observations are stable, and an ECG demonstrates sinus rhythm. A chest X-ray is clear. He undergoes a CT pulmonary angiogram (CTPA) to exclude a pulmonary embolus (PE). The CTPA is reported as finding no evidence of PE but notes a 7mm pulmonary nodule in the left lower lobe.

How should the pulmonary nodule be investigated or monitored?

A. Urgent CT-PET
B. Urgent image-guided biopsy
C. CT chest in three months
D. CT chest in one year
E. No further investigations needed

C. CT chest in three months

The correct answer is CT chest in three months. This is a pulmonary nodule which is larger than 5mm in size, and therefore has a significant potential of growing. Based on this, the British Thoracic Society (BTS) recommends surveillance. Only nodules which are greater than 8mm in diameter and have a greater than 10% risk of malignancy, as assessed by the Brock model (this can be accessed on the BTS website) should undergo PET-CT and then, based on outcomes, be assessed for obtaining a histological sample.

The timing of the follow-up CT is based on the nodule's size. A nodule of 5-6mm should be scanned at one year, whilst larger nodules should be scanned at three months (unless being otherwise investigated)

Question 11 of 133

A 60-year-old caucasian female attends the respiratory clinic for review, She has been referred by her GP after presenting complaining of a chronic cough which has persisted for 6 months. The cough is productive, however she denies any haemoptysis. The patient's husband has commented that she appears to have lost weight during this time period, and she admits to unintentionally going down a dress size.

Before the onset of her cough, the patient reports that she had been reasonably fit and well, walking half an hour every day. She has never smoked and does not have any underlying respiratory pathology. She denies any history of fever or night sweats and has recently traveled to Italy for a summer holiday where she visited a famous bird sanctuary.

You review her chest radiograph which was performed prior to the clinic. There is an increased opacity in the right upper lung field. You perform a number of further investigations detailed below:

Hb	140g/l	Na +	138 mmol/l
Platelets	400 * 10 /l 9	K +	5.0 mmol/l
WBC	11.0 * 10 /l 9	Urea	3.0 mmol/l
Neuts	6.6 * 10 /l 9	Creatinine	89 µmol/l
Lymphs	2.0 * 10 /l 9	CRP	18 mg/l
Eosin	0.01 * 10 /l 9	IFNγ-release assay	Negative
Sputum MC&S	Normal Flora	Sputum Acid Fast Bacilli (AFB)	Positive x 3

You also send the patient for a high resolution CT (HRCT) scan, which demonstrates a cavity in the right upper lobe with associated tree-in-bud appearances in the surrounding lung. There is no associated lymphadenopathy.

What is the most likely causative organism for this presentation?

A. Klebsiella species
B. Mycobacterium tuberculosis
C. Mycobacterium kansasii
D. Allergic bronco-pulmonary aspergillosis (ABPA)
E. Staphylococcal aureus

C. Mycobacterium kansasii

Non-tuberculous mycobacteria (NTM) pulmonary infection is increasing in prevalence within the non-HIV population. There are over 100 different species on NTM, the geographical distribution of which varies between region and country.

Diagnosis of pulmonary NTM infection can be challenging given the environmental nature of the organisms.

This question tests your understanding of the common presentations of pulmonary nontuberculous mycobacterium (NTM) infection in immunocompetent individuals. NTM infection in the non-HIV population is challenging diagnosis to make due to the environmental nature of NTM.

NTM can present in a variety of patterns commonly described based on the radiological characteristics demonstrated. The main types are:

- Hypersensitive-like disease
- Cavitating disease
- Bronchiectasis with or without nodules

Diagnosis can be made by following the joint American Thoracic Society / Infectious Diseases Society of America (ATS/IDSA) guidelines which were published in 2007. Diagnosis requires a range of clinical, radiological and microbiological characteristics to be fulfilled as described below:

Clinical: Pulmonary symptoms (e.g. cough, sputum, or constitutional) and exclusion of alternative diagnosis

Radiological: Chest X-ray or HRCT demonstrating cavities, bronchiectasis and or nodules Microbiological: Two or more positive sputum samples for an NTM species taken on separate days (or one of more positive sample from bronchoalveolar lavage or tissue biopsy)

In this question, although several of the alternative answers have the ability to cause cavitating disease of the lungs, the multiple positive AFB sputum samples point towards a mycobacterial cause. The negative IFNγ-release assay points away from the patient having tuberculosis, leaving Mycobacterium kansasii as the leading answer. However, it is worth noting that there are reports of cross reactivity between certain NTM species and IFNγ-release assay's.

Question 12 of 133

A 53-year-old man is reviewed in the respiratory outpatient clinic with progressive shortness of breath. He has a past medical history of chronic obstructive pulmonary disease and hypercholesterolaemia. He is on regular atorvastatin, salbutamol and symbicort. He still smokes 5 cigarettes daily and has a 40 pack-year history. He lives with his wife and his exercise tolerance is approximately 30 yards.

Clinical examination reveals hyper-resonance to percussion bilaterally and a barrel-shaped chest. There is moderate harsh-sounding wheeze on auscultation. The jugular venous pulse is not raised and there is no peripheral oedema. The heart sounds are normal and the pulse is regular. His fingertips are stained with nicotine. There is no clubbing.

Lung function tests:

FEV1	35% (predicted)
FVC	68% (predicted)
FEV1/FVC	51.4%

Blood tests:

Hb	154 g/L	Male: (135-180)Female: (115 - 160)
Platelets	211 * 10 /L 9	(150 - 400)
WBC	7.2 * 10 /L 9	(4.0 - 11.0)
Na +	133 mmol/L	(135 - 145)
K +	4.4 mmol/L	(3.5 - 5.0)
Urea	5.3 mmol/L	(2.0 - 7.0)
Creatinine	99 µmol/L	(55 - 120)
Bilirubin	14 µmol/L	(3 - 17)
ALP	91 u/L	(30 - 100)
ALT	34 u/L	(3 - 40)
Î³GT	66 u/L	(8 - 60)
D-dimer	333 ng/ml	(<500)
Albumin	36 g/L	(35 - 50)
CRP	4mg/L	(<5)
BNP	88 pg/ml	(<100)

Plain radiography of the chest demonstrates generalized increased lucency bilaterally but clear lung fields.

Given the clinical history, what is the appropriate next step in management?

A. Add a long-acting beta antagonist
B. Add furosemide and switch salbutamol to ipratropium bromide
C. Add theophylline
D. Switch salbutamol to ipratropium bromide
E. Add a long acting muscarinic antagonist (LAMA)

E. Add a long acting muscarinic antagonist (LAMA)

COPD - still breathless despite using SABA/SAMA and a LABA + ICS → add a LAMA

Add a long-acting muscarinic antagonist (LAMA) is correct. This patient has worsening shortness of breath on a background of COPD. Common differential diagnoses have been excluded such as pulmonary embolism (normal d-dimer), heart failure (clinical examination and normal BNP) and infection (normal WCC, CRP and chest x-ray). The explanation for his symptoms is therefore worsening COPD. If patients are still breathless despite using a SABA and LABA + ICS (i.e. symbicort), then a LAMA should be added.

Add a long-acting beta antagonist is incorrect. The patient is already taking a combined LABA + inhaled corticosteroids (i.e. symbicort).

Add theophylline is incorrect. This should only be trialled once inhaled options have been exhausted.

Switch salbutamol to ipratropium bromide is incorrect. This will not be sufficiently effective.

Add furosemide and switch salbutamol to ipratropium bromide is incorrect. The patient has no clinical evidence of heart failure or fluid overload and has a normal BNP. Therefore furosemide is not indicated.

Question 13 of 133

A 67-year-old gentleman presents to the respiratory clinic via the 2-week rule. He presented to his general practitioner with haemoptysis and a persistent cough. On examination in the clinic, you observe that his clothes are loosely fitted. Chest percussion reveals left upper zone dullness. Chest X-ray confirms the presence of a left upper zone mass. After discussion with your consultant an urgent staging Computerised Tomography (CT) is arranged which unfortunately confirms a large left upper zone mass, mediastinal lymph node involvement and hepatocellular metastases. His performance status is 1. Hyponatraemia is noted on the bloods you requested in the clinic.

Na +	126 mmol/l
K +	4.1 mmol/l
Urea	7.5 mmol/l
Creatinine	91 µmol/l

In the context of the most likely diagnosis and staging , what would be the most appropriate treatment option?

A. Lobectomy
B. Partial pneumonectomy
C. Palliation
D. Chemotherapy
E. Radiotherapy

D. Chemotherapy

From the clinical details given the candidate would be expected to understand that the most likely underlying diagnosis is small cell lung carcinoma. Hyponatraemia secondary to syndrome of inappropriate antidiuretic hormone secretion (SIADH) is a classic MRCP association.

Small cell lung carcinoma has a poor prognosis. National Institute for Clinical Excellence (NICE) guidelines state that in the context of small cell carcinoma with extensive disease chemotherapy should be offered as a treatment option provided after multidisciplinary team discussion the patient is deemed fit enough.

There is no role for surgical resection in small cell carcinoma, however, radiotherapy is an option in those with limited disease.

Question 14 of 133

A 60-year-old woman is referred to the general respiratory clinic with increasing shortness of breath over the last nine month months. This has been associated with pedal oedema. There is no orthopnoea, paroxysmal nocturnal dyspnea or cough. Her past medical history includes bilateral extensive pulmonary emboli and she is currently taking warfarin. She has no other lung disease and she does not smoke.

On examination her chest is clear but auscultation demonstrates a loud second heart sound. There is also a right ventricular heave with visible V-waves on jugular venous pressure.

A recent CT pulmonary angiogram showed resolution of the thrombus. Echocardiogram demonstrates severe right heart dilatation and impaired function with moderate tricuspid regurgitation.

What test will most likely provide a formal diagnosis?

A. Cardiac MR imaging
B. Right heart catheterisation
C. Left heart catheterisation
D. Brain natriuretic peptide (BNP) measurement
E. 6-minute walk test

B. Right heart catheterisation

This lady has right sided heart failure. The most likely diagnosis is chronic thromboembolic pulmonary hypertension and she requires a right heart catheterization to assess the pulmonary pressures (pulmonary hypertension = pulmonary arterial pressure 25 mmHg). Note these can be estimated on echocardiogram and cardiac MR imaging but should not be used to formally diagnose the condition. A cardiac MR can be used to further assess the right ventricular volumes, mass and function or for longitudinal follow up of patients treated for pulmonary hypertension.

A left heart catheterization is used to assess the coronary arteries and left side of the heart which should be relatively normal in this lady.

The BNP will invariably be raised given the severe right ventricular impairment and dilatation.

The 6-minute walk test is used as a prognostic measurement in pulmonary hypertension.

Question 15 of 133

A 67-year-old man presents to the emergency department with severe shortness of breath. He has a past medical history of COPD, type 2 diabetes mellitus and hypertension. On examination he appears very breathless and looks drowsy, with bilateral crackles and wheeze audible on auscultation. Following treatment with nebulisers, steroids, antibiotics and fluids he remains in type 2 respiratory failure with acidosis.

Arterial blood gas on admission:

pH	7.32
pO 2	7.3kPa
pCO 2	8.1kPa
HCO - 3	25mmol/L

Arterial blood gas at two hours:

pH	7.30
pO 2	8.2kPa
pCO 2	9.6kPa
HCO - 3	26mmol/L

He is started on non-invasive ventilation with a bi-level positive airway pressure.

What initial settings would be most appropriate for IPAP and EPAP?

A. IPAP 3cmH2O and EPAP 15cmH2O
B. IPAP 10cmH2O and EPAP 10cmH2O
C. IPAP 15cmH2O and EPAP 3cmH2O
D. IPAP 20cmH2O and EPAP 7cmH2O
E. IPAP 20cmH2O and EPAP 3cmH2O

C. IPAP 15cmH2O and EPAP 3cmH2O

The correct answer is IPAP 15cmH O and EPAP 3cmH O.

BTS advises that BiPAP should started with IPAP of 15cmH O in COPD. If the pH <7.25 then start with IPAP at 20cmH O. For neuromuscular disease needing NIV then starting with IPAP at 10cmH O is appropriate.

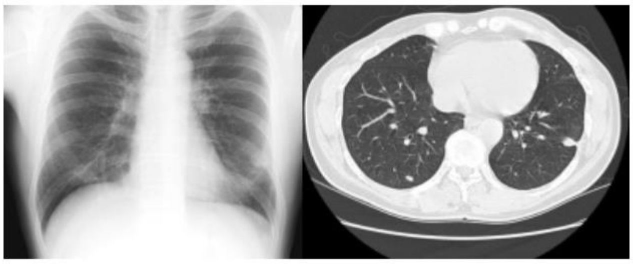
EPAP should be started at 3cmH O unless obstructive sleep apnoea is expected according to the British Thoracic Society, but it is common practice to start with an IPAP of 10cmH O and an EPAP of 4cmH2O. In either case, IPAP should be uptitrated with an aim of IPAP of 20cmH2O or as high as .the patient can tolerate

Question 16 of 133

A 48-year-old man presents to the respiratory clinic with a 2-month history of progressive shortness of breath and a dry cough. There is no history of fever or chest pain, although, he does complain of worsening lethargy. He works as a school teacher and visited India 6 months prior. He denies occupational exposure to hazardous chemicals and has never smoked. His past medical history includes childhood asthma, type 1 diabetes, and rheumatoid arthritis.

On examination, his observations are within normal limits. On auscultation, there are audible fine crackles at the bases with normal heart sounds. His calves are soft and non-tender with no visible oedema.

A chest x-ray and CT chest are organised, as shown below.



Which of the following conditions is the most likely diagnosis?

A. Allergic bronchopulmonary aspergillosis
B. Ankylosing spondylitis
C. Rheumatoid arthritis
D. Sarcoidosis
E. Tuberculosis

C. Rheumatoid arthritis

Causes of lower zone fibrosis: **MAID**

Most connective tissue diseases (e.g. rheumatoid arthritis)

Asbestosis

Idiopathic pulmonary fibrosis, infection

Drugs (e.g. methotrexate, amiodarone, bleomycin)

This patient's chest x-ray and CT scan show features of lower zone fibrosis with visible honeycombing on the CT scan. On closer inspection of the chest x-ray, there is a visible wellcircumscribed opacity in the left lower zone in keeping with a pulmonary nodule (e.g. rheumatoid nodule), and multiple smaller opacities can be seen throughout the slice of the CT scan. Of the conditions listed in the question, rheumatoid arthritis is the only disease that causes lower zone fibrosis, with the other conditions classically causing a pattern of fibrosis in the upper zones. Furthermore, the history of worsening lethargy and arthralgia are suggestive of rheumatoid arthritis, which commonly has an insidious onset over time.

Allergic bronchopulmonary aspergillosis (ABPA) causes a clinical picture similar to asthma and arises following exposure to *Aspergillus* spores. Symptoms include cough, wheeze, and shortness of breath. CT imaging shows dilated bronchioles (similar to bronchiectasis) and occasionally a 'finger-in-glove' appearance can be seen that represents the mucous filling of dilated bronchi. Lower zone pulmonary fibrosis is not a feature of ABPA.

Ankylosing spondylitis is a spondyloarthropathy that typically presents as a young man with lower back pain and stiffness that is worse in the morning. Ankylosing spondylitis causes apical fibrosis, unlike the changes seen on this CT chest that shows lower zone fibrosis.

Sarcoidosis is a multisystem disorder that causes respiratory symptoms such as shortness of breath and a dry cough, along with extra-pulmonary manifestations including fever, arthralgia, and erythema nodosum. Imaging findings in sarcoidosis include bilateral hilar lymphadenopathy, interstitial infiltrates, and fibrosis (typically in the upper lobes).

Tuberculosis is caused by the bacterium *Mycobacterium tuberculosis* with symptoms consisting of fever, productive cough, lethargy, night sweats, and weight loss. In contrast, the patient in this question describes progressive shortness of breath and a dry cough. Tuberculosis does not commonly cause fibrosis, but instead causes calcified lesions (typically apical in location).

Question 17 of 133

A 65-year-old woman slips on an icy road and has a neck of femur fracture. Her past medical history includes hypertension, type 2 diabetes and a pack-year history of 40 years. After six days in hospital, she becomes short of breath and develops haemoptysis. Her observations are:

- Blood pressure: 135/70 mmHg
- Pulse: 110 bpm
- Temperature: 36.4 deg C
- Respiratory rate: 24/min
- Oxygen saturation: 89% on air

A subsequent CTPA shows a right segmental pulmonary embolus and a 4mm solid nodule in her right upper lobe.

What is the most appropriate management of her nodule?

A. Antibiotics and repeat CT
B. Biopsy of lung nodule
C. No further follow-up required
D. PET-CT
E. Repeat CT in 3 months

C. No further follow-up required

Solitary, solid and non-calcified lung nodules of less than 5mm in size do not warrant further investigations or monitoring

Lung nodules are a common incidental finding in patients who have undergone CT scans. BTS guidelines recommend that solid nodules that are less than 5mm in size do not require any further follow-up, even in a smoker.

While chest infections are a common complication after surgery, there is no indication this patient has a chest infection and her CTPA did not show pneumonia. Antibiotics are therefore not indicated.

Larger (>8mm) nodules are more likely to be malignant, especially in patients who are smokers. Patients with such nodules should be ideally offered a PET-CT to help determine the risk of malignancy by combining nodule characteristics with patient characteristics. Patients with a higher risk of malignancy may be offered several options, including a biopsy to confirm malignancy or active surveillance such as a repeat CT in 3 months. This patient's nodule is too small.

Question 18 of 133

A 71-year-old man presents to his GP having suffered from daytime sleeping, which has become so interrupting that he has been placed on probation by his manager at work. He complains of having frequent morning headaches and often gets breathless on walking to the shops. He used to smoke 20 cigarettes per day for 20 years and drinks a pint of beer per evening at the pub. He is also clinically obese.

After being referred to the hospital, an evening arterial blood test is performed and reveals a PaO2 of 9.9 kPa and a PaCO2 of 4.4 kPa. The arterial blood gas is repeated the next morning and reveals a PaO2 of 10.0 kPa and a PaCO2 of 9.1 kPa.

What is the most likely diagnosis?

A. Pulmonary hypertension
B. Central sleep apnoea
C. Obesity hypoventilation syndrome
D. Benign intracranial hypertension
E. Multiple pulmonary emboli

C. Obesity hypoventilation syndrome

This patient most likely has obesity hypoventilation syndrome as characterised by his raised carbon dioxide levels in the morning, as opposed to the evening.

Question 19 of 133

A 67-year-old patient is reviewed in oncology clinic. He has recently been diagnosed with mesothelioma following a pleural biopsy. He has a past medical history of hypothyroidism, pseudogout and depression. He is concerned about the financial implications and is due to seek compensation. He has worked as a plumber for all of his professional life and wants to know when the asbestos exposure most likely occurred to cause him to develop mesothelioma.

How long ago would his asbestos exposure occurred?

A. >50 years ago
B. >30 years ago
C. >10 years ago
D. >5 years ago
E. >2 year ago

B. >30 years ago

The correct answer is >30 years ago. This is a gentleman with mesothelioma. It normally takes at least 10 years for pleural plaques to develop, 20 years for asbestosis and finally 30 years for mesothelioma. To develop mesothelioma a heavy exposure of asbestos will have occurred.

Question 20 of 133

A 71-year-old woman is referred by her out-of-hours GP service to the emergency department following her presentation with worsening shortness of breath and cough. The shortness of breath has been increasing in severity over the last 3 weeks and is associated by an occasional nonproductive cough.

She has an extensive cardiac past medical history including two myocardial infarctions which were treated with percutaneous coronary intervention, an aortic heart valve replacement and mild mitral regurgitation. Other history includes hypertension which is treated with amlodipine and ramipril, hypercholesterolaemia which is treated with atorvastatin and mild COPD for which he takes salbutamol and ipratropium bromide. She previously worked as a librarian, has smoked 10 cigarettes for the past 20 years and drinks on average 5 units of alcohol per week.

Examination reveals dull percussion and reduced breath sounds over the left mid and lower bases of the lung. Urgent chest x-ray reveals a left-sided pleural effusion. Pleural aspirate is taken and shows:

Protein content	45 g/L
Glucose	2.4mmol/L
pH	7.32

What is the most likely diagnosis?

A. Congestive cardiac failure
B. Bronchial carcinoma
C. Systemic lupus erythematosus
D. Mesothelioma
E. Boerhaave's syndrome

B. Bronchial carcinoma

The protein content of the pleural fluid is high (>30g/L) demonstrating that the fluid is an exudate. The low glucose and pH of the fluid, when coupled with the clinical symptoms of shortness of breath and cough indicates that the most likely diagnosis is bronchial carcinoma. There is no history of asbestos exposure and no clinical feature of systemic lupus erythematosus or Boerhaave's syndrome present.

Question 21 of 133

A 56-year-old woman with a diagnosis of asthma attends her respiratory outpatient appointment. She has had two exacerbations requiring hospitalisation over the past year and also has hypothyroidism and angina. She takes tiotropium, fluticasone, levothyroxine, simvastatin, aspirin and GTN spray. She was started on theophylline at a standard regime in the previous clinic appointment and has completed a course of ciprofloxacin the last week for a mild exacerbation. She is otherwise well and smokes 20 cigarettes a day. She has allergies to pollen.

On examination, she has a dry cough but is able to speak in full sentences. Her chest has scattered wheeze throughout.

Na +	145 mmol/l
K +	3.9 mmol/l
Urea	4.2 mmol/l
Creatinine	56 µmol/l
Chest x-ray	hyperexpanded lung fields
Theophylline level	6 mcg/ml (normal range 10-20)
TSH	5.2 mU/l

What is the likely cause of her subtherapeutic theophylline level?

A. Ciprofloxacin
B. Simvastatin
C. Smoking
D. Alcohol binge
E. Hypothyroid state

C. Smoking

Current smokers -> liver enzyme induction -> higher doses aminophylline required

Theophylline/aminophylline requirements vary between individuals. They are metabolised by the liver and therefore concentration depends on the rate of metabolism. Statins, ciprofloxacin and an alcohol binge or inhibit the liver increasing sensitivity to theophylline and increasing the risk of toxicity at smaller doses. In contrast, smoking is an inducer of liver enzyme activity and therefore smokers often require higher starting doses. The TSH suggests she is slightly hypothyroid at present but this does not significantly affect the metabolism.

Question 22 of 133

A 71-year-old man is being treated in a medical ward for an empyema. Shortly after admission, a chest drain is inserted under ultrasound guidance and antibiotics are started. Over the past few days, there have been significant volumes drained and the patient has been improving clinically.

On the daily morning ward round, his chest drain is reviewed. It is on suction. There is minimal drain output over the past 24 hours and it is noted that the chest drain is bubbling. This bubbling is more significant when the patient is asked to cough.

What does this indicate?

A. Blocked chest drain
B. Bronchopleural fistula
C. Chest drain moved displaced outside of the chest
D. Normal
E. Unresolved empyema

B. Bronchopleural fistula

Bubbling of a chest drain inserted to drain an empyema may indicates that a bronchopleural fistula has occurred

Bubbling of a chest drain inserted into the pleural space is indicative of an air leak. Therefore, given the clinical history of an empyema, it is likely that a bronchopleural fistula has formed. This is therefore the correct answer.

As there is bubbling of the chest drain, it cannot be blocked. This is therefore an incorrect answer.

If the chest drain is displaced outside of the chest and on suction, bubbling can occur. Distinguishing between a bronchopleural fistula and a displaced chest drain can be difficult however one simple test is to ask the patient to cough. As the patient coughs and the intrathoracic pressure rises, this will cause an increase in the air flowing into the pleural space and therefore the bubbling of the drain. If the drain is outside of the pleural space, the bubbling will be constant and unrelated to coughing.

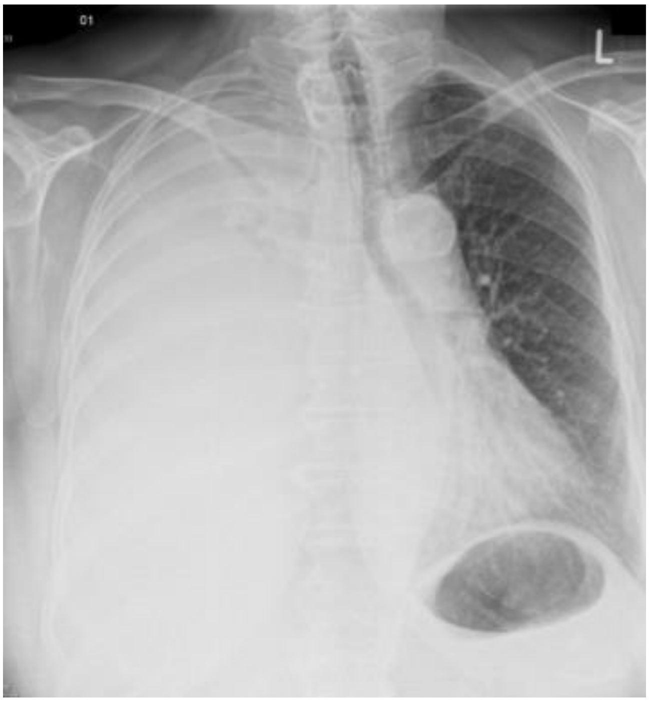
Bubbling from a chest drain is not a normal finding . Although swinging is normal with changes in intrathoracic pressure, bubbling is caused by air being entrained into the drain. The most common cause for this is the displacement of the drain outside of the thorax, however, a bronchopleural fistula is the diagnosis here as indicated by the changes on coughing.

An unresolved empyema is not necessarily indicated here by the drain activity as this would not cause bubbling of the chest drain. The drain is clearly patent, as indicated by the bubbling which is worse on coughing, and there is minimal drain output. It may be that the drain is not located within the empyema limiting its resolution, however further imaging would be required to determine this.

Question 23 of 133

A 69-year-old woman presents to the Emergency Department with a one month history of gradually worsening breathlessness. She is generally fit and well but last year was diagnosed with chronic obstructive pulmonary disease (COPD) after a 50-pack-year history of smoking. Around 10 years ago she retired from the local car production plant. On examination she is breathless at rest with a respiratory rate of 18/min and oxygen saturations of 93% on room air.

A chest x-ray is performed:

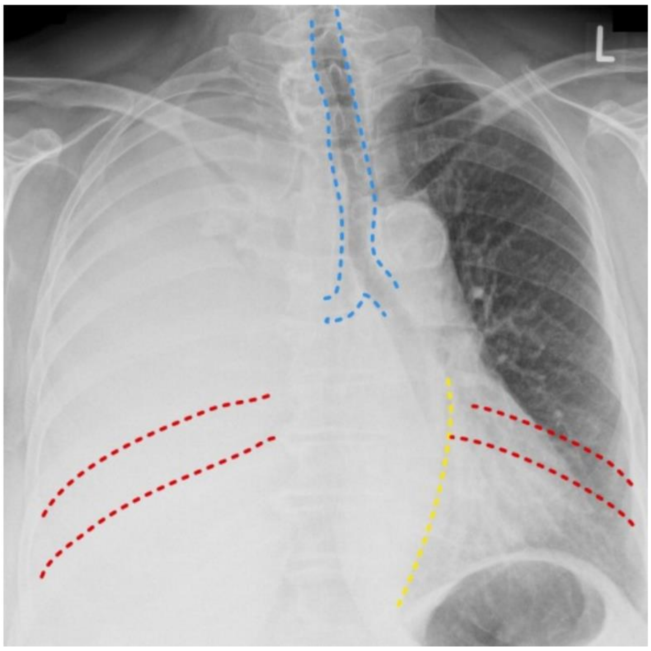


What is the most likely diagnosis?

A. Pneumonectomy
B. Mesothelioma
C. Lung collapse
D. Massive pleural effusion
E. Ewing sarcoma

D. Massive pleural effusion

The chest x-ray shows that the the trachea (blue) is pushed away the side of the white-out. The other signs of a positive mass effect include leftward bowing of the azygo-oesophageal recess (yellow) and splaying of the ribs on the right.



Question 24 of 133

A 39-year-old woman is seen in the respiratory clinic with a 6-week history of progressive shortness of breath and a dry cough. There is no history of fever or weight loss. She has a past medical history of recently-diagnosed asthma for which she is taking regular salbutamol and beclomethasone inhalers although, she reports that they have had little effect on her symptoms. There is no history of smoking and she denies any allergies.

On examination, there is diffuse expiratory wheeze but no audible crackles. Her cardiovascular examination is unremarkable.

Laboratory tests:

Hb	136 g/L	(115 - 160)
Platelets	201 * 10 /L 9	(150 - 400)
WBC	9.1 * 10 /L 9	(4.0 - 11.0)
Lymphocytes	1.6 * 10 /L 9	(1.5 - 4.5)
Neutrophils	5.5 * 10 /L 9	(2.0 - 7.5)
Eosinophils	1.2 * 10 /L 9	(0 - 0.4)
CRP	12 mg/L	(< 5)
Aspergillus IgE		Raised

What is the most appropriate next step in the management of this patient?

A. Intravenous amphotericin B
B. Intravenous co-trimoxazole and methylprednisolone
C. Oral montelukast
D. Oral prednisolone
E. Salbutamol and ipratropium nebulisers

D. Oral prednisolone

Oral glucocorticoids are the treatment of choice for allergic bronchopulmonary aspergillosis

The diagnosis here is allergic bronchopulmonary aspergillosis (ABPA). It is a hypersensitivity reaction to the fungus *Aspergillus* causing an eosinophilic pneumonia. Presenting symptoms are similar to asthma and include dyspnoea, dry cough and wheeze. Patients with ABPA are commonly misdiagnosed as having asthma and standard treatments for asthma (e.g. salbutamol inhalers) have little benefit. This patient describes similar symptoms of shortness of breath and a dry cough. Furthermore, her blood tests demonstrate a raised eosinophil count and IgE level, both of which point towards ABPA. The treatment of choice is oral prednisolone to help facilitate the clearing of pulmonary infiltrates.

Intravenous amphotericin B is used in the treatment of systemic fungal infections, including invasive *Candida albicans* or invasive aspergillosis. Invasive aspergillosis is uncommon and is mostly seen in immunosuppressed patients and presents with systemic illness. Amphotericin B is not routinely given first-line to treat infections with *Aspergillus*. Instead, fluconazole or itraconazole can be given, if indicated. However, the combination of wheeze, shortness of breath, and dry cough are more in keeping with ABPA, for which oral corticosteroids are the most appropriate treatment.

Intravenous co-trimoxazole and methylprednisolone are used in the treatment of *Pneumocystis jiroveci* pneumonia (PCP). PCP presents in a similar way to this patient with dyspnoea, fever and a dry cough. However, PCP is mostly seen in immunocompromised patients, such as those with HIV or on immunosuppressant medications. There is nothing in this patient's history to suggest immunosuppression, therefore it is likely that PCP is not the underlying diagnosis.

If asthma is not controlled with salbutamol and inhaled corticosteroid inhalers, the dose of the corticosteroid can be increased. Failing this, the next step in the management of asthma includes the addition of a leukotriene antagonist such as montelukast. However, this patient's history and laboratory findings point toward a diagnosis of ABPA over poorly controlled asthma, for which oral corticosteroids is the preferred method of treatment.

Salbutamol and ipratropium nebulisers are used in the acute treatment of asthma. However, it plays no role in the treatment of ABPA.

Question 25 of 133

A 35-year-old female with a past medical history of sinusitis and asthma for four years is admitted with a pyrexia and new onset dyspnoea and ankle swelling. Two weeks prior to admission she had noticed blood streaks were present in her sputum.

On examination, she had widespread wheeze and bi-basal crepitations on auscultation of her chest. Her saturations are 94% without oxygen but come up to 99% after treatment with salbutamol and ipratropium bromide nebulisers and oxygen therapy.

She has a heart rate of 112 beats per minute, normal heart sounds and a raised JVP. She has pitting oedema up to her knees. Examination of her abdominal system is unremarkable.

Haemoglobin	102 g/L
White cell count	11.2 Ã— 109/L
Eosinophil count	1.9 Ã— 109/L
Platelet count	478 Ã— 109/L
International normalised ratio	1.3
Serum sodium	135 mmol/L
Serum potassium	5.5 mmol/L
Serum urea	21 mmol/L
Serum creatinine	211 umol/L
CRP	22 mg/l
Blood cultures	Coagulase-negative staphylococci
ANCA positive	immunostaining awaited

Urine dip- positive for protein and blood

CXR: Bi-basal non specific shadowing

What is the most likely diagnosis?

A. Churg-Strauss syndrome
B. Systematic inflammatory response syndrome
C. Systematic lupus erythematous
D. Granulomatosis with polyangiitis
E. Microscopic polyangiitis

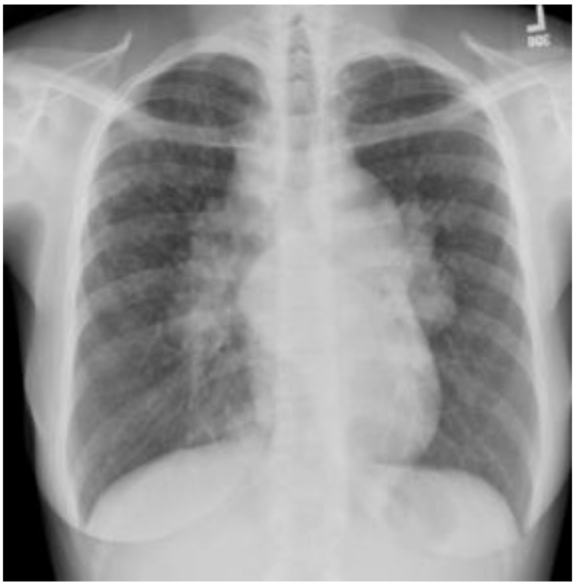
A. Churg-Strauss syndrome

This patient has essentially presented with acute kidney injury and is ANCA positive. ANCA may be positive in granulomatosis with polyangiitis, Churg Strauss syndrome and microscopic polyangiitis all of which can affect the kidneys. However, the history of asthma and sinusitis coupled with a raised eosinophil count make Churg Strauss syndrome the most likely option.

Systematic Inflammatory response syndrome is unlikely to explain the entire clinical picture. Additionally, although blood cultures are positive this is most likely to be a contaminant considering the organism and the trivial C-reactive protein.

Question 26 of 133

A 51-year-old woman is being investigated by the respiratory team. She reports a four-month history of dry cough with associated lethargy and weight loss. She has no relevant past medical history and has never smoked. A chest x-ray is displayed below:



Which further test would be most appropriate to aid in diagnosis?

A. Cytoplasmic antineutrophil cytoplasmic antibodies (cANCA) 3%
B. Interferon-gamma release assay (IGRA) 6%
C. Perinuclear antineutrophil cytoplasmic antibodies (pANCA) 2%
D. Serum angiotensin-converting enzyme (ACE) 84%
E. Sputum microscopy, culture, and sensitivity (MCS) 5%

D. Serum angiotensin-converting enzyme (ACE)

This woman has sarcoidosis. This explains her clinical symptoms and is confirmed by the bilateral lymphadenopathy on her chest x-ray. Serum ACE is a specific blood biomarker for sarcoidosis and is, therefore, the correct answer in this instance.

cANCA is a biochemical marker for granulomatosis with polyangiitis (previously known as Wegner's granulomatosis). This classically presents with haemoptysis, rhinitis, epistaxis, and potentially renal involvement. The typical x-ray findings include nodules with or without cavitations, pulmonary haemorrhages, and peripheral consolidation. As none of these symptoms or imaging features are to be found in this case, this is not the diagnosis and therefore pANCA is incorrect.

IGRA is a test used to diagnose tuberculosis (TB) infection. Although the symptoms here could be indicative of active pulmonary TB, this classically presents with upper lobe consolidation on chest x-ray. As this is not present, TB is not the diagnosis here. Therefore an IGRA would not be an appropriate test to order in this instance.

pANCA is a biochemical marker for eosinophilic granulomatosis with polyangiitis (previously known as Churg-Strauss syndrome). This typically presents with asthmatic features as well as other sequelae of vasculitis. The x-ray findings in eosinophilic granulomatosis with polyangiitis include peripheral consolidation and pleural effusions. As none of these symptoms or imaging features are to be found in this case, this is not the diagnosis and therefore pANCA is incorrect.

The x-ray here shows bilateral hilar lymphadenopathy rather than focal areas of consolidation and is, therefore, more indicative of sarcoidosis. Sputum MCS is very effective in identifying organisms and assessing sensitivities in infective pathologies however have no role in sarcoidosis as this is an autoimmune process. This is therefore an incorrect answer.

Question 27 of 133

A 50-year-old gentleman presents with shortness of breath. He is a current smoker of 20 cigarettes per day and used to work a coal miner. His body mass index (BMI) is 40 kg/m². His spirometry results are as follows:

Forced expiratory volume in 1 second (FEV1)	3.05 (79% predicted)
Forced vital capacity (FVC)	3.27 (70% predicted)
Diffusion capacity of the lung (DLCO)	86% predicted
Rate of carbon monoxide uptake (KCO)	105% predicted

What is the likely cause of this gentleman's shortness of breath?

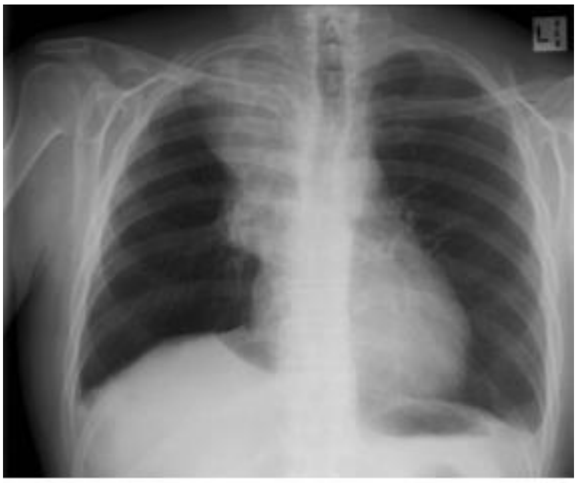
A. Obesity
B. Pneumoconiosis
C. Chronic obstructive pulmonary disease (COPD)
D. Idiopathic pulmonary fibrosis
E. Asthma

A. Obesity

This gentleman has evidence of restrictive lung disease on his spirometry results. A low DLCO but a high KCO implies that he has an extra-pulmonary cause of his restriction. Obesity is the only extra-pulmonary cause of a restrictive deficit from the list of options. COPD and asthma cause obstructive lung disease and pneumoconiosis and idiopathic pulmonary fibrosis and intrapulmonary causes of restrictive lung disease.

Question 28 of 133

A 79-year-old is investigated for weight loss.



What is the most likely diagnosis?

A. Paget's disease of the bone
B. Lung cancer
C. Liver cancer
D. Reactivation of pulmonary tuberculosis
E. Mesothelioma

B. Lung cancer

The x-ray shows a large right sided hilar mass with resultant collapse of the right upper lobe. As a consequence the hyperexpanded right middle and lower lobes are hyperlucent ('blacker').

Question 29 of 133

A 56-year-old woman presents to clinic for review following investigations and MDT discussion. She has chronic exertional dyspnoea and following a restrictive pattern detected on spirometry and changes on high-resolution CT scanning she has been diagnosed with idiopathic pulmonary fibrosis.

What medication can be used to modify disease progression?

A. Azathioprine
B. Prednisolone
C. Co-trimoxazole
D. Sildenafil
E. Pirfenidone

E. Pirfenidone

The correct answer is pirfenidone. Guidelines recommend supportive care, pulmonary rehabilitation and supplementary oxygen, but in very selective patients pirfenidone can be used. All of the other medication options have been attempted, but are advised by NICE that there is insufficient evidence to recommend their use. Patients are commonly prescribed azathioprine and prednisolone but this does not affect the disease course and does not help symptom control. Cotrimoxazole is useful primarily in immunosuppression from HIV to prevent infection, whilst sildenafil, a type 5 phosphodiesterase inhibitor, is more useful in pulmonary arterial hypertension.

Question 30 of 133

A 52-year-old woman presents to the respiratory clinic with breathlessness. She was referred from her GP who was concerned about breathlessness and a chest X-ray demonstrating left pleural effusion. She had recently undergone a left-sided wide local excision with axillary clearance for ductal carcinoma in sit.

She has unilaterally reduced breath sounds with dullness on percussion and deviation of the trachea away from that side of her chest. A plural aspirate is obtained and is milky white in colour. The two possibilities considered are chylothorax from pseudochylothorax.

What investigation would aid in distinguishing chylothorax from pseudochylothorax?

A. pH
B. Albumin
C. Microscopy
D. Triglycerides
E. Amylase

D. Triglycerides

The correct answer is triglycerides. Chylothorax is the accumulation of lymph in the pleural space. This can occur following damage to the thoracic duct in surgery or in malignant melanoma. It is most likely that the recent surgery is relevant to this. A chylothorax is high in triglycerides and chylomicrons whilst a pseudochylothorax would be high in cholesterol. A pseudochylothorax occurs with longstanding fibrotic pleura. pH would be most useful in determining the presence of empyema, amylase in pancreatitis associated effusions, whilst microscopy would help in malignant effusions.

Question 31 of 133

You are asked to review a 78 year-old male on the respiratory ward during a night shift. He is currently an inpatient being treated for an infective exacerbation of pulmonary fibrosis. You review his chest X-Ray which shows extensive left sided consolidation. His co-morbidities include endstage renal failure (haemodialysis dependent) secondary to type 1 diabetes mellitus. He escalation plan has been discussed previously and he did not want escalation to ICU or CPR in the event of a cardiac arrest.

He has been treated with high flow oxygen and intravenous antibiotics for the past few days but nursing staff inform you he has been deteriorating in the last 24 hours. On examination he is very dyspnoeic and distressed with saturations of 72% on 80% high flow humidified oxygen.

What is the correct course of action?

A. Increase the inspired oxygen concentration
B. 2.5mg midazolam subcutaneously
C. 2.5mg morphine subcutaneously
D. 5mg morphine orally
E. 100mcg alfentanil subcutaneously

E. 100mcg alfentanil subcutaneously

This gentleman is approaching the end of his life. He is on maximal oxygen therapy and has expressed a wish not for escalation beyond ward based care. His renal failure means that he is likely to accumulate morphine and the most appropriate option for palliation of his breathlessness is alfentanil - a short acting opiate.

Question 32 of 133

A 56-year-old lady is brought into the emergency department with a Glasgow Coma Scale of 5. She was found by a friend this morning unconscious and purple with no response. She had been unwell the previous days with an exacerbation of her COPD in which she was developing severe pleuritic chest pain. She had recurrent exacerbations of her COPD and had been hospitalised three times this year with one admission to ITU for intubation and ventilation.

In addition, she had hypertension, hypothyroidism and chronic regional pain syndrome. Her medications include fostair, ventolin, gabapentin, codeine, paracetamol, amlodipine, ramipril, levothyroxine and morphine sulfate. She had taken extra doses of oramorph to control her pleuritic pain. She has started a rescue pack of amoxicillin and prednisolone one day prior

On examination, she does not open her eyes which have 2mm pupils bilaterally that are reactive. She groans to pain but there is no motor response. Her chest has some wheeze across and her respiratory rate is 9 breaths per minute. She is saturating at 88% on 4 litres oxygen via nasal cannulae and there is no accessory muscle use. She has mild pitting oedema and is centrally cyanosed. She has a capillary refill of two seconds and there are no murmurs.

Hb	160 g/l	Na +	138 mmol/l
Platelets	310 * 10 /l 9	K +	5.1 mmol/l
WBC	9.0 * 10 /l 9	Urea	7.8 mmol/l
Neuts	7.8 * 10 /l 9	Creatinine	110 µmol/l
Lymphs	1.0 * 10 /l 9	CRP	30 mg/l

ABG (on arrival) pH	7.25
pO2	7.91 kPa
pCO2	7.6 kPa
HCO3	31mmol/l

Chest x-ray	poor inspiration, no overt consolidation
ECG	sinus rhythm

What is your first step in management?

A. Salbutamol nebuliser
B. Intubation and ventilation
C. Intravenous antibiotics
D. Naloxone
E. Non-invasive ventilation

D. Naloxone

Opioids can precipitate respiratory failure in patients with chronic respiratory disease

This lady has entered type 2 respiratory failure. A lot of the options are suitable here but the key is identifying what the quickest solution may be. Ventilation is key but there are some other actions that should be tried first. Intravenous antibiotics are useful but she is haemodynamically stable and not overtly septic. Salbutamol nebulisers will help her air entry. However, there are several clues in the question. She is taking lots of opioids and other sedatives and has increased her uptake of oramorph because of pleuritic pain. She has pin point pupils and bradypnoea and a stat dose of naloxone may rapidly recover her respiratory drive and allow her to recover without needing the other options.

Question 33 of 133

A 22-year-old man presents to rheumatology clinic with chronic lower back pain and early morning stiffness. He has past medical history of asthma and is on regular steroid inhalers and suffers from multiple exacerbations annually. He is due to see a respiratory physician to help better manage his symptoms. Clinical examination shows demonstrate Schober's sign. Pelvic Xrays demonstrate sacroiliitis. A diagnosis of ankylosing spondylitis is made.

The patient is concerned about using NSAIDs to control his symptoms; what factor may suggest that NSAIDsensitive asthma?

A. Previous reaction to beta-blockers
B. High frequency of exacerbations
C. Presence of nasal polyps
D. Adult-onset asthma
E. Previous ITU admission

C. Presence of nasal polyps

The correct answer is nasal polyps. Asthmatics are typically taught to be aware that NSAID's can worsen asthma but not all patients are sensitive. The presence of nasal polyps is associated with a higher chance of reaction to NSAID's, whilst having had NSAID's and suffered no reaction within the last 12 months reduces the chance of further reactions. The other options do not predict a reaction ITU admissions and high frequency of admissions can suggest poorly managed or brittle asthma.

Question 34 of 133

A 48-year-old male presents to the respiratory clinic with his partner. They are concerned as he has lost 5 kgs in weight over the last three months. He also reports a productive cough which has been troubling him for a similar time period. He reports that he is currently producing approximately half a cup of phlegm each day. He denies fevers or night sweats and systems review is unremarkable.

You send the patient for a chest X-ray which is reported as showing multiple cavitating lesions. Sputum microscopy, sensitivities and culture (MC&S) are all negative and a HIV test is negative. You refer the patient for a bronchoscopy and bronco-alveolar lavage (BAL) to aid your diagnosis. Several weeks later you receive a call from the microbiology registrar on call to inform you that the patients sputum has cultured a non-tuberculous mycobacterium (Mycobacterium intracellulare).

You call the patient back into clinic and find that his symptoms have persisted since his last visit.

What is the most appropriate management plan for this individual?

A. Rifampicin, isoniazid, ethambutol, pyrazinamide
B. Repeat sputum cultures
C. Clarithromycin monotherapy
D. Rifampicin, clarithromycin and ethambutol

E. Rifampicin and isoniazid

D. Rifampicin, clarithromycin and ethambutol

This question tests your understanding of the diagnostic criteria for non-tuberculous mycobacteria (NTM) and appropriate treatment of common causative species. Diagnosis can be made by following the joint American Thoracic Society / Infectious Diseases Society of America (ATS/IDSA) guidelines which were published in 2007. Diagnosis requires a range of clinical, radiological and microbiological characteristics to be fulfilled as described below:

Clinical: Pulmonary symptoms (e.g. cough, sputum, or constitutional) and exclusion of alternative diagnosis

Radiological: Chest X-ray or HRCT demonstrating cavities, bronchiectasis and or nodules Microbiological: Two or more positive sputum samples for an NTM species taken on separate days (or one of more positive sample from bronco-alveolar lavage or tissue biopsy)

Once a diagnosis of NTM is reached, it is important to know the basic treatment for common pulmonary NTM infections. It is important to have a basic understanding of two commonly identified groups: Mycobacterium avium complex (MAC) and Mycobacterium kansasii.

- MAC: Rifampicin, clarithromycin and ethambutol. Treatment should continue until the patients sputum has remained negative for MAC for 12 months

- M.kansasii: Rifampicin, isoniazid and ethambutol. Treatment should continue until the patient has been sputum culture negative for 12 months.

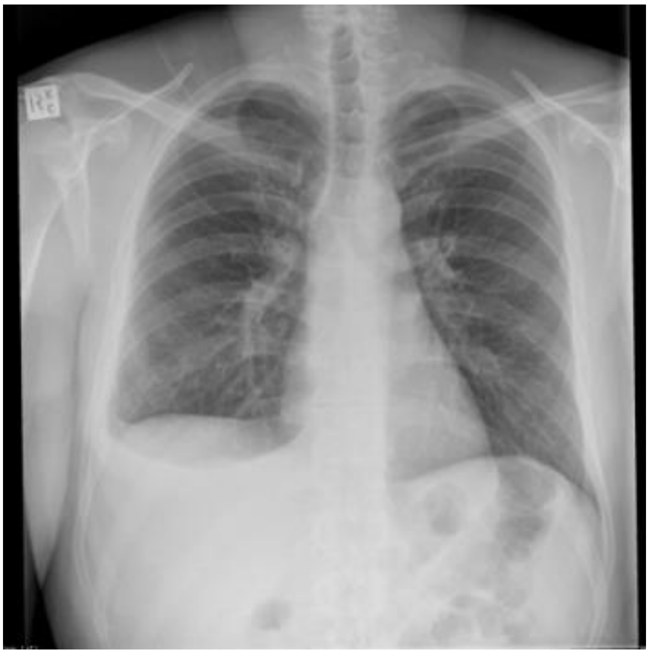
It is important that patients with NTM infection should never receive clarithromycin monotherapy due to concerns over development of resistance.

Question 35 of 133

You review the chest x-ray of a 35-year-old man who started treatment for a chest infection two weeks ago. He completed a one week course of antibiotics but still feels short-of-breath on exertion. He has a 15 pack-year history of smoking.

On examination his respiratory rate is 16/min, saturations 98% on room air and auscultation of his chest is unremarkable.

His chest x-ray is shown below:



What does the chest x-ray show?

A. Right lower lobe collapse
B. Lung tumour
C. Pleural effusion
D. Elevated right hemidiaphragm
E. Persistent consolidation

C. Pleural effusion

The chest x-ray shows a right basal opacity obscuring the right costodiaphragmatic recess with a meniscus, findings consistent with a pleural effusion. Lungs and pleural spaces are otherwise unremarkable. Cardiomediastinal contour within normal limits

Question 36 of 133

A 72-year-old gentleman presents for review in the medical clinic. He has had COPD for five years and is on regular bronchodilator treatment with a combination budesonide and formoterol fumarate inhaler and salbutamol inhaler as needed. He has found that he suffers from regular coughing bouts and finds his exercise tolerance has reduced.

His past medical history includes macular degeneration, osteoarthritis, mild memory impairment and diverticulosis. He has noticed that the distance he can walk has reduced from being able to go to the local shop, roughly one mile away, to having to stop half-way to catch his breath.

What investigation would be most useful to help determine the severity of his COPD?

A. FEV1/FVC
B. Chest X-ray
C. High resolution CT scan of the chest
D. Partial pressure of oxygen on arterial blood gas
E. FEV1% of predicted

E. FEV1% of predicted

The correct answer is FEV1% of predicted. All of the mentioned investigations, in addition to routine blood tests, can be useful in assessing COPD, but the primary investigation standard for COPD is FEV1% of predicted. This compares the patient's ability to exhale over one second with what someone of the same sex, height, weight and ethnicity. It is a marker of the extent of airway obstruction and therefore a marker of COPD severity.

FEV1/FVC is useful in diagnosis but not the severity of COPD.

Imaging can show radiological changes, but these are not as standardised as spirometry changes.

Hypoxia on arterial blood gases is more useful in establishing the need for home oxygen than COPD severity.

Question 37 of 133

A 68-year-old female attends the acute medical unit with dyspnoea. She states that the dyspnoea is present at rest and exacerbated by exertion. She has a past medical history of hypothyroidism, recurrent urinary tract infections, and anxiety. Her drug history includes nitrofurantoin, levothyroxine, paracetamol, bisoprolol and sertraline. On examination you note right basal crepitations and stony dull percussion at the right lung base.

A chest x-ray demonstrates a right sided pleural effusion. What drug is the most likely cause of the pleural effusion?

A. Levothyroxine
B. Bisoprolol
C. Sertraline
D. Paracetamol
E. Nitrofurantoin

E. Nitrofurantoin

Several drugs are known to cause pleural effusion including nitrofurantoin, methotrexate, and amiodarone

Several drugs are known to cause pleural effusion including nitrofurantoin, methotrexate, and amiodarone. None of the other options are known to cause pleural effusions.

Nitrofurantoin is sometimes used long term for prophylaxis of urinary tract infections. Respiratory complications of nitrofurantoin include acute pulmonary reactions (e.g. consolidation) and chronic reactions (e.g. fibrosis and effusions). Patients should have spirometry every 3-6 months while on nitrofurantoin.

Question 38 of 133

A 57-year-old is admitted with confusion, fever and decreased oral intake. HIs past medical history is significant for type 2 diabetes mellitus, hypertension, osteoarthritis and gout. Blood cultures are positive for *E. coli*. Despite antibiotic therapy, he slips into respiratory failure. He is intubated and placed on mechanical ventilation with an FiO of 70%, tidal volume of 370 ml and respiratory rate of 14/min.

His current arterial blood gases are:

pH	7.45
pO_2	7.86 kPa
pCO_2	3.99 kPa
HCO_3	21 mmol/l

Which of the following is the best next step in managing this patient?

A. Increase tidal volume
B. Increase respiratory rate
C. Increase the fraction of inspired oxygen
D. Add positive end-expiratory pressure
E. Decrease the fraction of inspired oxygen

D. Add positive end-expiratory pressure

Fever, confusion, decreased food intake and blood cultures positive for *E. coli* all point towards a diagnosis of sepsis, a common cause of acute respiratory distress syndrome (ARDS). ARDS is characterised by diffuse injury to the pulmonary capillary endothelium and alveolar epithelium that ultimately leads to alveolar collapse and impaired pulmonary gas exchange.

The patients blood gases reflect hypoxaemia and a slight respiratory alkalosis, meaning that despite high FiO settings and sufficient ventilation, his arterial oxygenation remains inadequate. Generally speaking, oxygenation may be improved by further increasing the FiO or by adding positive end-expiratory pressure (PEEP). High FiO is contraindicated due to the risk of pulmonary oxygen toxicity. Thus, the goal in managing mechanically ventilated patients should be to keep the FiO below 40% at all times. Adding PEEP is the next best step here. PEEP prevents alveolar collapse, directly counteracting the means by which ARDS causes hypoxaemia. It may also reopen some alveoli that have already collapsed.

Choice 1: This patients level of ventilation is adequate as confirmed by a slightly low pCO . Increasing the tidal volume would unnecessarily increase the minute ventilation and peak inspiratory pressure, thereby worsening the respiratory alkalosis and increasing the risk of barotrauma respectively.

Choice 2: This patients level of ventilation is adequate as confirmed by a slightly low pCO . Increasing the respiratory rate would worsen the respiratory alkalosis and would not improve oxygenation.

Choice 5: Decreasing the FiO at this point would further reduce the patients oxygenation. This patients FiO does need to be reduced soon in order to avoid pulmonary oxygen toxicity, but this should be accomplished by first increasing oxygenation by another means, such as by increasing PEEP.

Question 39 of 133

A 73-year-old woman presents to the respiratory clinic with progressive breathlessness over the last 18 months. Her past medical history includes angina, breast cancer 14 years ago (treated with mastectomy and radiotherapy) and rheumatoid arthritis (on sulfasalazine and methotrexate). She previously smoked 15 cigarettes per day for 20 years but stopped after her diagnosis of breast cancer.

On examination, the patient has evidence of clubbing with a regular pulse of 68 beats per minute. She is not cyanosed and has no evidence of CO2 retention. On examination of her lungs, there are fine end-inspiratory crackles most affecting the upper zones.

What is the most likely cause of this patient's lung disease?

A. Chronic obstructive pulmonary disease
B. Idiopathic pulmonary fibrosis
C. Methotrexate pneumonitis
D. Radiation pneumonitis
E. Rheumatoid arthritis'

D. Radiation pneumonitis

radiotherapy may result in radiation pneumonitis, typically causing upper zone lung fibrosis

This patient likely has interstitial lung disease (ILD) as evidenced by clubbing and fine endinspiratory crackles.

Of the causes listed, **radiation pneumonitis** is a cause of ILD which is most likely to affect the upper zones and the patient has a history of previous radiotherapy, making radiation pneumonitis the most likely cause.

Chronic obstructive pulmonary disease (COPD) does not fit with the clinical findings of clubbing and end-inspiratory crackles.

Idiopathic pulmonary fibrosis is a form of ILD which typically affects the lower zones more than the upper zones and is, therefore, less likely to be the cause.

Rheumatoid arthritis is a form of ILD which typically affects the lower zones more than the upper zones and is, therefore, less likely to be the cause.

Methotrexate pneumonitis typically presents with cough, breathlessness and fever. Methotrexate can also cause lung fibrosis but this would also typically affect the lower zones more than the upper zones so would be the less likely diagnosis in this instance.

Question 40 of 133

A 74-year-old ex-miner is reviewed in the Respiratory Clinic with a 4-month history of episodic dyspnoea and dry cough. The attacks last for several days at a time and are accompanied by fever, malaise, and chest tightness. He recovered from the most recent episode 2 weeks ago.

His past medical history is unremarkable and he states that he is generally well between episodes. He does report some long-standing shortness of breath on exertion, but this has been stable for the past 6 years and he estimates his exercise tolerance at 200 yards.

He worked as a miner until the early 1980s and then retrained as a plumber. He continued to work until his retirement in the early 2000s. He now enjoys housing pigeons in his back garden. He does not smoke.

On examination, his respiratory rate is 14/min and his oxygen saturations are 95% breathing room air. His fingers are clubbed and there are bibasal crackles on auscultation of the chest. His heart sounds are dual with nil added.

A high-resolution CT (HRCT) scan of the thorax is reported as showing subpleural bibasal reticular opacities with some evidence of honeycombing but no appreciable ground-glass opacification.

What is the most likely cause of his symptoms?

A. Asbestosis
B. Hypersensitivity pneumonitis
C. Silicosis
D. Coal worker's pneumoconiosis
E. Idiopathic pulmonary fibrosis

B. Hypersensitivity pneumonitis

the key to successfully answering this question is to appreciate that it is asking you to explain his symptoms rather than the examination and investigation findings. MRCP questions often include varying amounts of distracting information in order to simulate the complexity of clinical practice, although in this case there is enough information in the opening paragraph to make the diagnosis irrespective of the confounding information presented in the rest of the vignette.

The occurrence of episodic dyspnoea, cough, and flu-like symptoms in response to an environmental antigen is consistent with the acute presentation of hypersensitivity pneumonitis. Patients are well in between episodes and during the early phase of the disease examination and investigation can be normal.

The abnormalities on the HRCT are consistent with usual interstitial pneumonia (UIP); a pathological pattern of interstitial lung disease rather than a diagnosis in itself. UIP has numerous causes, including asbestosis and idiopathic pulmonary fibrosis (IPF). Both of these diagnoses would be in keeping with the clubbing and bibasal crepitations found on examination, but neither would cause the relapsing and remitting symptoms described.

The patient does describe some longstanding dyspnoea on exertion, which would be more in keeping with asbestosis or stable IPF. The absence of ground glass on the HRCT implies there is no active lung inflammation and also suggests stable disease.

Silicosis and coal worker's pneumoconiosis cause upper zone fibrosis. Both conditions typically cause chronic rather than episodic symptoms.

Question 41 of 133

A 58-year-old smoker was referred to the respiratory outpatient clinic by his GP with a history of progressively worsening shortness of breath. His symptoms started several months ago with shortness of breath on extreme exertion but he gradually developed shortness of breath with minimal activity. He also complained of increasing tiredness and a non-productive cough had lost approximately 3 pounds in 6 months. He denied the presence of sputum production, haemoptysis or chest pain, and also denied the presence of both orthopnoea and paroxysmal nocturnal dyspnoea. He had a past medical history comprising ankylosing spondylosis diagnosed 30 years ago as well as hypertension and hypercholesterolaemia. His drug history comprised naproxen 500mg BD, lansoprazole 30mg OD, felodipine M/R 2.5mg OD and atorvastatin 20mg OD. He stated that he had been trialled with a course of methotrexate a few years ago for his ankylosing spondylosis and was subsequently stopped.

Examination revealed the presence of a well male with a blood pressure of 146/86 mmHg, heart rate 74 bpm and respiratory rate of 18/min. His oxygen saturations were 93% on air. There was no BCG scar seen on examination of his arm. Examination of his respiratory system revealed the presence of bilateral fine upper zone crackles but nil else and no respiratory distress. Examination of his cardiovascular system revealed no abnormalities including a normal JVP and the absence of pedal oedema, and examination of his gastrointestinal system was likewise unremarkable.

Initial investigations revealed the following:

Hb	166 g/l
Platelets	341 * 10 /l9
WBC	6.3 * 10 /l9
ESR	34 mm/hr
CRP	26 mg/l

Chest x-ray: Bilateral apical fibrosis

Pulmonary function testing

- FEV1 2.4 l (predicted value 2.3)
- FVC 2.6 l (predicted value 4.8)
- FEV1/FVC ratio 92%
- TLCO transfer factor 86% of predicted value

What is the most likely diagnosis?

A. Pulmonary hypertension
B. Pulmonary tuberculosis
C. Pulmonary fibrosis secondary to methotrexate
D. Extrinsic allergic alveolitis (EAA)
E. Pulmonary fibrosis secondary to ankylosing spondylosis

E. Pulmonary fibrosis secondary to ankylosing spondylosis

Upper zones lung fibrosis: ankylosing spondylitis

This gentleman has presented with a gradual history of progressing worsening shortness of breath on exertion and a dry cough. His spirometry illustrates a restrictive picture and chest x-ray confirms bilateral apical fibrosis. Of the above options, the most likely option is being secondary to ankylosing spondylosis. Methotrexate causes a lower zone fibrosis and of the remaining options, there is little evidence of exposure to suggest a diagnosis of EAA. There is little evidence to suggest the presence of clinically symptomatic pulmonary hypertension as manifest by the presence of a normal JVP and absence of peripheral oedema

. Question 42 of 133

A 10-year-old patient is referred to the respiratory outpatient clinic with recurrent chest infections associated with purulent sputum. His family life is chaotic and his parents frequently did not bring him to routine hospital appointments.

On examination, there are bilateral coarse crackles heard on chest auscultation, which clear on coughing. He is of thin body habitus. The nails are normal.

A chest x-ray shows evidence of bronchiectasis. A sputum sample grows *Burkholderia cepacia* . Genetic testing reveals that he is homozygous for the delta F508 mutation.

Given the likely diagnosis, which of the following is an appropriate treatment option?

A. Low calorie, low fat diet
B. Lumacaftor/Ivacaftor
C. Lung transplant
D. Mepolizumab
E. Octreotide

B. Lumacaftor/Ivacaftor

Lumacaftor/Ivacaftor (Orkambi) can be used to treat cystic fibrosis patients who are homozygous for the delta F508 mutation

Lumacaftor/Ivacaftor is the correct answer. The patient has cystic fibrosis as evidenced by recurrent chest infections, bronchiectasis and homozygosity for the delta F508 mutation of the cystic fibrosis transmembrane conductance regulator (CFTR) gene. It is known as 'Orkambi'. It has been shown to improve pulmonary function, increase weight and decrease pulmonary exacerbations. This patient's chaotic family life explains the delayed diagnosis.

Lung transplant is incorrect. This patient is colonized with *Burkholderia cepacia* , which is a CF contraindication to lung transplant.

Mepolizumab is incorrect. This is an IL5 inhibitor that is used in the treatment of refractory asthma, rather than CF. Given it is an immunosuppressant medication, it would probably have deleterious effects in CF where infections are common.

Low calorie, low-fat diet is incorrect. Patients with CF are often of low body weight and should be encouraged to have a high fat, high-calorie diet.

Octreotide is incorrect. This has been postulated to be of benefit in yellow nail syndrome, which is a rare cause of bronchiectasis. However, the nails are normal in this case and there are no pleural effusions, making this diagnosis unlikely

Question 43 of 133

A 27-year-old non smoker Chinese lady presents to the emergency department with sudden onset left pleuritic chest pain and shortness of breath. She explains that she has suffered from gradual deterioration in her exercise tolerance over the last year.

Chest x-ray demonstrated a large pneumothorax that is aspirated successfully. Repeat chest x-ray shows resolution of the pneumothorax but reticulonodular changes in the bases bilaterally.

CT chest: cystic changes in the lung bases with a minimal left sided pleural effusion

. What is the most likely diagnosis?

A. Neurofibromatosis
B. Alveolar proteinosis
C. Langerhans cell histiocytosis
D. Lymphangioleiomyomatosis
E. Hermansky Pudlak Syndrome

D. Lymphangioleiomyomatosis

Lymphangioleiomyomatosis (LAM) affects premenopausal women causing proliferation of atypical smooth muscle cells in the lungs, lymphatics and uterus, most likely caused by oestrogens. There is associations with tuberous sclerosis and renal angiomyolipomas.

Presentation is with dyspnoea due to progressive interstitial lung disease, pneumothorax or chylous pleural effusion. Lung damage is similar to emphysema with pulmonary function tests showing reduced FVC, TLCO and KCO with increased TLC.

Medroxyprogesterone is an anti-oestrogen used to delay disease progression.

It is important to exclude metastatic sarcoma and atypical Langerhans cell histiocytosis. Langerhans cell histiocytosis affects smokers 20-40 years due to proliferation of Langerhans cells (antigen presenting cells containing Birkbeck bodies on electron microscopy). Presentation of LCH is with exertional dyspnoea and cough, spontaneous pneumothorax, clubbing and manifestation of granuloma infiltration in bones and the hypothalamus (diabetes insipidus is commonly seen).

Alveolar proteinosis most commonly affects males and can be secondary to insecticides, silica, HIV.

There is accumulation of lipoproteinaceous material in the alveolar spaces. Presentation involves dyspnoea, chest pain, haemoptysis and clubbing. Chest x-ray shows bat wings and diagnosis is confirmed with bronchoalveolar lavage (milky fluid, PAS positive material).

Hermansky Pudlak syndrome consists of oculocutaneous albinism, abnormal platelet function and interstitial lung disease.

Question 44 of 133

A 28-year-old man is referred by A&E to the acute medical team. He is a Swedish student studying for his PhD at the local university. He complains of a 6-week history of a non-productive cough associated with fevers. He denies haemoptysis or weight but admits that his exercise tolerance has reduced over the same period. He also reports pains in his knees, ankles and wrists and some painful red swellings on his legs. He admits to having had these before several years ago but they went away without any medical treatment and left no scars. He denies any bowel symptoms. On examination, he is afebrile and cardiovascularly stable. His respiratory rate is 18 at rest and his saturations are 96% on air. He has no palpable lymphadenopathy and his respiratory examination is normal, as is cardiovascular and abdominal examinations. His shins have bilateral tender red nodules on them.

His bloods results done by A&E are:

Hb	145 g/l
Platelets	365 * 10 /l9
WBC	9.1 * 10 /l9
Neuts	5.1 * 10 /l9
Na +	140 mmol/l
K +	4.2 mmol/l
Urea	5.4 mmol/l
Creatinine	82 µmol/l
Bilirubin	4 µmol/l
ALP	59 u/l
ALT	23 u/l
Albumin	43 g/l
CRP	5 mg/l
Adjusted calcium	2.65 mmol/l

His chest x-ray shows bilateral hilar lymphadenopathy with some small bilateral pulmonary infiltrates.

He goes on to have a CT chest which confirms the above findings. A bronchoscopy is performed

which is normal and transbronchial biopsies of the hilar lymph nodes show non-caseating granulomas.

What is the most appropriate treatment for this gentleman?

A. Prednisolone
B. NSAIDs
C. R-CHOP
D. Methotrexate
E. Nothing

A. Prednisolone

This gentleman has Lofgren's syndrome a presentation of Sarcoidosis characterised by Arthralgias, fevers, erythema nodosum and bilateral hilar lymphadenopathy. It is typically more common in Scandinavian patients and less common in Afro-Caribbean patients and typically has a much better prognosis. This gentleman has stage 2 sarcoidosis as indicated by the presence of BHL and pulmonary infiltrates on CXR. In this case, oral corticosteroids are the preferred treatment. For less symptomatic patients then supportive measures and NSAIDs for arthralgia are the preferred treatment options. For patients who are not responsive to steroids or steroid intolerant then immunosuppressant agents such as methotrexate can be tried. RCHOP is a chemotherapy regime used in lymphoma patients but the presence of non-caseating granulomas should suggest a diagnosis other than lymphoma

. Question 45 of 133

A 52-year-old woman presents to the respiratory clinic for review. She recently had a cough for which she received a week-long course of amoxicillin by her GP. As the cough had not settled within ten days, she returned to the GP who organised a chest X-ray. Afterwards, she was referred to the respiratory clinic due to the findings on it. This surprised her as by this point all of her symptoms had resolved. She has a past medical history of idiopathic intracranial hypertension, polycystic ovaries and type 2 diabetes mellitus. She takes only metformin and has no allergies. She has never smoked and has no occupational exposure of note. She drinks roughly one glass of wine per day. On examination she is obese but her chest is clear on auscultation. The chest X-ray demonstrates bilateral hilar lymphadenopathy.

Hb	127 g/l
Platelets	417 * 10 /l9
WBC	6.2 * 10 /l9
ESR	43 mm/hr
Na +	138 mmol/
K +	3.7 mmol/l
Urea	4.8 mmol/l
Creatinine	82 µmol/l
Corrected calcium	2.1 mmol/l

What is the most appropriate management plan?

A. Discharge without further investigation
B. Start ACE inhibitors
C. Monitoring of lung function tests
D. Start methotrexate
E. Start corticosteroids

C. Monitoring of lung function tests

he correct answer is to monitor lung function tests. This is a patient who has X-ray features consistent with sarcoidosis. As these are stage I X-ray features, there is no involvement of other

organs and the calcium is normal there is no need to start treatment with corticosteroids. Corticosteroids would be the mainstay of treatment if indicated. Sarcoidosis patients should have lung function assessed and a decline in lung function should warrant treatment.

Question 46 of 133

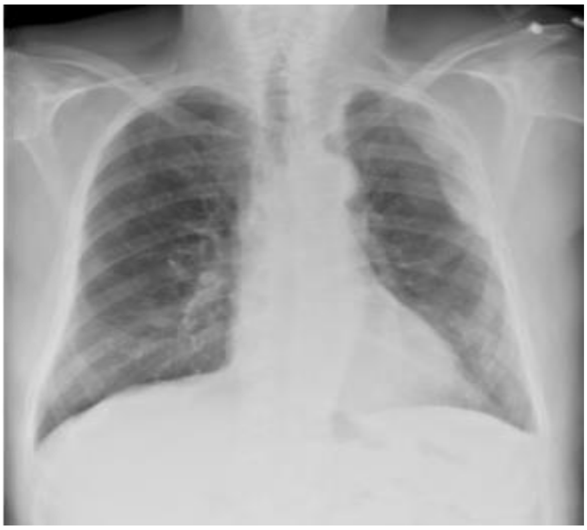
A 68-year-old builder presents to the emergency department with a 2-week history of worsening left-sided chest pain. The pain is sharp in nature and worsens on inspiration and movement, making him almost restricted to sitting down all day. He feels occasionally short of breath. His past medical history consists of peripheral vascular disease and chronic obstructive pulmonary disease (COPD).

On examination, he is afebrile. His heart rate is 88bpm with a blood pressure of 116/77mmHg. His respiratory rate is 21 breaths per minute with saturations of 91% on room air. On auscultation, there is mild wheeze bilaterally but no coarse crackles. Heart sounds are normal and abdominal examination is unremarkable. There is mild reproducible left-sided chest wall tenderness.

A number of investigations are requested. Laboratory results:

Hb	101 g/L	Male: (135-180)
MCV	71 fL	(80-100 fL)
Platelets	299 * 10 /L	(150 - 400)
WBC	8.1 * 10 /L	(4.0 - 11.0)
D-Dimer	501 ng/mL	(< 400)
Na	136 mmol/L	(135 - 145)
K	4.1 mmol/L	(3.5 - 5.0)
Urea	6.2 mmol/L	(2.0 - 7.0)
Creatinine	98 µmol/L	(55 - 120)
CRP	67 mg/L	(< 5)

Chest x-ray



What is the most likely diagnosis?

A. Asbestosis
B. Loculated pleural effusion
C. Mesothelioma
D. Pulmonary embolism
E. Small cell carcinoma

C. Mesothelioma

This patient has mesothelioma, a type of malignancy of the mesothelium, the tissue surrounding visceral organs including the lungs and chest wall, heart and abdominal cavity. Most commonly the pleura are affected. The biggest risk factor is asbestos exposure. With this patient having worked as a builder, it is very possible that he has come into contact with asbestos during his career. There is a visible opacification on the left lateral chest wall of the chest x-ray, in keeping with a pleural mass. Unlike other types of pulmonary malignancy that commonly affect the bronchi and lung parenchyma, mesothelioma affects the pleura and would therefore be located peripherally. One of the most common and important symptoms to recognise is severe chest wall pain as the tumour invades into the intercostal nerves.

Asbestosis is characterised by the development of lung fibrosis secondary to asbestos exposure. Important distinguishing features between asbestosis and mesothelioma are the presence of chest pain, progressive shortness of breath and constitutional symptoms such as fever, weight loss and night sweats. This patient is experiencing chest pain, fatigue and shortness of breath. Furthermore, his full blood count demonstrates microcytic anaemia which suggests the possibility of an underlying malignancy.

A pleural effusion is a collection of fluid within the pleural space. An exudative pleural effusion is more likely to become loculated. Appearances of loculated pleural effusions can sometimes appear similar to the chest x-ray shown in the question. However, the absence of a raised white cell count, in combination with a significant history of chest wall pain suggests an alternative diagnosis.

A pulmonary embolism is an important differential diagnosis given the raised D-dimer and pleuritic-sounding chest pain. Occasionally, a pulmonary embolism may have chest x-ray findings such as a wedge infarction. However, a raised D-dimer can also indicate an underlying malignancy. Furthermore, a peripherally-located lesion is unlikely to represent a pulmonary embolism.

Small cell carcinoma is a pulmonary malignancy commonly associated with tobacco use. As this patient has a history of COPD, it is likely that he has a significant smoking history. Asbestos exposure and tobacco use are synergistic to one another and increase the risk of malignancy. A small cell carcinoma tends to be located more centrally on chest x-ray films compared to mesothelioma as seen here

Question 47 of 133

A 62-year-old man was admitted to the hospital with a productive cough. Chest x-ray showed

right basal pneumonia. He responded well to intravenous antibiotics and was discharged after

being successfully weaned off oxygen.

What, if any, follow-up should this man receive?

A. CT thorax in 6 weeks to exclude malignancy
B. No follow-up required
C. Repeat chest x-ray in 6 weeks
D. Repeat chest x-ray in 6 weeks and routine respiratory clinic follow up
E. Respiratory clinic follow up routinely

D. Repeat chest x-ray in 6 weeks

All cases of pneumonia should have a repeat chest X-ray at 6 weeks after clinical resolution

Repeat chest x-ray in 6 weeks: the British Thoracic Society recommends repeating a chest x-ray for patients with confirmed pneumonia 6-12 weeks after clinical resolution. This is to exclude malignancy which can be obscured by active consolidation.

CT thorax in 6 weeks to exclude malignancy: a CT wound not be indicated in the absence of any other signs or symptoms. There would be no justification for the high level of radiation.

No follow up required: a chest x-ray is required to exclude underlying malignancy.

Repeat chest x-ray in 6 weeks and routine respiratory clinic follow up: there is currently no recommendation to arrange routine clinic follow up for pneumonia patients.

Respiratory clinic follow up routinely: there is currently no recommendation to arrange routine clinic follow-up for pneumonia patients.

Question 48 of 133

A 72-year-old gentleman presents with a five month history of dry cough and worsening shortness of breath on exertion. He undergoes extensive investigations and is diagnosed with idiopathic pulmonary fibrosis.

Bloods performed as part of the diagnostic work up show:

Hb	141 g/l
Platelets	255 * 10 /l9
WBC	5.2 * 10 /l9
ESR	32 mm/h
Na +	139 mmol/l
K +	3.6 mmol/l
Urea	4.7 mmol/l
Creatinine	92 µmol/l
CRP	15 mg/L

His lung function tests show a forced vital capacity (FVC) of 65% of predicted.

On examination the patient has obvious finger clubbing and fine end-inspiratory crepitations on auscultation of the chest.

Of note in his past medical history he smoked for ten years between the ages of 20 and 30.

He is understandably very distressed by the diagnosis and asks about potential therapies. He is enrolled on a pulmonary rehabilitation programme but is keen to try a pharmacological therapy.

Which drug should be considered in this patient?

A. Azathioprine
B. Prednisolone
C. Bosentan
D. Pirfenidone
E. Sildenafil

D. Pirfenidone

Idiopathic pulmonary fibrosis (IPF) is a chronic progressive disease with a dismal prognosis. In the UK the median survival is three years from diagnosis although a sub -group (approximately 20%) survive over five years. The disease rarely presents below the age of 45 and presenting symptoms include; breathlessness on exertion, persistent cough and fatigue.

Management of IPF has changed dramatically over the last decade. Previous small trials had shown small benefits of using a combination of corticosteroids with azathioprine and this combination remained the only pharmacological therapy for many years. Later larger trials demonstrated that this treatment regime confers no benefit and may actually cause harm thus the National Institute for Clinical Excellence (NICE) states that immunosuppressant therapies such as azathioprine, prednisolone and mycophenolate mofetil should not be used in IPF.

The only pharmacological therapy that has shown any benefit in IPF is pirfenidone, an immunosuppressant that has been shown to have anti-inflammatory and antifibrotic effects. The mechanism of action of pirfenidone is not completely understood but is thought that it suppresses fibroblast proliferation thus reducing the production of fibrosis-associated proteins and cytokines.

The only other therapeutic option for patients is lung transplantation and NICE recommends that any patients wishing to explore this option should be referred providing they have no absolute contraindications. Unfortunately, the speed of progression of the disease and availability of suitable organs means that this treatment modality is only used in a very small minority of patients.

It is clear from these guidelines that pirfenidone is the correct answer. Bosentan is an endothelin receptor antagonist used in the treatment of primary pulmonary hypertension. Sildenafil is a phosphodiesterase inhibitor which has vasodilating effects and is best known for its use in the treatment of erectile dysfunction. It was initially devised as a therapy for primary pulmonary hypertension and it has beneficial effects in this disease.

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A 78-year-old man with Chronic Obstructive Pulmonary Disease is reviewed in Respiratory clinic. He is complaining of decreased exercise tolerance with shortness of breath at rest. He suffers from exacerbations infrequently and does not have a productive cough. Oxygen saturations on air are 90%. His FEV 1 is 27% predicted.

He has a past medical history of osteoarthritis, ischaemic heart disease and chronic kidney disease.

His medications include Paracetamol, Ramipril, Aspirin, inhaled long acting beta agonist with inhaled corticosteroid and inhaled long acting muscarinc agonist. He has previously undergone pulmonary rehabilitation. He does not smoke

Investigations:

Hb	19.8 g/dL
WCC	6.0 *10^9/l
Platelets	180 *10^9/l
MCV	88fL

Echocardiogram: No abnormality seen.

CT Chest: Widespread mild bullous disease throughout upper and lower lung fields. Arterial blood gas on air:

Current	Four weeks previously	
pH	7.40	7.37
pCO2	5.1 kPa	4.7 kPa
pO2	7.5 kPa	7.4 kPa
HCO3	25 mmol/l	24 mmol/l

What is the most appropriate treatment?

A. Lung volume reduction surgery
B. Mucolytic therapy
C. Long term antibiotic prophylaxis
D. Oral corticosteroids
E. Long term oxygen therapy

E. Long term oxygen therapy

Lung volume reduction surgery. This is a possibility for patients with optimal medical therapy with symptomatic dyspnoea and severe COPD. It has improved mortality in patients with upper lobe predominant emphysema. This patient also has significant co-morbidities that make this option incorrect.

Mucolytic therapy. This patient is not suffering from a chronic productive cough, therefore, this is less likely to benefit the patient. It would be advisible to trial in patients for 3-4 weeks with a productive cough e.g. Carbocisteine.

Long term antibiotic prophylaxis. Minimal exacerbations in this patient and poor overall data make this option incorrect.

Oral Corticosteroids. This patient is not suffering from an exacerbation. Maintenance corticosteroids may be required in advanced disease if unable to wean off after an exacerbation.

Long term Oxygen therapy. This patient is suffering from severe COPD (FEV1 <30%). He has polycythaemia, it is likely to be secondary to COPD as he has normal platelets and MCV. LTOT is indicated in COPD if the pO2 is <7.3 on two separate ABGs 3 weeks apart. It can be offered to patients with a pO2 7.3-8 kPa with secondary polycythaemia, peripheral oedema, nocturnal hypoxaemia or pulmonary hypertension. He will need to be monitored on oxygen for hypercapnia and acidosis.

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A 54-year-old woman is reviewed in gastroenterology clinic following the insertion of a transjugular intrahepatic portosystemic shunt (TIPS). She is known to have alcohol related hepatic cirrhosis with portal hypertension and was found to have large varices on endoscopy and therefore underwent the insertion of a TIPS two months ago. However, she has started to feel breathless in the last month and found that her exercise tolerance has been reduced. She has otherwise felt well and denies any pain, confusion or nausea. On examination, there is evidence of chronic liver disease including palmar erythema, Dupuytren's contracture, spider naevi and leukonychia. Her chest is clear on auscultation. There is a loud P2 sound but otherwise, there are no abnormalities on cardiac auscultation.

What is the most likely cause of breathlessness?

A. Hepatorenal syndrome
B. Pulmonary hypertension
C. Anaemia
D. Pleural effusion
E. COPD

B. Pulmonary hypertension

The correct answer is pulmonary hypertension. This is a known complication of TIPS as the procedure causes an increased cardiac preload by diverting blood past the cirrhotic liver, thereby significantly reducing peripheral resistance. This can lead to pulmonary hypertension which can explain the patient's breathlessness. The absence of respiratory findings makes pleural effusion and COPD unlikely. With effusions, dullness and reduced air entry would be expected and COPD could occur with cirrhosis in anti-trypsin deficiency, but the absence of wheeze makes this very unlikely. Anaemia can occur, but is unlikely to be related to TIPS.

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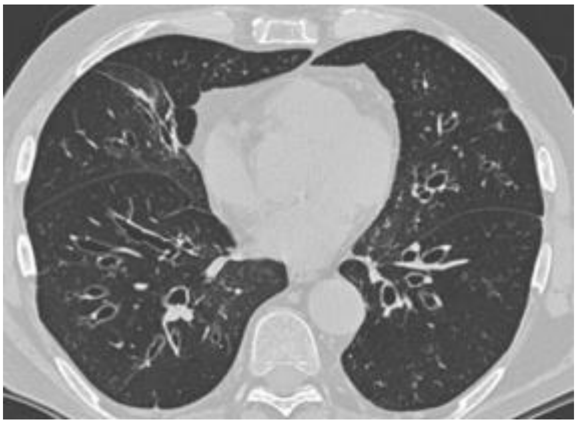
A 45-year-old man is referred to the respiratory department due to increasing shortness-of-breath and frequent chest infections. He is a smoker (20 cigarettes/day). When he was a child he used a salbutamol inhaler but stopped this around the age of 6-7 years. He also had whooping cough as a child. Pulmonary function tests performed by his GP show the following:

FEV1	3.6 L (predicted 3.8 L)
FVC	4.3 L (predicted 4.5 L)
FEV1/FVC	84% (normal > 75%)

On auscultation of his chest scattered crepitations are noted. His oxygen saturations are 96% on room air.

A CT chest is requested:



What is the most likely diagnosis?

A. Lung cancer
B. Previous Bordetella pertussis infection
C. Emphysema
D. Kartagener's syndrome
E. Cystic fibrosis

B. Previous Bordetella pertussis infection

The CT scan shows signet-rings and tram-tracks consistent with bronchiectasis. This, in combination with normal pulmonary function tests makes a diagnosis of emphysema less likely.

We can also exclude cystic fibrosis (CF). Some patients with CF now survive to 45 years but it would be very unusual to reach this age without significant respiratory problems in the past. An exam question would never include such a bizarre history for CF.

There is also no evidence of situs inversus excluding Kartagener's syndrome.

The bronchiectasis associated with lung cancer is confined to the area distal to the tumour. It would therefore not explain the bilateral symptoms.

The most likely diagnosis is therefore *Bordetella pertussis* infection, a known cause of bronchiectasis.

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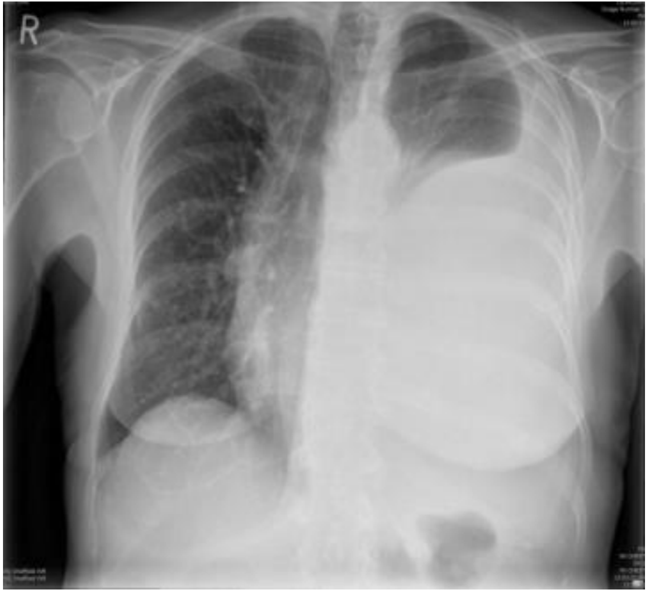
A 52-year-old woman is referred to the Acute Medical Unit after developing progressive dyspnoea over the past 4 weeks. She has no significant past medical history of note.

On examination her respiratory rate is 18/min, pulse 84/min, blood pressure 102/64 mmHg and oxygen saturations 96% on room air. Temperature is 37.1ºC. A chest x-ray is performed:



What is the most appropriate initial management?

A. Insert a 26F chest drain after performing a thoracostomy
B. Arrange a high resolution CT chest
C. Prescribe intravenous antibiotics
D. Insert a 18F chest drain using the Seldinger technique
E. Pleural aspiration

E. Pleural aspiration

The first step in the management of this patient is pleural aspiration, preferably under ultrasound

guidance. This will help guide further management and determine the aetiology of the effusion.

There is no need for immediate chest drain insertion as she is stable.

The x-ray shows a large left-sided pleural effusion. The meniscus is well demonstrated on the superolateral aspect indicating fluid in the pleural space.

Question 53 of 133

A 60-year-old man is diagnosed with idiopathic pulmonary fibrosis after presenting with an 18- month history of progressive dyspnoea. His latest FVC is 60% of predicted.

What is the average life expectancy for such a patient from the time of diagnosis?

A. 3-6 months
B. 6-12 months
C. 12-18 months
D. 3-4 years
E. 8-12 years

D. 3-4 years

The correct answer in this case is **3-4 years**. Idiopathic pulmonary fibrosis (IPF) is a progressive, irreversible lung disease characterized by fibrosis of the lung parenchyma, leading to worsening dyspnoea and reduced lung function. The prognosis for IPF varies widely, but on average, the median survival from the time of diagnosis is around 3-4 years. In this patient with an FVC of 60% predicted, his life expectancy would also fall within this range.

The option **3-6 months** is incorrect because it represents a significantly shorter life expectancy than what is typically seen in patients with IPF. While some patients may have rapidly progressive disease or other complicating factors that could lead to a shorter survival time, the average life expectancy for someone with IPF is longer than this.

Similarly, the option **6-12 months** is also incorrect as it underestimates the average life expectancy for a patient with IPF. Although some patients may have a more aggressive form of the disease or other comorbidities that contribute to a worse prognosis, most patients can expect to live longer than one year following their diagnosis.

The option **12-18 months** still underestimates the typical life expectancy for someone diagnosed with idiopathic pulmonary fibrosis. While there are cases where individuals may experience rapid progression of their disease or have additional health issues that impact their prognosis, on average, individuals diagnosed with IPF tend to live longer than 18 months.

Lastly, the option **8-12 years** overestimates the average life expectancy for someone with idiopathic pulmonary fibrosis. Although there are cases where individuals may survive beyond this timeframe due to various factors such as early diagnosis and effective management strategies, these cases are less common and do not represent the average life expectancy for a patient with IPF

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A 63-year-old is reviewed following admission with an infective exacerbation of COPD. He originally presented with shortness of breath, tight-chestedness and feeling generally unwell. He was initially managed with medical treatment including IV piperacillin with tazobactam, IV hydrocortisone, IV fluids, nebulised salbutamol and nebulised ipratropium bromide but his hypercapnia and acidosis failed to improved. He was then initiated on non-invasive ventilation (NIV) starting with IPAP of 10 cm H2O and EPAP of 4 cm H2O. He has managed to have the IPAP increased to 14 cm H2O but he is struggling to tolerate this as he is feeling increasingly anxious. He has only had NIV for three hours so far.

Arterial blood gas results:

On admission	Current	
pH	7.11	7.23
pCO_2	14.7	12.1
pO_2	8.4	10.7

What is the most appropriate method to help him settle him?

A. Give him haloperidol
B. Give him diazepam
C. Increase IPAP
D. Increase EPAP alone
E. Stop NIV

A. Give him haloperidol

The correct answer is to give him haloperidol. Many patients struggle to tolerate an NIV mask as it is tight-fitting and blows air uncomfortably at the patient and it can be difficult to accustom too. When first introducing the mask to the patient explain what is happening to the patient as they may be confused, especially if hypercapnic, and gently place the mask on the patient's face. It can help to place the mask in the patient's hand and guide it to their face to facilitate cooperation. Show the patient how to take on and off the mask if they are able to do so as this may ease the process. In the acute phase it is important that there are frequent reviews to ensure the mask is being used. Many can begin to tolerate the mask over time.

Increasing the IPAP would make it more uncomfortable and would likely make him refuse the treatment entirely, even though it is likely to be needed to help relieve the hypercapnia as it creates greater ventilatory support.. Decreasing the IPAP or stopping NIV would be more comfortable but would be inappropriate as treatment is then likely to fail with greater hypoxia and acidosis. Diazepam is contraindicated in this situation as the added drowsiness can be dangerous, but haloperidol or morphine may help settle him sufficiently to tolerate the mask. Increasing EPAP without increasing IPAP would reduce the amount of ventilatory support and would be inappropriate

. Question 55 of 133

A 68-year-old is seen in a cardiology clinic. He has a part history of long-standing atrial fibrillation, hypertension and ischaemic heart disease. He describes progressive shortness of breath over the past year and a dry cough. His medication history includes aspirin, amiodarone, ramipril, doxazosin and gliclazide.

On examination, he is comfortable on lying flat but is noticeably dyspnoeic when mobilising. He has minimal ankle oedema, his heart rate is 78 beats per minute and he has fine bibasal crepitations on auscultation of his chest. His oxygen saturations on air are 92%.

An echocardiogram reveals a left ventricular ejection fraction of 45%, a mildly hypertrophied left ventricle and well established right ventricular hypertrophy, pulmonary artery pressures are raised at 32 mmHg (normal: < 25 mmHg). A chest x-ray shows non-specific changes on both bases.

What is the most appropriate next investigation?

A. Exercise stress test
B. Cardiac MRI
C. Overnight oximetry
D. Repeat echo
E. HRCT chest

A. HRCT chest

The raised pulmonary artery pressures and right ventricular hypertrophy points to a respiratory pathology. Despite the history of atrial fibrillation a cardiac cause is less likely to be causing his dyspnoea in view of the fact that he is comfortable when lying flat (i.e. no orthopnoea), he has minimal ankle oedema and that his left ventricular function is only mildly impaired. This patient should be investigated for interstitial lung disease in view of his history of amiodarone use. In light of this an exercise stress test, a cardiac MRI and a repeat echo are unlikely to be of any benefit and there is no indication to perform overnight oximetry. An HRCT is warranted to help aid establish a diagnosis.

Question 56 of 133

A 65-year-old lady with known severe emphysema attends the emergency department with breathlessness and is diagnosed with a non-infective exacerbation of chronic obstructive pulmonary disease. It is her 6th exacerbation this year. She tells you that she has been referred by her respiratory team for lung volume reduction surgery and is awaiting an appointment

Which of the following criteria would make the patient an unsuitable candidate for lung volume reduction surgery?

A. Predominantly upper lobe emphysema and high exercise capacity.
B. Non-upper lobe emphysema and high exercise capacity.
C. Predominantly upper lobe emphysema and low exercise capacity.
D. Upper lobe emphysema and carbon monoxide diffusing capacity 40%
E. Non-upper lobe emphysema and low exercise capacity

B. Non-upper lobe emphysema and high exercise capacity

The National Emphysema Treatment Trial was a prospective, randomised, multi-centre trial which compared the results of lung volume reduction surgery to medical therapy which showed that there were 3 groups of patients that tend to benefit:

Group 1: Upper lobe emphysema and low exercise capacity. These patients show improvement in both functional outcomes and survival after lung volume reduction surgery compared to medical therapy.

Group 2: Patients with predominantly upper lobe emphysema and high exercise capacity. These patients have improved functional outcomes but no difference in survival compared to medical therapy.

Group 3: Patients with non-upper lobe emphysema and low exercise capacity. These patients have improved survival after surgery but there is no difference in survival compared to medical therapy.

The trial also identified patients with emphysema that are unlikely to do well from lung volume reduction surgery and have a high risk of death. This includes:

1. Patients with non-upper lobe emphysema and high exercise capacity.

2. Patients with extremely poor pulmonary function (forced expiratory volume in 1 second (FEV1) 20% or less than predicted) and either homogenous distribution of emphysema on

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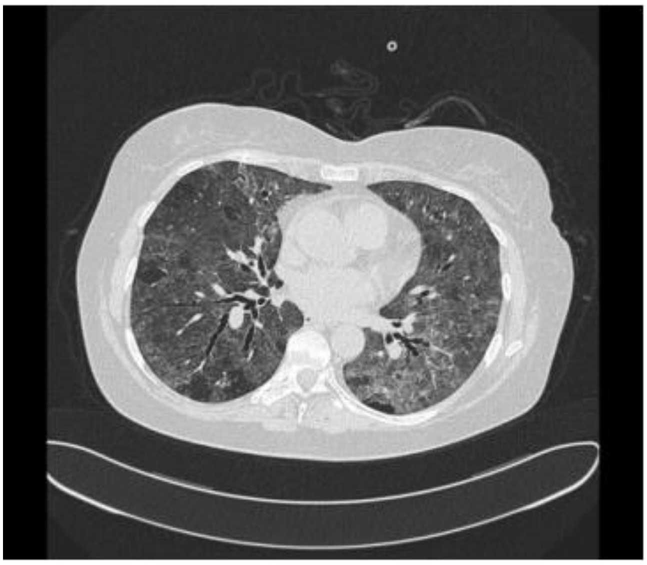
A 60-year-old woman is investigated for recurrent episodes of dyspnoea. She describes becoming progressively more short-of-breath over the past two years with frequent exacerbations associated with fever, lethargy and coughing bouts. Her symptoms often settle down for a time but she does feel her breathing is getting worse over time.

She smokes 20 cigarettes/day, drinks 10 units of alcohol per week and works in a poultry farm. Her past medical history includes hypothyroidism and depression.

On examination her oxygen saturations on air are 94%. A CT scan is requested:



What is the most likely diagnosis?

A. Bronchiectasis
B. Hypersensitivity pneumonitis
C. Sarcoidosis
D. Histiocytosis
E. Histoplasmosis

B. Hypersensitivity pneumonitis

This lady most like had acute hypersensitivity pneumonitis to start with which has now progressed to the sub-acute/chronic form. The diagnosis is suggested by her occupation, intermittent symptoms and CT findings which show multuple centrilobular ground glass nodules.

Question 58 of 133

A 54-year-old male is reviewed in the respiratory clinic. He originally presented with progressive shortness of breath and a dry cough, while having no other significant history or exposure to chemicals or environmental triggers. He is a non-smoker and does not take any medications.

Examination is consistent with bibasal inspiratory crackles and there is evident clubbing. There is no signs of cardiac failure. He currently saturates at 94% on air and has a known FVC of 70% measured at his last appointment.

His pulmonary function test reveals a restrictive pattern and a high resolution CT is undertaken concluding:

'Bilateral lung volume loss with >5% honeycombing extensively at the bases and subpleural areas with evidence of peripheral traction bronchiectasis, extensively at the lung bases'.

Prior blood investigations have been negative with regards to any connective tissue disease.

He has already been referred for pulmonary rehabilitation but is wondering whether any medication could potentially alter the course of his disease.

Which medication can be used to reduce functional decline in this group of patients?

A. Azathioprine
B. Sildenafil
C. Prednisolone
D. Bosentan
E. Pirfenidone

E. Pirfenidone

Pirfenidone and nintedanib can be used as drug management options in idiopathic pulmonary fibrosis as they may decrease the functional decline, prolong survival and potentially reduce acute exacerbations

The patient in question suffers from idiopathic pulmonary fibrosis (usually interstitial pneumonia type) as evident by the restrictive pattern, lack of history suggestive of other causes of fibrosis alongside the pathognomonic CT features. Therefore the question really focuses on the pharmacological management of this disease bearing in mind its irreversible nature.

Pirfenidone and nintedanib are two antifibrotic agents currently licensed for use in the UK provided the forced vital capacity (FVC) between 50% and 80% predicted given the evidence in literature currently.

Sildenafil is a phosphodiesterase-5-inhibitor that has been trialled in such patients in order to examine the potential of improved oxygen delivery; a study in 2010 concluded that there was no potential benefit in the use of sildenafil in such patients, except where pulmonary hypertension exists. Prednisolone and steroid use in

patients with idiopathic pulmonary fibrosis as a disease modifier has not been proven to be beneficial.

Bosentan is a dual endothelin receptor antagonist that is currently licensed for the treatment of pulmonary arterial hypertension. As per the BUILD-3 trial, the use of bosentan does not delay idiopathic pulmonary fibrosis progression or death.

Azathioprine is a purine analogue which is used in a variety of autoimmune conditions. It has been used extensively in the past in caring for patients with idiopathic pulmonary fibrosis as part of the triple therapy of prednisolone/azathioprine/N-acetylcysteine; however due to PANTHER-IPF study and the increased hospitalisation of patients with side-effects of immunosuppression the study has been put on hold and this treatment is encouraged to be avoided.

Question 59 of 133

A 24-year-old male is reviewed on the trauma ward 5 days after a road traffic collision. He suffered multiple injuries most significant of which included an extradural haematoma, right-sided pneumothorax, and a femoral fracture. The patient was initially intubated, had a chest drain inserted, and underwent intramedullary nailing. The extradural haematoma was managed conservatively.

The patient was successfully extubated 3 days ago and has had his oxygen requirements progressively weaned. Today on the ward round the patient reports his breathing is improving but he has still some dyspnoea on exertion and is requiring 2L via nasal prongs. The chest drain is swinging and has on-off bubbling. There is good air entry heard throughout the chest.

What is the most appropriate course of action?

A. Apply suction to the drain
B. Remove the chest drain
C. Reposition the drain and increase the patient oxygen delivery
D. Seek a thoracic surgical opinion
E. Leave the drain in situ and review in 24 hours

D. Seek a thoracic surgical opinion

When draining a pneumothorax, if after 3-5 days there is persistent air leak (e.g. bubbling chest drain) or failure of the lung to re-expand, a thoracic surgical opinion should be sought

Repositing of the drain is incorrect as there is no evidence that the drain has moved or become displaced. Oxygen delivery can assist resolve a pneumothorax however as it has been over 5 days further management may be required.

The drain could be left in situ and review in 24 hours however it has already been 5 days since it was originally inserted and there is evidence of a persistent leak and therefore surgical opinion should be sought.

Question 60 of 133

An 82-year-old gentleman with known chronic obstructive airways disease (COPD) is about to be discharged from hospital after being admitted with an infective exacerbation. He is now off antibiotics and has just completed a seven-day course of prednisolone. He remains hypoxic on room air S 0 91%.

Bloods show:

Hb	134 g/l
Platelets	350 * 10 /l9
WBC	10.2 * 10 /l9

Arterial blood gas analysis shows:

P 0 a 2	7.8 kPa
P CO a 2	6.5 kPa
HCO 3 -	30 mmol/L

The patient's wife asked about having oxygen at home.

What other investigations are needed to assess whether this patient would benefit from long-term oxygen therapy?

A. Oxygen saturations before and after exercise
B. Pulmonary function tests
C. Computed tomography (CT) of the chest
D. Chest x-ray
E. Echocardiogram

E. Echocardiogram

Long-term oxygen therapy (LTOT) refers to the prolonged domiciliary use of oxygen for patients with medical conditions that result in a state of chronic hypoxaemia (P 0 at or below 7.3kPa). The patient is assessed so that the flow rate of oxygen they receive results in a waking oxygen tension above 8kPa. LTOT is usually prescribed indefinitely and to be of benefit has to be used for a minimum of 15 hours a day including at night time. The indications for LTOT are well established.

The British Thoracic Society (BTS) advises that LTOT may be of benefit in patients with the following conditions:

- Chronic obstructive pulmonary disease
- Severe chronic asthma
- Interstitial lung disease
- Cystic fibrosis
- Bronchiectasis
- Pulmonary vascular disease
- Primary pulmonary hypertension
- Pulmonary malignancy
- Chronic heart failure

Patients with any of the above conditions can be assessed to see if they would benefit from LTOT.

BTS guidelines currently state that patients should have a P 0 consistently at or below 7.3kPa on air at a time when they are clinically stable (absence of exacerbation in previous five weeks).

Another group of patients who can be prescribed LTOT are those with chronic hypoxaemia and a Pao2 between 7.3kPa and 8kPa and the presence of either secondary polycythaemia or clinical and/or echocardiographic evidence of pulmonary hypertension.

In the above case, the patient has chronic hypoxaemia. Ideally, this patient would have his arterial blood gases analysed five weeks following this exacerbation, but assuming that his results are similar and his haemoglobin remains the same then he would require evidence of pulmonary hypertension to benefit from LTOT thus an echocardiogram is the best answer to this question.

Question 61 of 133

A 62-year-old man with metastatic small cell lung cancer is admitted with a shortness of breath, cough and purulent sputum.

His chest x-ray shows a right lower lobe pneumonia with partial collapse of the right lung. He has a bulky primary tumour at the right hilum and extensive hilar lymphadenopathy.

He is treated with IV antibiotics, fluids and oxygen. His symptoms improve over the next 5 days and his breathing is comfortable on 2 litres of oxygen via nasal cannulae. His pain is controlled on 20mg long-acting morphine sulphate twice daily. The decision is made to discharge him to a hospice for end-of-life care. However, due to the proximity of his lung tumour to major vessels, he is at risk of a major terminal bleed.

Which medication should the hospice be advised to give in the event of a major bleed?

A. Haloperidol
B. Midazolam
C. Morphine
D. Tranexamic acid
E. Vitamin K

B. Midazolam

This patient may experience a terminal catastrophic bleed due to erosion of his lung tumour through a major vessel. Patients at risk of such an event should be issued with a 'crisis pack' containing midazolam and green or blue towels which can help soak up blood whilst masking some of the colour. Midazolam can be administered intravenously if access available or otherwise intramuscularly. The aim is to minimise the anxiety and distress.

Haloperidol is a useful anxiolytic and antiemetic in end-of-life care but is not as effective in this situation. Morphine is used for pain and breathlessness in the terminal setting.

Tranexamic acid may be given is cases of slow gastrointestinal bleeding due to malignancy. Vitamin K is used to lower the INR in patients with a bleed on warfarin or with liver failure.

Question 62 of 133

A 38-year-old woman is referred to the respiratory clinic due to a pulmonary nodule detected on a CT scan. She was admitted to her local hospital two months ago with aspiration pneumonia and treated with intravenous antibiotics. A chest X-ray demonstrated left-sided consolidation. She was also managed for alcohol withdrawal. She required three days of in hospital before being discharged with oral antibiotics and community alcohol liaison services to follow-up. Her GP repeated her chest X-ray six weeks following discharge and was concerned that an area of consolidation remained on the repeat chest X-ray. The GP, therefore, arranged for a CT chest which demonstrated a pulmonary nodule 5mm in diameter on the right side. There was no evidence of consolidation.

How should this nodule be investigated or monitored?

A. Urgent CT-PET
B. Urgent image-guided biopsy
C. CT chest in three months
D. CT chest in one year
E. No further investigations needed

D. CT chest in one year

The correct answer is CT chest in one year. As the patient has a pulmonary nodule 5mm in size there will need to be further monitoring. As it is 5-6mm in size this means that monitoring should take form in terms of a CT scan in one year

Question 63 of 133

An 83-year-old man attends hospital with an infective exacerbation of COPD. He had become increasingly short of breath with an episode of rigors and a dry cough. He is started on salbutamol and ipratropium nebulisers and steroids but fails to improve clinically. After careful discussion, he is offered non invasive ventilation and he tolerates this well. He is slow to improve and 36 hours later, he starts to become more breathless.

On examination, he has crackles and wheeze on the right hemithorax. The left hemithorax has reduced air entry throughout and is resonant to percuss. There are no added sounds., no JVP or leg oedema that can be seen but there is accessory muscle use. He is tired and feels 'absolutely awful'. The non-invasive ventilation is currently set at IPAP 26 cm H2O and EPAP 6 cm H2O. The mask is well adjusted and there is no significant mask leak. A repeat chest x-ray is pending.

Chest X-ray (on admission): hyperinflation, no consolidation seen
ABG (admission)

pH	7.32
PaO2	7.6kPA
PaCO2	7.4 kPA
HCO3	30mmol/L

ABG (1hr after starting NIV)

pH	7.36
PaO2	9.2kPA
PaCO2	6.2 kPA
HCO3	28mmol/L

ABG (now)

pH	7.34
PaO2	6.5kPA
PaCO2	7.0 kPA
HCO3	28mmol/L

What is the cause of this deterioration?

A. Poor compliance with NIV Poor compliance with NIV
B. Patient-ventilator dyssynchrony
C. Pneumothorax
D. Aspiration
E. Hospital acquired pneumonia

C. Pneumothorax

Pneumothorax can occur following high pressure non-invasive ventilation

The patient has deteriorated from a respiratory viewpoint on NIV despite earlier improvement. First, we must consider if he is breathing against the machine (dyssynchrony) but we are told he is tolerating it well. There is no evidence to suggest that he has not complied with the NIV. He may have aspirated but there is no history of vomiting or copious secretions. He examination signs are consistent with a pneumothorax on the left side rather than a hospital acquired pneumonia. Pneumothorax is a recognised complication of NIV particularly when higher inflation pressures are used. Please not that initial pressures started are 12cm H20 IPAP and 4 cm H2O EPAP and this gentleman was on significantly higher pressures.

Question 64 of 133

You are called by the general surgical registrar for a medical consult for their patient.

A 19-year-old Caucasian male presented with a two-day history of epigastric pain associated with nausea and vomiting. He was found to have an elevated serum amylase level and thus admitted under the surgical team for management of acute pancreatitis. The surgical team have asked for a medical consult due to the patient reporting worsening productive cough.

Upon your review, the patient reports that this is his third presentation with acute pancreatitis. He does not drink alcohol and an ultrasound of the abdomen did not reveal any gallstones. The cough has been present for several months. Initially, this was a dry cough but more recently has been become productive of green sputum with no haemoptysis. He denies fever, chills, chest pain and dyspnoea. Three weeks prior to this presentation he had received a course of oral antibiotics from his General Practitioner after presenting with nasal congestion and mild facial pain. He states having had multiple courses of antibiotics in the past for similar presentations.

There is no further past medical history or family history of note.

He takes no regular medications and has been receiving simple analgesia and intravenous crystalloid fluids whilst under the care of the surgical team. He is a lifelong non-smoker and works full time in administration.

On examination his height was 178cm with a weight of 58kg, heart rate 90/minute, blood pressure 100/60 mmHg, temperature 37.2ºC, respiratory rate 16/min and oxygen saturation 94% on room air. Respiratory examination revealed early clubbing, no cervical lymphadenopathy, a central trachea, normal chest expansion, mild coarse inspiratory crepitations that improved somewhat after coughing at both posterior bases. Abdominal examination revealed mild epigastric tenderness but no signs of peritonism. Remaining clinical examination was unremarkable.

Hb	130 g/l	Na	136 mmol/l
Platelets	400 * 10 /l	K	4 mmol/l
WBC	12 * 10 /l	Urea	6 mmol/l
Neuts	10 * 10 /l	Creatinine	65 µmol/l
Lymphs	1 * 10 /l	CRP	30 mg/l
Eosin	1 * 10 /l		

Chest x-ray was normal.

Sputum microscopy and culture results revealed *Pseudomonas aeruginosa*

Which of the following would be the most appropriate management for this patient?

A. Intravenous ceftazidime and nebulised tobramycin for 14 days
B. Intravenous benzylpenicillin and oral doxycycline for 7 days
C. Oral ciprofloxacin for 7 days
D. Nebulised saline
E. Observe and repeat sputum microscopy and culture in the community once fullyrecovered from the acute pancreatitis

A. Intravenous ceftazidime and nebulised tobramycin for 14 days

Attempts at *Pseudomonas aeruginosa* eradication are important in patients with cystic fibrosis

This patient who presents with recurrent sinusitis, recurrent pancreatitis, clubbing and signs of bronchiectasis on examination likely has undiagnosed cystic fibrosis. *Pseudomonas aeruginosa* eradication is important in such patients and should be treated aggressively.

Whilst traditionally cystic fibrosis (CF) is considered a paediatric disease, it is becoming increasingly important for adult physicians to be aware of the clinical manifestations and complications of CF for two main reasons:

- 10% of patients are still identified when they are more than 18 years of age
- With advances in diagnosis and treatment, the median age of survival for patients with CF is around 40 years, meaning you are likely to care of adult patients with CF during your clinical practice

Patients born before widespread neonatal screening became available are the cohort that may present with CF in adulthood. They also tend to have a more mild phenotype therefore not attracting medical attention earlier in life and may present with other CF complications, such as pancreatitis, sinusitis, intestinal obstruction or male infertility.

Chronic infection with *Pseudomonas aeruginosa* in patients with CF is associated with more rapid loss of lung function, frequent pulmonary exacerbations and a shorter median survival. It is also very difficult to eradicate *Pseudomonas aeruginosa* once chronic infection has been established. The key is to try to eradicate the organism once it is a first isolated, which requires acute aggressive treatment. In this case, given the diagnosis of CF is new, it should be presumed that this is a new sputum isolate of *Pseudomonas aeruginosa* .

There is no consensus on which anti-microbial strategy is best for *Pseudomonas aeruginosa* eradication. Options include:

- Intravenous anti-pseudomonal antibiotic plus inhaled aminoglycoside for 14 days
- A prolonged course of oral Ciprofloxacin (e.g. 6 weeks)

Option 1 is the correct answer.

Option 2 is a regime used more commonly for moderate community acquired pneumonia and would not be effective at eradicating *Pseudomonas aeruginosa*

Option 3 would have anti-pseudomonal cover but the duration is too short

Option 4 may help with sputum clearance but would not eradicate the *Pseudomonas aeruginosa*

Option 5 would increase the likelihood of chronic *Pseudomonas aeruginosa* infection developing and adversely affect the patient's prognosis

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A 23-year-old gentleman presents with severe shortness of breath and tight-chestedness. On examination, he is very wheezy, his respiratory rate is high but he is speaking in full sentences. He is a known to have asthma and was out in a storm when he suddenly noticed shortness of breath, wheezing and felt his chest becoming tight. His asthma is normally well controlled only occasionally requiring the use of a salbutamol inhaler. He is thought to have thunderstorm asthma.

What is thought to be the aetiological cause?

A. exacerbation
B. Electrical charge causing irritation
C. Sudden spore and pollen release due to pressure changes
D. Increase in winds causing exacerbation
E. Anxiety triggering asthma attack

C. Sudden spore and pollen release due to pressure changes

The correct answer is sudden spore and pollen release due to pressure changes. Thunderstorm asthma is a rare event in which the pressure changes in a thunderstorm causes the release of pollen and spores which trigger asthma exacerbation, which can occur in people not previously known to have asthma. The wet conditions at the beginning of a thunderstorm can cause pollen grains to rupture leading to increased amounts of allergen as well. Cold air and anxiety may also provoke asthma attacks, but these causes are not specifically associated with thunderstorms.

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A 45-year-old man presents to the emergency department with a 48-hour history of shortness of breath, wheezing, and chest tightness. He has a past medical history of asthma.

Upon arrival his observations are as follows; respiratory rate 30/min, oxygen saturations 88% on room air, heart rate 110/min. Auscultation revealed decreased air entry with wheeze throughout both lung fields.

Oxygen was administered via a non-rebreather mask. Nebulised salbutamol and ipratropium bromide were administered back-to-back. He also received intravenous corticosteroids and magnesium sulphate.

Following initial therapy, his oxygen saturations are 98% on 15L via a non-rebreather, his respiratory rate is 34/min, his heart rate is 108/min, and his blood pressure is 112/73mmHg. An arterial blood gas is performed.

pH	7.40	(7.35 - 7.45)
pO2	15.7kPa	(>8)
pCO2	5.0 kPa	(4.5 - 6.0)
HCO3-	22.7mEq/L	(22 - 26)
Base excess	-1.2mEq/L	(-2 to +2)

What is the most appropriate ongoing management plan for ongoing respiratory support?

A. Bi-level positive airway pressure (BiPAP)
B. Continue current management
C. Continuous positive airway pressure (CPAP)
D. Referral to intensive care to intubation and ventilation
E. Wean oxygen

D. Referral to intensive care to intubation and ventilation

Patients with features of acute life-threatening asthma who fail to respond to treatment, should be referred to intensive care for potential intubation and ventilation

Despite being tachypnoeic, this patient has a normal pCO2 which is a sign of life-threatening asthma. This is despite therapy with bronchodilators, corticosteroids, and magnesium. Therefore, this patient needs **referral to intensive care for intubation and ventilation**.

BiPAP is not generally used in the treatment of asthma as there is significant potential for air trapping so ventilation should be carried out in intensive care. Therefore this option is incorrect.

To **continue current management** would not be an appropriate response in this instance. This patient has failed to respond to therapy and is showing signs of life-threatening asthma. Therefore, this patient needs a referral to intensive care.

CPAP, similar to BiPAP, is not routinely used in the management of asthma. CPAP does not help with ventilation and therefore CO2 clearance. It also poses a significant risk of air trapping in asthmatics. Therefore, referral to intensive care would be more appropriate.

Although this patient has adequate saturations, it would be inappropriate to **wean oxygen** as this does not address the normal pCO2 which is a life-threatening sign.

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A 76-year-old woman presents to clinic with shortness of breath. This has been progressing over the last 18 months and is associated with a non-productive cough. She is still able to complete her usual day-to-day tasks but struggles with more exertional activities such as gardening.

She finished chemotherapy for non-Hodgkin's lymphoma two years ago. She has no other relevant medical history.

Her current medications include allopurinol, bisoprolol, aspirin, simvastatin, paracetamol and codeine.

On examination, she has finger clubbing and diffuse fine crackles on chest auscultation.

Given the likely diagnosis, which of the following spirometry results would you expect?

A. FEV1 - 1.4L (71%), FVC - 2.5L (102%), FEV1/FVC ratio - 56%, DLCO - 90% of predicted
B. FEV1 - 1.3L (66%), FVC - 1.6L (65%), FEV1/FVC ratio - 81%, DLCO - 75% of predicted
C. FEV1 - 1.9L (97%), FVC - 2.4L (98%), FEV1/FVC ratio - 79%, DLCO - 110% of predicted
D. FEV1 - 0.9L (45%), FVC - 2.1L (86%), FEV1/FVC ratio - 42%, DLCO - 90% of predicted
E. FEV1 - 0.9L (45%), FVC - 1.1L (45%), FEV1/FVC ratio - 81%, DLCO - 120% of predicted

B. FEV1 - 1.3L (66%), FVC - 1.6L (65%), FEV1/FVC ratio - 81%, DLCO - 75% of predicted

The patient likely has a restrictive lung defect, caused by pulmonary fibrosis. This may be due to a chemotherapy agent, such as bleomycin from her chemotherapy.

Obstructive spirometry:

- FEV1 reduced (<80% predicted normal)
- FVC is usually reduced but to a lesser extent than FEV1
- FEV1/FVC ratio reduced (<0.7)

Restrictive spirometry:

- FEV 1 reduced (<80% predicted normal)
- FVC reduced (<80% predicted normal)
- FEV 1 /FVC ratio normal (>0.7)

DLCO (diffusing capacity of the lung for carbon monoxide) measures the ability of the lungs to transfer gas from inhaled air to the red blood cells in pulmonary capillaries

Decreased DLCO

Restrictive lung disease

- Idiopathic pulmonary fibrosis
- Occupational lung disease
- Hypersensitivity pneumonitis
- Miliary tuberculosis
- Pneumonectomy

Obstructive lung disease

- Cystic fibrosis
- Emphysema

Other causes

- Chronic pulmonary embolism
- Congestive heart failure
- Primary pulmonary hypertension

Increased DLCO

Causes

- Pulmonary haemorrhage
- Polycythaemia
- Left to right cardiac shunting

Answer 1 and 4 both show obstructive patterns. Answer 3 is a normal spirometry pattern. Answers 2 and 5 both show a restrictive pattern. However, answer 5 has a raised transfer factor. In druginduced restrictive lung disease this would be low.

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A 32-year-old man is admitted to the acute medical unit with acute shortness of breath. Blood cultures are positive for Staphylococcus aureus. He has a pan systolic murmur heard loudest over the left sternal edge.

A chest x-ray confirms a cavitating mass in the right lung with associated pleural effusion. You perform a pleural tap which is as follows:

pH	7.15
Protein	42 g/l
Appearance	Serous

What test can be performed to assess if the effusion is an empyema?

A. Video-assisted Thoracoscopic Surgery (VATS)
B. Thoracic ultrasound
C. Chest x-ray
D. Centrifugation of the pleural aspirate
E. Plasma C-reactive protein (CRP)

D. Centrifugation of the pleural aspirate

If the pleural fluid is turbid or milky it should be centrifuged. If the supernatant (liquid which lies above the sediment) is clear, the turbid fluid was due to cell debris and empyema is likely

The history and clinical features are suggestive of right sided endocarditis with septic emboli to the lungs. The pleural fluid is an exudate, and the pH < 7.20 indicates that this is a complicated parapneumonic effusion which requires drainage.

Aspiration of frank pus is diagnostic of an empyema and no other investigations are required to establish the diagnosis, with the exception of pleural fluid microbiology to guide antibiotic therapy. If the aspirate does not reveal frank pus, further analysis is required to assess whether it is a complicated parapneumonic effusion. This involves measurement of the pleural fluid pH, total protein concentration, LDH level, glucose concentration, and white cell differential.

If an effusion is not frankly purulent, but empyema is suspected, then the sample should undergo centrifugation and if the supernatant becomes clear, empyema is the most probable diagnosis; otherwise, the differential diagnosis is between lipid pleural effusions and

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A 70-year-old lady with a background of type 2 diabetes mellitus and alcohol abuse presents with a fever and productive cough. She reports no weight loss or haemoptysis. She was born in England and has never travelled outside of the United Kingdom. She has never smoked cigarettes. On examination, she has crackles in her right upper lobe but is otherwise well and stable. A chest radiograph reveals consolidation which is cavitating in her right upper lobe.

What is the most likely diagnosis?

A. Lung cancer
B. Tuberculosis
C. Pneumococcal pneumonia

D. Klebsiella pneumonia
E. Aspergillosis

D. Klebsiella pneumonia

Klebsiella most commonly causes a cavitating pneumonia in the upper lobes, mainly in diabetics and alcoholics

Klebsiella pneumonia is particularly associated with diabetic and alcoholic patients. It leads to a cavitating upper lobe pneumonia. Differentials for this lady would include lung cancer and tuberculosis but these would usually be associated with weight loss and travel to eastern Europe or Asia respectively. the optimal treatment for Pneumocystis jirovecii which can cause severe bilateral pneumonia. However, it is most commonly seen as an opportunistic infection in patients with impaired cellmediated immunity, classically patients with HIV infection. In this case, there is no suggestion of significant immunocompromise and a typical pathogen has been isolated. It is therefore more appropriate to target therapy against this organism.

Change to IV ertapenem is incorrect. Ertapenem is a carbapenem (the same class as meropenem) with extremely broad-spectrum antimicrobial activity. However, the main difference between ertapenem and meropenem is that the former lacks activity against Pseudomonas species. It would therefore not be effective in this case

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A 65-year-old man presents to his GP after his wife complains he snores very loudly and occasionally stops breathing at night. He also falls asleep easily during the day while watching television or reading. He had bilateral tonsillectomies and adenoidectomy as a child. On examination, his height is 1.80m and his weight is 100kg. He has lost 15kg of weight in the last six months but his symptoms have persisted. His GP refers him for a sleep study which is suggestive of moderate sleep apnoea.

What initial management option should be recommended?

A. Further weight loss and repeat sleep study thereafter
B. Lifestyle advice including sleep hygiene
C. Mandibular advancement splint
D. Referral for CPAP
E. Referral to ENT for surgery

D. Referral for CPAP

Following weight loss, CPAP is the first-line treatment for moderate/severe obstructive sleep apnoea

Patients with symptoms suggestive of obstructive sleep apnoea (OSA) that affect their quality of life should be offered continuous positive airway pressure (CPAP) if they have moderate or severe OSA, in addition to lifestyle advice.

Weight loss as part of lifestyle advice may be appropriate as first-line treatment for patients with mild sleep apnoea. However, this patient has already tried to lose weight without any benefit. Furthermore, they have moderate sleep apnoea and should be offered CPAP alongside lifestyle advice. Patients who feel that their OSA has resolved following lifestyle modification can have a trial of stopping treatment for two weeks and then an evaluation of their symptoms or a repeat sleep study.

Lifestyle advice regarding sleep hygiene, reducing alcohol and smoking and weight loss is important but NICE recommends that it should be provided alongside CPAP in patients with moderate to severe OSAA mandibular advancement splint is not first-line in treating sleep apnoea but can be offered to those who do not tolerate CPAP and have good dental health. This patient should thus be offered CPAP first.

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A 57-year-old male presents with a three-month history of unintentional weight loss (13kg over 3 months) and a chronic non-productive cough. He has just returned from a months holiday in India. He denies haemoptysis or chest pain. He is a lifelong smoker. He has no past medical history except a period of generalised limb weakness two years ago when he was referred to outpatient neurology clinic and diagnosed with Lambert-Eaton myasthenic syndrome following investigations. His blood tests are unremarkable. However, his chest x-ray demonstrates a rounded opacity in his right midzone, about 4 cm from his right main bronchus.

What is the most likely diagnosis?

A. Tuberculosis
B. Aspergilloma
C. Small cell lung carcinoma
D. Non-small cell carcinoma
E. Pneumocystis jirovecii infection

C. Small cell lung carcinoma

The patient was treated four years ago for Lambert-Eaton syndrome (LEMS), a neurological disorder characterised by limb weakness that improves with exercise, affecting proximal muscles more than distal muscle. It is caused by antibodies directed against voltage-gated calcium channels and has a strong association with malignancy. Up to half of all LEMS patients has an underlying malignancy, the most common of which is small cell lung carcinoma. Neurological symptoms often precede malignancy diagnosis by around 5 years

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A 32-year-old taxi driver attends the emergency department with pleuritic right-sided chest pain. He is a current smoker of 15 cigarettes/day. He reports feeling unwell for 10 days with a cough and fever. The chest pain started gradually over the last 2 days becoming slowly more severe. A chest x-ray shows right sided consolidation and an effusion. He is commenced on penicillin antibiotics. Pleural aspirate under ultrasound guidance shows a simple effusion and the following results:

Appearance	Serous
pH	7.30
Protein	46 g/l
LDH	400 IU/l
Glucose	3.7mmol/l
Gram stain	No organisms or malignant cells seen

Culture	No growth

What is the next best step?

A. Intercostal drain
B. Continue antibiotics and observe
C. CT thorax with contrast
D. Repeat pleural aspirate in 24 hours
E. Further investigation with bronchoscopy

B. Continue antibiotics and observe

This patient has evidence of pneumonia. The unilateral effusion is confirmed as an exudate and can be assumed to be a parapneumonic effusion. There is no indication for chest drain currently (purulent fluid, pH<7.2, organisms on Gram stain or culture) so he can continue antibiotics and be observed. If he fails to improve he would need repeat pleural sampling. If there was no clear infective cause a CT thorax with contrast would be performed to look for underlying malignancy. Bronchoscopy is only performed when there is suspicion of bronchial obstruction.

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A 45-year-old lady presented to clinic with a four-month history of worsening cough with bloodstained sputum and exertional dyspnoea. She reported having lost over 8 kg in weight during this time, with a reduced appetite. She started smoking as a teenager and currently has 15 cigarettes daily. She has been diagnosed with small cell lung cancer. Her staging CT reveals a classification of T1a N2 M0. She attends for a follow-up appointment to see you in the oncology clinic

Which of the following treatment options should be considered first line?

A. Pembrolizumab
B. Cisplatin
C. Consider surgery
D. Thoracic radiotherapy
E. Palliation of symptoms

B. Cisplatin

Stage	Treatment
Early stage (T1-2a,N0,M0)	Surgery
Early stage (T1-2a,N0,M0)-Limited disease (T1-4,N0-3,M0)	4-6 cycles cisplatin based chemotherapy carboplatin if poor renal function/poor performance status +/- radiotherapy
Extensive disease (T1-4, N0-3, M1a/b)	6 cycles platinum based combination chemotherapy +thoracic radiotherapy if good response

Pembrolizumab is an example of an immunosuppressant therapy currently being researched for use in non-small cell lung cancer.

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A 28-year-old presents to Accident and Emergency with symptoms of increasing shortness of breath, wheeze and a cough.

This is her first hospital attendance with asthma which is usually well controlled. She has a past medical history of perennial rhinitis.

She has no known drug allergies and takes beclomethasone (200 micrograms inhaled twice a day) and salbutamol which she rarely uses. She has never smoked

On examination her respiratory rate is 24, her pulse is 98 bpm and has widespread wheeze on auscultation of her chest. Her oxygen saturations on air are 93%. She is quiet, anxious and seems disoriented.

Blood tests reveal a raised white cell count of 11 x 10 /L, her chest x-ray is normal.

She is started on nebulised salbutamol and ipratropium bromide and given an oral dose of prednisolone.

An arterial blood gas is taken 5 minutes after being started on 40% oxygen:

pH	7.36
PaO2	16.0 kPa
PaCO2	5.98 kPa
Bicarbonate	19 mmol/l

What would be the most appropriate next step?

A. Secure a cannula and give 200mg of hydrocortisone IV
B. Start empirical antibiotics
C. Magnesium infusion
D. Arrange a review by an anaesthetists/intensivist
E. Repeat an arterial blood gas after 10 minutes

D. Arrange a review by an anaesthetists/intensivist

The PaCO2 level which is at the upper limit of normal is very worrying and categorises this patient as having life-threatening asthma according to British Thoracic Society (BTS) guidelines. While a magnesium infusion may be of benefit, the description that she is anxious and possibly disoriented are concerning features and may indicate that shes tiring. In light of this getting a timely review by an anaesthetist/intensivist is of the utmost importance and should be the next immediate step. Giving hydrocortisone intravenously is unlikely to be of any benefit and considering antibiotics is not a priority in this situation where time is of the essence. Hence waiting another 10 minutes to do another arterial blood gas with to preceding action may potentially be fatal.

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A 57-year-old woman is admitted with an infective exacerbation of bronchiectasis. Multiple sputum cultures in the community have grown *Pseudomonas aeruginosa* and her GP had started oral ciprofloxacin. However, despite 4 days of this, she has developed worsening breathlessness, hypoxia, and fever and is now requiring oxygen.

Widespread coarse crepitations are heard and her chest x-ray demonstrates bilateral infiltrates.

What is the most appropriate course of action?

A. Add IV clarithromycin
B. Add IV vancomycin

C. Add high-dose trimethoprim/sulphamethoxazole and prednisolone
D. Change to IV ertapenem
E. Change to IV piperacillin-tazobactam

E. Change to IV piperacillin-tazobactam

Appropriate treatment of *Pseudomonas* positive bronchiectasis failing to respond to ciprofloxacin includes IV tazocin, ceftazidime, aztreonam or meropenem

The correct answer is **change to IV piperacillin-tazobactam**. *Pseudomonas aeruginosa* is a recognised cause of complex respiratory tract infections in patients with bronchiectasis or cystic fibrosis. Where oral treatment with ciprofloxacin fails, options include IV piperacillin-tazobactam, ceftazidime, or meropenem.

Add IV clarithromycin is incorrect. This is active against common pathogens causing communityacquired pneumonia as well as some atypical organisms. However, in this case, an organism has been isolated and it would be more appropriate to convert to IV therapy active against this. The combination of clarithromycin with quinolones also increases the risk of QT interval prolongation.

Add IV vancomycin is incorrect. This has purely gram-positive cover and is not active against *Pseudomonas aeruginosa*.

Add high-dose trimethoprim/sulphamethoxazole and prednisolone is incorrect. This would bethe optimal treatment for Pneumocystis jirovecii which can cause severe bilateral pneumonia.

However, it is most commonly seen as an opportunistic infection in patients with impaired cellmediated immunity, classically patients with HIV infection. In this case, there is no suggestion of significant immunocompromise and a typical pathogen has been isolated. It is therefore more appropriate to target therapy against this organism.

Change to IV ertapenem is incorrect. Ertapenem is a carbapenem (the same class as meropenem) with extremely broad-spectrum antimicrobial activity. However, the main difference between ertapenem and meropenem is that the former lacks activity against Pseudomonas species. It would therefore not be effective in this case.

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A 45-year-old woman presents to the emergency department with abdominal pain. It is severe and progressed to its maximum intensity over six hours. She has not noticed any associated vomiting or diarrhoea. She last opened her bowels yesterday. She has a history of alcoholic liver disease and has had previous admissions for decompensated liver failure. She is not jaundiced or confused currently. She has generalised abdominal tenderness, worse towards the epigastrium. A chest Xray demonstrates a right-sided pleural effusion. It is aspirated and found to have a protein count of 37g/l.

What is the most likely cause of the effusion?

A. Meig's syndrome
B. Hepatic failure
C. Pancreatitis
D. Nephrotic syndrome
E. Metastatic cancer

C. Pancreatitis

The correct answer is pancreatitis. This is a patient with epigastric abdominal pain and an exudative pleural effusion on a background of heavy alcohol intake. Exudative effusions occur due to increased leakiness of pleural capillaries. This can be due to infection, inflammation or malignancy. Of the available options, this includes pancreatitis and metastatic cancer. Transudates are caused by increased venous pressure, occurring in cardiac failure, renal failure, hepatic failure, as well as in Meig's syndrome, hypothyroidism and fluid overload. Given the epigastric pain and alcohol history, pancreatitis is a more likely cause than cancer and should be confirmed with serum and fluid amylase

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A 62-year-old gentleman presents to clinic with increasing shortness of breath on exertion. His symptoms have progressed over three months and he has now become concerned as he is struggling to walk through the park without stopping to catch his breath. He has also noticed a new, raised lesion on his chin. He has a few unexplained temperatures within the last week as well and has noticed mild joint pain. He assumed that the latter was arthritis but has not suffered from this before. He has a past medical history of hypertension, glaucoma and has been hospitalised with pneumonia within the last year.

On examination, he has a 1cm wide, raised lesion on his chin which is purple in colour with some telangiectasia. He does not have a fever currently and his observations are all within normal limits. There is no finger clubbing. There is bilateral cervical lymphadenopathy and auscultation demonstrates bibasal fine inspiratory crackles. These do not shift with coughing. A chest X-ray is requested.

What is it most likely to show?

A. Cavitating lesion
B. Pleural effusion
C. Bilateral consolidation
D. Bilateral hilar lymphadenopathy
E. Apical fibrosis

D. Bilateral hilar lymphadenopathy

The correct answer is bilateral hilar lymphadenopathy. This is a patient who has presented with typical symptoms and signs of sarcoidosis: progressive exertional dyspnoea, arthralgia, unexplained pyrexia and fine inspiratory crepitations that do not shift on coughing. The absence of a cough, the fact that the crepitations do not shift, and the absence of clubbing all make bronchiectasis a much less likely diagnosis. The skin lesion described is likely to be lupus pernio.

The most common X-ray finding in sarcoidosis is bilateral hilar lymphadenopathy, but pleural effusions can also occur. However, pleural effusion is not supported by the examination findings.

Again, the fact that the crepitations are basal makes apical fibrosis a less likely finding to see on imaging, and hilar lymphadenopathy is a far more common radiological sign.

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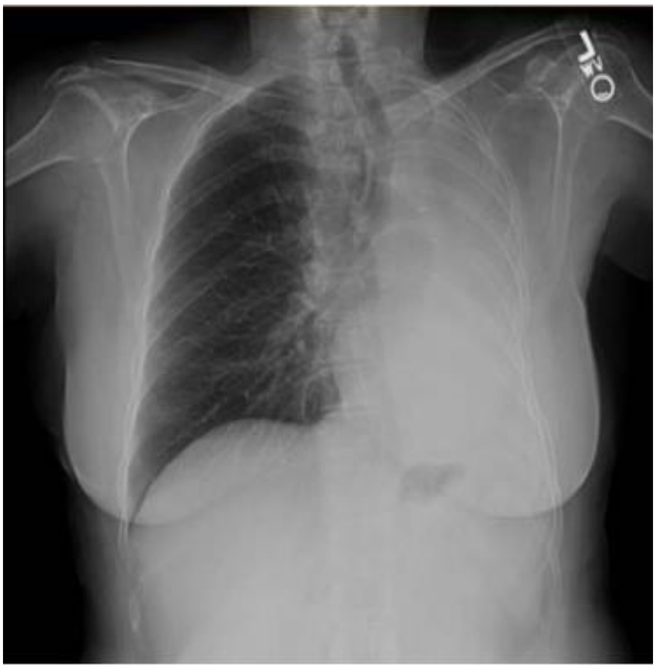
A 65-year-old woman is admitted to the Emergency Department after collapsing in a department store. A chest x-ray is done shortly after arriving in the department:



What is the most likely explanation for the abnormality on the left side?

A. Rotated film
B. Massive pleural effusion
C. Pneumonectomy
D. Mesothelioma
E. Pneumonia

C. Pneumonectomy

The key to making the correct diagnosis is to look at the trachea. In the film it is clearly pulled to the left side. This makes a diagnosis of pleural effusion, pneumonia or mesothelioma unlikely. A rotated film would not give such dramatic findings

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A 56-year-old woman with severe asthma attends with two days of shortness of breath and a tight feeling in her chest. She has a history of multiple exacerbations and was intubated three months ago for a severe exacerbation. She usually takes montelukast, beclometasone and salbutamol.

On arrival, she is cyanosed and saturating at 92% in air. There is accessory muscle use and she is not able to vocalise beyond groans. Her respiratory rate is 22/min and there is quiet wheeze heard bilaterally in the chest. She has failed to respond to nebulisers, steroids and magnesium sulfate. She is planned for intubation and initiation of aminophylline.

Hb	156 g/l	Na^+	145 mmol/l
Platelets	561 * 10 /l 9	K^+	3.2 mmol/l
WBC	10.0 * 10 /l 9	Urea	4.5 mmol/l
Neuts	7.6 * 10 /l 9	Creatinine	65 µmol/l
Lymphs	0.8 * 10 /l 9	CRP	32 mg/l
Eosin	1.4 * 10 /l9		
Chest x-ray	hyperexpanded film with no consolidation or pneumothorax		

What recording is essential when administering aminophylline?

A. Liver function tests
B. Aminophylline levels
C. Repeat potassium
D. Serial ECGs
E. Cardiac monitor

E. Cardiac monitor

Aminophylline loading dose should be done with cardiac monitoring

Aminophylline is a methylxanthine and acts as a phosphodiesterase inhibitor and adenosine receptor antagonist. It therefore can act oppositely to adenosine in the heart and induce tachyarrhythmias. Therefore, for loading of aminophylline, a cardiac monitor needs to be applied. Aminophylline levels would be needed if this patient was already taking theophylline and are used to check maintenance levels. Aminophylline can induce hypokalaemia but this is less important than cardiac monitoring. Aminophylline can induce the liver but monitoring of LFTs is not usually required.

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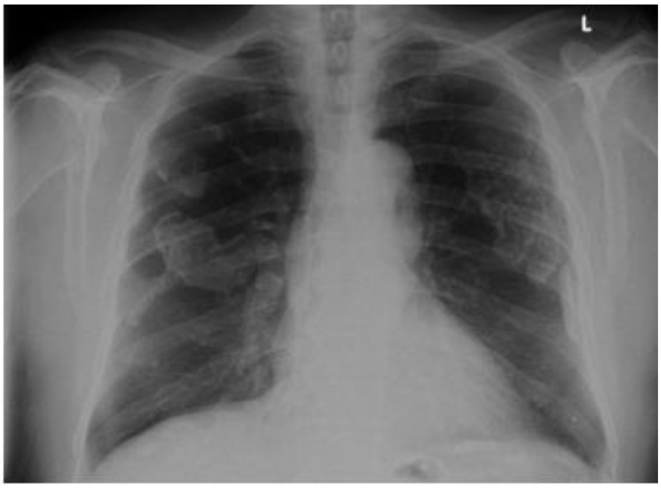
A 67-year-old retired plumber is seen in the respiratory clinic following an incidental finding of a chest x-ray ordered by his general practitioner. He recently recovered from an infection with *Mycobacterium tuberculosis* 6 weeks ago and reports to have been feeling well since with no complications. The remainder of his past medical history includes mild gastritis and hypertension.

On examination, his observations are recorded as follows:

- Temperature 36.1ºC
- Heart rate 89bpm
- Respiratory rate 22 breaths/min
- Blood pressure 118/65mmHg
- Saturations 99% on room air

On auscultation, chest sounds are clear and heart sounds are normal. Calves are soft and nontender.

A repeat chest x-ray is ordered (shown below):



What is the single biggest risk factor for this patient's investigation findings?

A. Mycobacterium tuberculosis
B. Asbestos
C. Aspergillus
D. Silica
E. Tobacco

B. Asbestos

This chest x-ray demonstrates multiple pleural plaques. They appear as slightly opacified pleural markings of a variety of different shapes with a visible contrast between their outlining border and adjacent normal lung markings. This patient's pleural plaques are most visible in the lateral aspect of the left upper zone and throughout the middle zone of the right lung. Pleural plaques are areas of thickened pleural tissue which arise secondary to asbestos exposure. This patient is a retired plumber and it is likely that his profession was the means of exposure to asbestos. The development of pleural plaques is not immediate and can take up to 20-40 years until visible on imaging. Pleural plaques do not undergo malignant change. Therefore, no follow-up is required for these patients.

Mycobacterium tuberculosis can cause a variety of lung pathology ranging from miliary tuberculosis to parenchymal calcification and necrosis. The infection does not give rise to pleural plaques as demonstrated in this chest x-ray, making it incorrect.

Aspergillus is a fungal infection responsible for a variety of different presentations including invasive aspergillosis and aspergillomas. An aspergilloma is a round mass of the fungus Aspergillus that occupies existing lung cavities such as those from previous *Mycobacterium tuberculosis* infections. It can be detected on a chest x-ray as a spherical or ovoid mass, commonly in the upper zones of the lung.

Silica exposure can lead to silicosis, the progressive inflammation of lung tissue. Stonemasons, mining, pottery and ceramics are the most common routes of silica exposure. Silicosis commonly causes an upper zone pulmonary fibrosis which is not seen here.

Tobacco use is most commonly associated with chronic obstructive pulmonary disease and malignancy (usually squamous cell carcinoma of the lung or small cell carcinoma). Asbestos and tobacco exposure are synergistic with one another the risk of malignancy. However, tobacco use alone is not associated with pleural plaques

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A 65-year-old man presents to the respiratory clinic with worsening haemoptysis the past 3 months. He is also concerned as he has noticed a loss of 6kg in the past 2 months. He previously completed a course of tuberculosis (TB) treatment 20 years ago, and he is currently a smoker, with a 50 pack year history.

On examination he is cachectic. Temperature is 36.8ºC, heart rate 80 bpm, respiratory rate 18 breaths per minute. There are decreased breath sounds and dullness to percussion over the right upper lobe, and conjunctival pallor. His fingers are clubbed. The rest of his examination is unremarkable.

Investigations:

Na+	131 mmol/l
K+	4.3 mmol/l
Urea	6.1 mmol/l
Creatinine	102 µmol/l
Serum corrected calcium	2.35 mmol/l
Serum bilirubin	21 µmol/l
Serum alkaline phosphatase	85 IU/l
Serum aspartate aminotransferase	16 IU/l
C Reactive protein (CRP)	15 mg/l
Haemoglobin	110 g/l
Mean Corpuscle Volume	77 fL
White cell count	11.2 x 10^9/L
Neutrophils	7.5 x 10^9/L
Eosinophils	1.9 x 10^9/L
Lymphocytes	1.1 x 10^9/L
Basophils	0.0 x 10^9/L
Platelets	490 x 10^9/L
INR	1.0

Chest x-ray: Intracavitary mass in the right upper zone

What is the next most useful diagnostic test?

A. Aspergillus precipitins
B. Bronchoalveolar lavage
C. Sputum cultures
D. Bronchoscopy and biopsy
E. Skin prick test

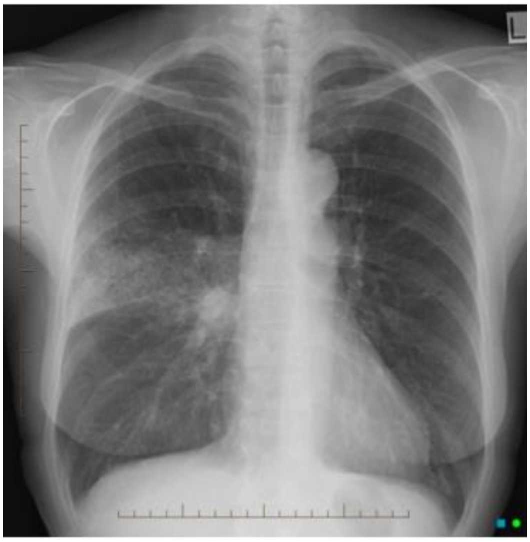
A. Aspergillus precipitins

This gentleman has haemoptysis, weight loss, previous TB and a mass in the right upper zone. The differentials would include recurrent TB, malignancy or an aspergilloma. In view of his eosinophilia in the differentials, the most likely diagnosis would be an aspergilloma, where a fungal ball of aspergilli grows in a pre-existing pulmonary cavity unchecked, as the immune system is unable to penetrate the cavity.

The diagnosis of aspergillosis includes the finding of eosinophilia on the full blood count, as well as raised IgE. Skin prick tests for aspergillus spp. may not be positive in patients with an aspergilloma. Serological tests for aspergillus precipitins are more sensitive than sputum cultures for diagnosing aspergillomas. Bronchoalveolar lavage and bronchoscopy are less effective in diagnosing aspergillomas. Another useful test is a CT scan of the chest, which can also reveal malignancy if suspected.

Question 82 of 133

A 71-year-old woman is admitted with shortness-of-breath to the acute medical unit. Her carers report that she has been feverish and confused for the past two:



What is the main finding on the film?

A. Bronchiectasis
B. Right middle lobe consolidation
C. Tuberculosis
D. Pulmonary abscess
E. Right upper lobe consolidation

E. Right upper lobe consolidation

This film demonstrates classical signs of right upper lobe consolidation - abnormal opacity within the right upper lobe abutting the horizontal fissure.

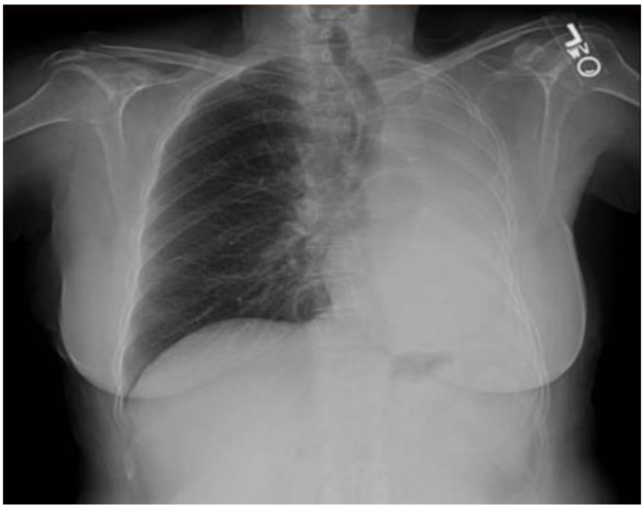
Question 83 of 133

A 70-year-old homeless woman is admitted to the Emergency Department after being found

unconscious by staff at a local homeless shelter. She has a history of alcohol excess according to

staff. On arrival in the department she appears to be intoxicated and confused. On examination you noticed heavy tar staining of the fingers and a pulse of 110/min which is irregularly irregular.

Her respiratory rate is 16/min with oxygen saturations of 92% on room air. Breath sounds are reduced on the left side of the chest. A chest x-ray shows the following:



What is the most likely diagnosis?

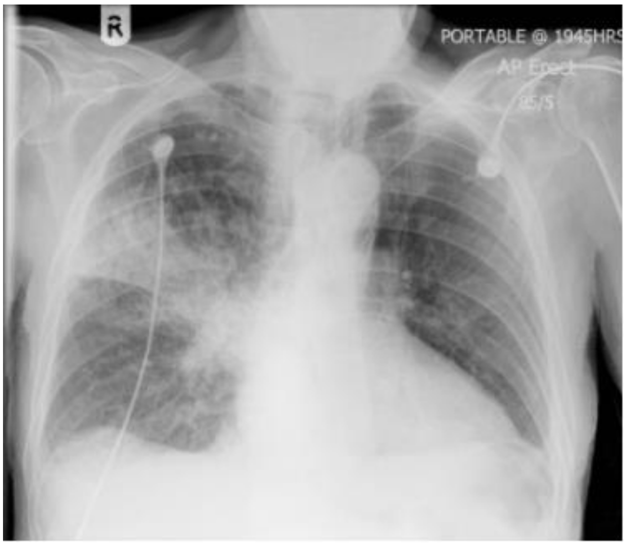
A. Pleural effusion
B. Pneumonia
C. Pneumonectomy
D. Diaphragmatic hernia
E. Mesothelioma

C. Pneumonectomy

The x-ray shows an opacified left hemithorax with mediastinal and tracheal shift towards the affected side. Crowding of the ribs over the affected side with compensatory overinflation of normal lung is also seen.

Question 84 of 133

A 75-year-old man is admitted to the Emergency Department with dyspnoea. A chest x-ray is performed upon arrival:



What is the main finding on the film?

A. Bronchiectasis
B. Right sided pleural effusion
C. Right upper lobe consolidation
D. Pulmonary oedema

E. Right middle lobe collapse

C. Right upper lobe consolidation

This film demonstrates classical signs of right upper lobe consolidation - abnormal opacity within the right upper lobe abutting the horizontal fissure.

Question 85 of 133

A 66-year-old man is referred to the Acute Medical Assessment Unit for progressive shortness of breath by their GP. Previous medical history includes ischaemic heart disease, left ventricular hypertrophy with reduced ejection fraction, hypertension and type 2 diabetes. He is a retired dockworker and has smoked 20 cigarettes a day for the last 50 years.

Hb	150 g/L	Male: (135-180)Female: (115 - 160)
Platelets	254 * 10^9/L	(150 - 400)
WBC	11.6 * 10^9/L	(4.0 - 11.0)
Na^+	132 mmol/L	(135 - 145)
K^+	3.9 mmol/L	(3.5 - 5.0)
Urea	6.5 mmol/L	(2.0 - 7.0)
Creatinine	119 µmol/L	(55 - 120)
CRP	15 mg/L	(< 5)

A chest x-ray shows bilateral pleural effusions, cardiomyopathy and pleural plaques.

He is admitted for intravenous diuresis to treat his pleural effusions. He initially responds to treatment but there is a significant residual pleural effusion on the right-hand side.

What investigation would you undertake next for the residual right-sided pleural effusion?

A. Bronchoscopy
B. Chest drain with a sample of pleural fluid sent for investigation
C. Continue diuresis and review
D. Diagnostic aspiration
E. High resolution computed tomography of the thorax

D. Diagnostic aspiration

In the assessment of a pleural effusions, if the clinical picture suggests a transudate, then you should treat the cause initially. If this does not resolve the effusion then perform a diagnostic aspiration

Diagnostic aspiration is correct. A pleural aspiration is appropriate now that a malignant or exudate pleural effusion is suspected. Initially, the bilateral pleural effusions were suspected to be transudate due to the patient's cardiovascular history and were treated with diuresis. However, the residual effusion, which fails to resolve despite the resolution of the left-sided effusion, needs further investigation. This can either be a small or large volume aspiration depending on the patient's clinical picture and degree of respiratory compromise. A small volume would be for diagnostic purposes, if the patient remains compromised by the effusion, for example, work of breathing, oxygen requirement or exercise tolerance, then a large volume aspiration can be performed. It is very important to ensure that you have all the sampling equipment ready at the time of the intervention as multiple sample collection devices are often required. A chest drain may be required based on the results of the diagnosis aspiration.

Bronchoscopy is incorrect as this does not investigate pleural effusions. Uses for bronchoscopy include; suspected tumour, biopsy (including end-bronchial ultrasound), foreign body, infection or inflammation. Indications for this include localised wheeze from a possible foreign body, haemoptysis, biopsy of proximal tumour seen on CT, mucus sample collection for atypical infections and diagnosis of sarcoidosis.

Chest drain with a sample of pleural fluid sent for investigation is incorrect. The initial investigation for a possible malignant or exudate effusion would be a pleural aspiration and a review of samples taken. Should this continue to be a transudate effusion chest drain insertion would not be the next step.

Continue diuresis and review is incorrect. The initial suspicion was that the patient had bilateral transudate effusions. These do not need aspiration of the fluid and the patient was treated with a diuretic. However, there has been a failure to resolve unilaterally and given the patient's pleural plaques and, likely asbestos exposure in his employment, further investigation is warranted.

High resolution computed tomography of the thorax is incorrect. This investigation would be more appropriate to investigate idiopathic interstitial pneumonia, asbestosis, inflammation, drug reaction or diffuse alveolar damage.

Question 86 of 133

You are reviewing a 70-year-old man in the respiratory outpatient clinic.

He recently presented to the hospital with dyspnea. This has been gradually getting worse over several months and then acutely worsened in the days leading up to his hospital admission. His exercise tolerance had changed from a baseline MRC dyspnoea score of 3 to 5. There was an associated productive cough of white sputum. There was no history of a purulent cough, fevers or chest pain. He is a smoker with a 50 pack year history and his previous jobs were in administration. He has no pets. He denies any other medical or surgical conditions.

Examination at the time of hospital presentation revealed reduced breath sounds globally, expiratory wheeze and fine bibasal crepitations that did not change with coughing.

You reviewed this gentleman in the hospital and he was managed as a non-infective exacerbation of newly diagnosed chronic obstructive pulmonary disease, with nebulized bronchodilators and a short course of prednisolone. He was discharged on a combination Glycopyrronium and Indacaterol inhaler and you had arranged further outpatient investigations.

The results of these tests are available in today's clinic:

Tests	Result
FEV1	108% predicted
FVC	107% predicted
Total lung capacity	121% predicted
Diffusion capacity for carbon monoxide	36% predicted
High-resolution CT thorax	centrilobular emphysematous change in the upper lobes bilaterally. There

	are subpleural reticular opacities and honeycombing in both lung bases, with no traction bronchiectasis or ground glass opacities

Whilst his dyspnoea is improved, he still has an MRC dyspnoea score of 3-4. Repeat examination today in the clinic is unchanged from his examination findings in hospital.

The patient and his wife are very concerned about the deterioration in his breathing.

Which of the following is correct regarding his condition?

A. He has combined pulmonary fibrosis and emphysema (CPFE)
B. He has emphysema with non-specific interstitial pneumonia (NSIP)
C. His risk of developing lung cancer is similar when compared to patients with emphysemaalone
D. His risk of developing pulmonary arterial hypertension is similar when compared topatients with emphysema alone
E. The changes seen in the upper lobes on the high resolution CT thorax are atypical forsmoking related emphysema.

A. He has combined pulmonary fibrosis and emphysema (CPFE)

Combined pulmonary fibrosis and emphysema (CPFE) has a much higher rate of lung cancer than emphysema alone

This gentleman has a syndrome called combined pulmonary fibrosis and emphysema (CPFE). Patients with CPFE have significantly higher rates of lung cancer compared to those with emphysema (or idiopathic pulmonary fibrosis) alone. They also have an increased risk of developing pulmonary arterial hypertension, which is often less responsive to medical treatments, meaning lung transplantation should be considered in such cases.

CPFE is characterized by exertional dyspnoea, upper-lobe emphysema and lower-lobe fibrosis, preserved lung volume and severely reduced capacity of gas exchange. The preserved lung volumes are thought to arise because of the counterbalanced effects of hyperinflation from emphysema and the restrictive effects of pulmonary fibrosis. Both mechanisms lead to reduced gas exchange, hence the significantly reduced DLCO.

Option A - the correct answer

Options B - the HRCT findings of basal predominance fibrosis and honeycombing is consistent with a usual interstitial pneumonia (UIP) pattern. Traction bronchiectasis may or may not be present in UIP. Non-specific interstitial pneumonia (NSIP) is radiologically characterized by symmetric bilateral areas of ground-glass opacity with superimposed fine reticular opacities but with no or only minimal honeycombing

Options C and D - as described above, patients with CPFE have a significantly higher risk of developing lung cancer and/or pulmonary arterial hypertension

Option E - Emphysema secondary to smoking is typically upper lobe predominant, centrilobular and commonly manifests as small, localized areas of low attenuation within the central portion of the secondary pulmonary lobule on HRCT

Question 87 of 133

A 76-year-old man presented with vague lower abdominal pain. He was known to have underlying type 2 diabetes mellitus,hypertension and severe chronic obstructive pulmonary disease(COPD). He was a smoker with 50 pack years smoking history.

A CT abdomen was taken which showed diverticular disease of the colon. Incidentally it also showed a 2.5 cm mass in the periphery of the lower lobe of the left lung. Subsequently he underwent a positron emission tomography(PET) scan which showed the lung lesion with standardised uptake value(SUV) of 12.5 which was highly suspicious of cancer. There was no evidence of spread to lymph nodes or distant sites. A CT guided biopsy of the lung lesion was done which showed squamous cell carcinoma. His performance status was 2 in Eastern Cooperative Oncology Group(ECOG) scale. His lung functions showed an FEV1 of 0.8L(25% predicted) and FVC of 1.3L(30% predicted). TLCO was 30% predicted.

Which of the following is a suitable choice of treatment for him?

A. Submit answer
B. Lobectomy
C. Chemotherapy
D. Radical radiotherapy
E. Lobectomy+chemotherapy
F. Best supportive care

C. Radical radiotherapy

This is a stage IB lung cancer. Surgery is the best curative option provided the lung functions and performance status are suitable for surgery.

NICE guidelines says- 'Patients who have poor lung function but are otherwise suitable for radical radiotherapy should still be offered radiotherapy, provided the volume of irradiated lung is small'.

European Society for Medical Oncology (ESMO) guidelines-2014 says 'in those patients who are not suitable for surgery and with a peripheral tumour(like the patient in the question), Stereotactic Ablative Radiotherapy(SABR) given at a biologically equivalent dose of >100Gy is the preferred treatment of choice. A systematic review comparing outcomes of SABR and surgery in patients with severe COPD revealed a higher 30-day mortality following surgery but similar overall survival at 1 and 3 years. NCCN(National Comprehensive Cancer Network) guidelines-2015 for non-small cell lung cancer says SABR is an appropriate option for patients with high surgical risk. SABR and sublobar resection both achieve comparable cancer specific survival and primary tumour control.

Question 88 of 133

A 64 year-old female patient with known chronic obstructive pulmonary disease presents for routine review at respiratory outpatients. She is currently taking inhaled tiotropium and salmeterol/fluticasone at optimal doses. You note she has had two exacerbations in the previous 12 months requiring oral steroids and antibiotics. A recent high resolution CT showed severe emphysema affecting all lobes.

Her ABGs in clinic today is a follows:

pH	7.38
pO2	7.91 kPa
pCO2	6.7 kPa

HCO3	30.1 mmol/L
Sats	88%

Her blood tests today show:

Hb	16.6 g/dL
Platelets	256 x 10 9 /L
WCC	6.7 x 10 9 /L

How would you optimise her management further?

A. Refer to lung volume reduction surgery
B. Make no changes to current management
C. Consider long term oxygen therapy
D. Start prophylactic antibiotics
E. Refer for assessment for home non-invasive ventilation

C. Consider long term oxygen therapy

This lady is polycythaemic and has a resting pO2 less than 8.0kPa and therefore is a candidate for long term oxygen therapy. It may be prudent to repeat an ABG in one month to ensure a findings are similar prior to staring oxygen.

Question 89 of 133

A 72-year-old man was admitted to hospital with an exacerbation of his longstanding chronic obstructive airways disease. The patient's regular treatment for COPD included home oxygen, home bronchodilator nebulisers and high-dose inhaled steroids. Normal exercise tolerance was very limited with the patient becoming profoundly breathless after mobilising only short distances around his house. Following an admission to the intensive care unit for respiratory support the previous winter the patient had expressed a wish that he would not want non-invasive ventilation or intubation in the future and stated that control of his symptoms was his priority.

Treatment with prednisolone, nebulised bronchodilators and antibiotics was started on admission. Having been profoundly dyspneoic at rest at admission the patient's symptoms improved gradually over the next week until the patient returned to his baseline of dyspnoea on minimal physical exertion. He again requests if there is any other treatments that can ameliorate his symptoms.

Recent investigations are summarised below.

Forced vital capacity: 115 % predicted Forced expiratory volume (1s): 34 % predicted FEV1 / FVC: 30 % predicted

Haemoglobin	170 g / dL
White cell count	15.8 * 10 /l9
Platelets	167 * 10 /l9
Urea	6.7 mmol / L
Creatinine	98 micromol / L
Sodium	140 mmol / L
Potassium	4.1 mmol / L
Packed cell volume	0.42

What treatment is the best choice for relief of dyspnoea in this patient?

A. Short-acting liquid morphine sulphate prn
B. Venesection
C. Diazepam 2mg qds
D. High-flow oxygen (15L, 100%)
E. Refer to thoracic surgery for consideration of pleurodesis

A. Short-acting liquid morphine sulphate prn

The patient has end-stage COPD and has himself indicated that control of symptoms is his priority. As such, consideration of the use of opioid or benzodiazepine medications for symptomatic relief of breathlessness is appropriate. A recent prospective study demonstrated that lower dose opioid use (< 30 mg daily oral morphine equivalent) does not increase risk of hospitalisation or increase mortality in patients with COPD on long-term oxygen therapy.

Clinical Knowledge Summaries also support this approach:

Offer an opioid as first-line treatment to help breathlessness in people with end-stage chronic obstructive pulmonary disease (COPD) that is unresponsive to other medical treatment.

Benzodiazepines were shown to not increase admission rates but did increase mortality. The patients packed cell volume is normal; therefore venesection has no role in this case

Question 90 of 133

A 63 year old gentleman presented to the acute medical unit with a 3 day history of increasing shortness of breath and cough. He is known to have chronic obstructive pulmonary disease (COPD) and is treated as an infective exacerbation of COPD.

He has a past medical history of ischaemic heart disease, type 2 diabetes mellitus, hypothyroidism, hypercholesterolaemia, hypertension, epilepsy and osteoarthritis.

Despite nebulisers, steroids and antibiotics, he remains acutely breathless and his oxygen saturations are only 83% on air.

Blood gas analysis is as follows:

pH	7.21
pCO2	10.1 kPa
pO2	7.3 kPa
BE	-4.2 mmol/l
HCO3-	36 mEq/l

You opt to try non invasive ventilation (NIV) and he is transferred to the NIV unit. Initial settings on NIV are IPAP (inspiratory positive airways pressure) 10 cm H2O and EPAP (expiratory positive airways pressure) 4 cm H2O. He remains tachypnoeic and has oxygen saturations of 88%. His repeat blood gas after 1 hour is as follows:

pH	7.25	
pCO2	9.9 kPa	
pO2	9.3 kPa	
BE	-4.0 mmol/l	
HCO3-	35 mEq/l	

Firstly, how would you adjust* the *NIV settings?

A. Increase IPAP by 2 cm H2O
B. Increase EPAP by 2 cm H2O
C. Increase IPAP and EPAP by 2 cm H2O
D. No change in settings
E. Add extra inspired oxygen to NIV at 24%

A. Increase IPAP by 2 cm H2O

This gentleman is in type 2 respiratory failure secondary to an infective exacerbation of COPD. Initial treatment has failed and NIV is commenced. This helps somewhat initially, but an adjustment in NIV settings is required for optimisation. Given that his main issue is CO2 retention, an increase in IPAP is the correct response as this helps 'blow off' CO2. increasing EPAP splints the airways open and is effectively PEEP and therefore increases oxygenation. This is not particularly required in this gentleman as, although his pO2 is low, this is probably approaching normal for a COPD patient.

Question 91 of 133

A 91-year-old man is admitted into hospital with a severe exacerbation of his COPD. He is breathless on rest and although alert and orientated, is starting to become drowsy. His observation reveal a temperature of 37.9ºC, a heart rate of 82 beats per minute, blood pressure of 116/74 mmHg, respiratory rate of 28 breaths per minute and oxygen saturations of 84% on a 28% Venturi mask.

An arterial blood gas is performed and reveals a PaO2 of 9.0 kPa, PCO2 of 11.4kPa, HCO3 of 31 mmol/l and a pH of 7.29.

A decision is made to commence the man on non-invasive ventilation –

which of the following is the most appropriate management for this patient?

A. Inspiratory positive airways pressure (IPAP) of 10cm water and expiratory positive airways pressure (EPAP) of 4cm water at 60% flow rate of oxygen
B. Inspiratory positive airways pressure (IPAP) of 10cm water and expiratory positive airwayspressure (EPAP) of 4cm water at 20% flow rate of oxygen
C. Inspiratory positive airways pressure (IPAP) of 10cm water and expiratory positive airwayspressure (EPAP) of 4cm water at 40% flow rate of oxygen
D. Inspiratory positive airways pressure (IPAP) of 10cm water and expiratory positive airwayspressure (EPAP) of 6cm water at 40% flow rate of oxygen
E. Inspiratory positive airways pressure (IPAP) of 4cm water and expiratory positive airwayspressure (EPAP) of 10cm water at 40% flow rate of oxygen

C. Inspiratory positive airways pressure (IPAP) of 10cm water and expiratory positive airwayspressure (EPAP) of 4cm water at 40% flow rate of oxygen

The British Thoracic Guidelines state that oxygen saturations of 90-92%, with an initial noninvasive ventilation setting of Inspiratory positive airways pressure (IPAP) of 10cm water and expiratory positive airways pressure (EPAP) of 4cm water. To assess the effect of the treatment, an arterial blood gas should be taken an hour after the non-invasive ventilation is set up.

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A 24 year-old student was referred to the Acute Medical Unit by his general practitioner with a 4- week history of increasing cough and breathlessness. The cough was generally non-productive but he had coughed up a small amount of blood on 3 occasions. His past medical history consisted only of asthma which was well controlled with a salbutamol inhaler. There was no family history of venous thromboembolism. His recent travel history included a trip to Sierra Leone 3 months ago. He was a non-smoker and drank 20 units of alcohol per week.

On examination, his temperature was 37.6oC, heart rate 80 beats per minute, blood pressure 124/88 mmHg, respiratory rate 22 breaths per minute and oxygen saturations 92% on room air. He was able to talk in full sentences. A few bibasal crackles were evident on auscultation of the chest. His JVP was not elevated and heart sounds were normal.

Investigations:

Haemoglobin	144 g/L (130-180)
White cell count	7.0 * 10 /l
Neutrophil count	3.5 * 10 /l
Lymphocyte count	1.6 * 10 /l
Eosinophil count	1.3* 10 /l
Platelets	330 * 10 /l
Sodium	138 mmol/L
Potassium	3.9 mmol/L
Urea	7.0 mmol/L
Creatinine	65 mol/L
Alkaline phosphatase	40 IU/L
Alanine aminotransferase	33 IU/L
Gamma-glutyl transferase	18 IU/L
Bilirubin	14 mol/L
Albumin	40 g/L

Chest x-ray: Bilateral lower zone haziness ECG: Normal sinus rhythm

What is the most appropriate treatment for the underlying ***condition?***

A. Mebendazole
B. Low molecular weight heparin
C. Fluconazole
D. Doxycycline
E. Artesunate

A. Mebendazole

This patient has presented with features of eosinophilic pneumonia secondary to parasite infection, also known as Loefflers syndrome. The most likely causative organism in this case is the roundworm *Ascaris lumbricoides*, which can cause Loefflers syndrome during the pulmonary migration phase of its life cycle. The clinical features and eosinophilia could also point to idiopathic pulmonary eosinophilia or allergic bronchopulmonary aspergillosis, but the treatment for both of these (corticosteroids) is not listed as a potential answer. The chest x-ray findings and normal ECG make pulmonary embolism a less likely diagnosis.

Question 93 of 133

A 63-year-old man has a check chest x-ray 6 weeks after being treated for community-acquired pneumonia. He reports feeling much better in himself from a chest point of view. However, he has noticed his face has been getting rounder and he has put on weight, particularly in his abdominal region. He has also noticed purplish stripes on his abdomen which has alarmed him.

He has recently been diagnosed with hypertension and reports his GP told him he was borderline diabetic. His medications at present are ramipril. He drinks 4-6 units of alcohol per week, has a 40 pack-year smoking history and is a retired salesman.

Initial chest x-ray	Bibasal patch opacification in keeping with pneumonia. Clinical correlation advised.
Check chest x-ray	Compared to the previous x-ray, the consolidation has resolved. However, there is a right hilar mass suspicious for malignancy. Further investigation advised.

The patient has been informed of his chest x-ray results and understands that he will be referred urgently to the lung cancer clinic.

Given the presentation, what type of lung cancer is the patient likely to have?

A. Metastatic deposits
B. Mesothelioma
C. Adenocarcinoma of the lung
D. Squamous cell lung cancer
E. Small cell lung cancer

E. Small cell lung cancer

Small cell lung carcinoma secreting ACTH can cause Cushing's syndrome

his man has a significant smoking history and the repeat chest x-ray has revealed a suspicious mass, highlighting the importance of getting a follow-up x-ray for patients 6 weeks after community-acquired pneumonia (to check for resolution and any evidence of underlying lesions). The central adipose deposition, 'moon face', new hypertension and impaired glucose tolerance are all in keeping with Cushing's syndrome, This is a paraneoplastic phenomenon seen in small cell lung cancer due to the tumour secreting ACTH.

If these were metastatic deposits, the chest x-ray would show multiple lesions rather than a solitary hilar mass.

Mesothelioma is a cancer of the pleura due to asbestos exposure. There is nothing in the vignette to suggest asbestos exposure. In questions where asbestosis exposure may be relevant, the occupation of the patient is usually that of a plumber or builder.

Adenocarcinoma of the lung is associated with gynaecomastia.

Squamous cell lung cancers can secrete parathyroid hormone-related peptide which causes hypercalcaemia.

Question 94 of 133

A 58-year-old gentleman presents with shortness of breath and haemoptysis which has worsened over ten days. He is currently coughing up an estimated 200ml of fresh red blood every day. He has night sweats and feels cold and sweaty at times. He has not lost any weight and was well prior to this.

He has mild COPD and takes fostair twice a day with no exacerbations in the past 5 years. He moved to the UK 15 years ago but lived 10 years in Louisiana as a mining operator extracting minerals with exposure to bats and birds during this time. He drinks 10 units of alcohol a week and was born in Syria where he received a BCG vaccine as a child. He has not been around any ill contacts and lives with his wife and two children. There is no recent foreign travel and he keeps no pets.

On examination, he has crepitations in the right side mid and upper zones. He has palpable lymph nodes in his axillae and neck but his abdomen is non tender with no hepatosplenomegaly. He is breathing rapidly and appears cachectic. His observations show temperature 38.3ºC, respiratory rate 28/min, heart rate 98/min, saturations 88% in air. An arterial blood gas has not been done.

Hb	110 g/l	Na +	139 mmol/l
Platelets	450 * 10 /l 9	K +	3.4 mmol/l
WBC	13 * 10 /l 9	Urea	4.1 mmol/l
Neuts	9.8 * 10 /l 9	Creatinine	78 µmol/l
Lymphs	0.78 * 10 /l 9	CRP	130 mg/l
Eosin	0.1 * 10 /l9		
HIV serology	negative		
Sputum culture	no acid fast bacilli or other growth seen		
Chest x-ray	bilateral hilar lymphadenopathy with patchy right upper zone consolidation		
Beta-D-glucan	strongly positive		

What is the likely ***diagnosis?***

A. Aspergillosis
B. Tuberculosis
C. Histoplasmosis
D. Pneumocystis jirovecii
E. Lung carcinoma

C. Histoplasmosis

Histoplasmosis can affect the lung and present with acute respiratory failure

This gentleman has an acute history of respiratory failure with deterioration. All the causes above are possible but he is not HIV positive so *pneumocystis* is unlikely. This presentation is very acute for lung carcinoma. He has been vaccinated against tuberculosis.

The raised Beta-D-glucan suggests a fungal cause of infection. Given his previous exposure to bats and their droppings in the Mississippi area, he is likely to have developed invasive histoplasmosis which is known to present many years after initial exposure to the fungus.

Question 95 of 133

An 18-year-old male is referred to the medical assessment unit by the emergency department.

He presents with a 3-day history of a cough productive of green sputum, haemoptysis, subjective fevers, exertional dyspnea and right-sided pleuritic chest pain. This is the third presentation in 12 months. The first time he presented to his general practitioner and his symptoms improved with a course of oral amoxicillin. The second presentation was a few months ago where he required inpatient management with intravenous antibiotics. He failed to attend follow up as he became busy with his studies. Over the last 12 months, he has also noticed increasing lethargy and weight loss. He denies any other past medical or surgical history. He admits to smoking when he goes out with his friends at the weekends and to binge drinking at the weekends. There is no history of intravenous or other illicit drug use.

He was born in Nigeria and has been living in the United Kingdom for the last 12 months. He is studying for his undergraduate degree in mathematics and also works part-time in a restaurant.

On examination, observations revealed a respiratory rate of 19/min, oxygen saturation 94% on air, heart rate 110/min, blood pressure 100/60 mmHg and a temperature of 38ºC. Examination of the respiratory system revealed no clubbing or cervical lymphadenopathy. There were reduced breath sounds and dullness to percussion at the right base. Cardiovascular examination was normal. Calves were soft and non-tender, with no pedal oedema.

Hb	140 g/l	Na	136 mmol/l
Platelets	400 * 10 /l	K	4 mmol/l
WBC	12 * 10 /l	Urea	6 mmol/l
Neuts	11 * 10 /l	Creatinine	70 µmol/l
Lymphs	1 * 10 /l	CRP	30 mg/l

Chest x-ray showed an elevated right hemidiaphragm, depressed right hilum and obliteration of the right costophrenic angle.

Computed tomography (CT) of the thorax is obtained. This shows a small right pleural effusion, right lower lobe collapse and a smooth round mass in the proximal right main bronchus, partially obscuring its lumen, which had marked post-contrast enhancement. There was no hilar or mediastinal lymphadenopathy.

Three early morning sputum samples are sent which are negative for acid-fast bacilli. Mycobacterial culture is awaited. No malignant cells were seen on sputum cytology. Bronchoscopy is performed and biopsy of the mass is obtained.

What is the most likely histological diagnosis?

A. Adenocarcinoma
B. Squamous cell carcinoma
C. Small cell carcinoma
D. Large cell carcinoma
E. Carcinoid tumour

E. Carcinoid tumour

Bronchial carcinoid is the most common lung cancer in adolescents and can present with recurrent pneumonia

Bronchial carcinoid is a rare form of lung cancer but is much more common in younger patients, typically presenting in late adolescence. They are typically slow growing, well-differentiated tumours, explaining the protracted history we see in this patient. The majority (approximately 75%) of bronchial carcinoid tumours arise in the proximal airways, hence why symptoms include dyspnea, wheeze, cough, haemoptysis and/or recurrent pneumonia. Patients may receive several courses of antibiotics to treat recurrent pneumonia before the tumour is diagnosed, as in this case.

Studies have shown a positive association with smoking and the development of bronchial carcinoid, but causality has not been proven as with smoking and more common types of lung cancer.

Cavitation is rare. Pleural effusion is also uncommon but may be seen as a complication of pneumonia caused by the obstruction from the tumour. The chest x-ray description, in this case, is consistent with right lower lobe collapse and a right pleural effusion.

Only 5-20 per cent of typical low-grade lung neuroendocrine tumours (NETs) are associated with hilar or mediastinal adenopathy. Another clue in this case to carcinoid is that contrast-enhanced CT scans frequently show marked enhancement due to the vascular nature of the tumours.

Lung NETs tend to produce lower quantities of serotonin compared to intestinal NETs, which explains why lung NETs have lower rates of carcinoid syndrome, which is characterized by flushing, diarrhoea, bronchospasm and can reflect metastases to the liver. Option 1-4 - unlikely given the patient's age, relatively light smoking history and absence of lymphadenopathy. Central carcinomas are more likely to provide positive sputum cytology for malignant cells, whereas that is not the case with central bronchial carcinoid due to an intact bronchial mucosa usually overlying NETs.

Option 5 - the correct answer

Question 96 of 133

A 60-year-old man comes to his GP complaining of cough and dyspnoea on exertion. He has had the cough for over 2 years, but it has gotten worse recently along with increasing shortness of breath. He has no haemoptysis, chest pain or leg swelling. The patient has a 60 pack-year smoking history and drinks a moderate amount of alcohol. His temperature is 37.2ºC, blood pressure is 140/80 mmHg, pulse is 80/min, and respirations are 20/min.

Chest x-ray shows prominent bronchovascular markings and mild diaphragmatic flattening. Pulmonary function test results are as follows:

Forced expiratory volume in 1 second	67% of predicted
Forced vital capacity	95% of predicted
FEV1/FVC ratio	0.65

Carbon monoxide diffusion capacity is 100% of predicted value.

Which of the following is the most likely cause of this patients symptoms?

A. Asbestosis
B. Chronic bronchitis
C. Panacinar emphysema
D. Pulmonary fibrosis
E. Rheumatoid lung

B. Chronic bronchitis

The patients pulmonary function test (PFT) findings are consistent with obstructive lung disease. The carbon monoxide diffusing capacity of the lung (DLCO) measures the lungs ability to transfer gas to the blood. This patients normal DLCO indicates intact alveolar and capillary structure.
The patients chronic smoking, productive cough, dyspnoea on exertion and decreased FEV1/FVC ratio suggests COPD. COPD is a group of chronic disorders; the main subtypes being emphysema or chronic bronchitis. Patients with predominant emphysema are generally thin with severe dyspnoea, hyperinflated chest, decreased vascular markings, decreased DLCO (due to alveolar destruction) and moderate oxygen desaturation

Patients with predominant chronic bronchitis have chronic productive cough for 3 months over 2 consecutive years not due to other causes (e.g. bronchiectasis). It usually presents with prominent bronchovascular markings, mildly flattened diaphragm and normal DLCO (as seen in this patient).

Choices 1, 4 & 5: These conditions are examples of restrictive lung diseases that cause reduced lung volumes, normal FEV1/FVC ratio, decreased FVC and low DLCO.

Choice 3: Panacinar emphysema is typical for α-1-antitrypsin deficiency; centriacinar emphysema is more characteristic for smoking-induced COPD. Panacinar emphysema usually causes a low DLCO

Question 97 of 133

A 58-year-old woman presents to the acute medical take with chest pain and ongoing coryzal symptoms for the last few weeks. The chest pain is retrosternal in origin, dull in character, around 3/10 intensity and does not radiate. When questioned, the patient is having a cough with white sputum and small streaks of blood, which she says has become worse over the last two weeks.

For the past two years, she has been working in the Mississippi river basin with a team of geologists. She has never smoked, does not keep any pets and has not travelled anywhere else.

On examination, she is pyrexial at 38ºC, has bibasal crepitations with increased vocal resonance and dullness to percussion at both bases.

Troponin	3 ng/L	(<14)
Chest x-ray	Airspace shadowing with consolidation involving multiple lung segments	

What is the best* management *option?

A. Co-trimoxazole
B. Fluconazole
C. Itraconazole
D. Ivermectin
E. Pentamidine

C. Itraconazole

Amphotericin or itraconazole are the pharmacological agents of choice for histoplasmosis

Itraconazole is the correct answer as this is the preferred treatment for histoplasmosis. The main clue for the diagnosis of histoplasmosis is the endemic nature of the fungal spores to Mississippi with 80% of residents in this area having positive *Histoplasma* serology, but most remaining asymptomatic. This woman has chest symptoms so requires active treatment. If an individual is immunocompromised then the histoplasmosis can progress to involve the central nervous system

Co-trimoxazole is an incorrect option as this is the first-line treatment option for *Pneumocystis carinii* pneumonia (PCP).

Pentamidine is incorrect as this is indicated for the treatment of *Pneumocystis carinii* (PCP).

Fluconazole is an incorrect option, even though this is an antifungal agent like Itraconazole, it is less effective.

Ivermectin is incorrect as this is an anti-parasitic drug, not anti-protozoal, working effectively against *Strongyloides* and severe scabies infestations.

Question 98 of 133

A normally fit and well 32-year-old black woman complains of a fever and tiredness over the past month. Her temperature is 38.2ºC, blood pressure is 115/70 mmHg, pulse is 75/min and respirations are 18/min.

On examination there were multiple non-tender cervical and axillary lymph notes. Lung auscultation reveals fine crackles throughout bilaterally. A chest x-ray film demonstrated hilar lymphadenopathy with diffuse interstitial infiltrates. A subsequent lymph node biopsy showed non-caseating granulomas.

Which of the following is the most appropriate therapy?

A. Supportive therapy
B. Methotrexate
C. Ciclosporin
D. Glucocorticoids
E. Infliximab

D. Glucocorticoids

Indications for corticosteroid treatment for sarcoidosis are: parenchymal lung disease, uveitis, hypercalcaemia and neurological or cardiac involvement

The patient's presentation is consistent with sarcoidosis. Characteristically the patient is usually asymptomatic, with disease detection occurring via an abnormal chest x-ray film which demonstrates bilateral symmetric hilar adenopathy usually with para-tracheal adenopathy and/or parenchymal infiltrates. In this case, the patient has reached the threshold for starting steroid treatment. None of the other medications have been shown to be beneficial in treating this grade of sarcoidosis

Question 99 of 133

A 38-year-old man presents to his general practitioner with shortness of breath and a productive cough of yellow sputum. He normally has a dry cough only. This is his third episode this year and he has received four courses of steroids and antibiotics. He is has only ever smoked once or twice at university. He works in a factory producing computer parts.

He was fully vaccinated as a child and well until 3 years ago and is otherwise well. He has no other past medical history.

On examination, he has scattered wheeze but no crepitations. He has a resonant hyperinflated chest and respiratory rate is 28 breaths per minute with saturations of 94% in air. There is no clubbing or cyanosis. He is awaiting lung function testing.

Hb	140g/l	Na +	138 mmol/l
Platelets	340 * 10 /l 9	K +	4.7 mmol/l
WBC	14.1 * 10 /l 9	Urea	6.2 mmol/l
Neuts	12 * 10 /l 9	Creatinine	89 µmol/l
Lymphs	3.2 * 10 /l 9	CRP	50 mg/l

Eosin	1.0 * 10 /l9
Chest x-ray	hyperinflated lung fields, flattened hemidiaphragm, no consolidation seen
Sputum culture	Moraxella spp

What additional test would benefit the patient?

A. Alpha-1-antitrypsin
B. Sweat test
C. CFTR genotyping
D. Saccharin test
E. Immunoglobulins

A. Alpha-1-antitrypsin

COPD in a non smoker should prompt suspicion of alpha-1 antitrypsin deficiency

This young man has recurrent infective episodes with wheeze. The main differentials are cystic fibrosis, bronchiectasis and COPD. Importantly, he was well as a child and only recently has developed these episodes as an adult. He has some smoke exposure and is clearly hyperinflated with wheeze and no crepitations. He has no other features of cystic fibrosis/bronchiectasis. These features point towards COPD. The main cause of COPD is smoking but young patients with severe alpha-1-antitrypsin deficiency can develop COPD even in the absence of cigarette smoking.

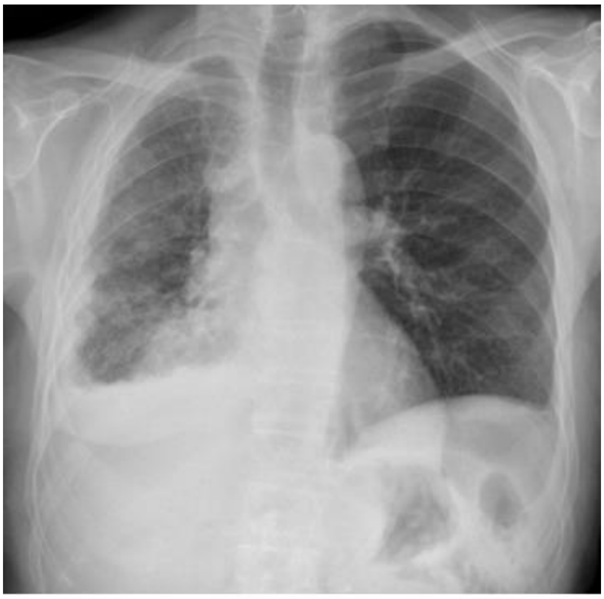
The sweat test and CFTR genotyping will diagnose cystic fibrosis. Saccharin testing is positive in ciliary dyskinesias and low immunoglobulins can cause bronchiectasis. The only test above that would explain this early COPD picture is the alpha-1-antitrypsin test

Question 100 of 133

A 71-year-old man presents to the hospital with a 4-month history of progressive shortness of breath. His wife brought him into the hospital today as he can no longer walk up the stairs without having to take a break. He has a past medical history of hypertension, hypercholesterolaemia and diet-controlled type 2 diabetes.

On examination, he has a heart rate of 101 beats/min with a blood pressure of 140/98mmHg. His oxygen saturations are 90% on air with a respiratory rate of 27 breaths/min. There is reduced air entry throughout the right lung field.

A chest x-ray is requested, as shown below:



What is the most likely diagnosis?

A. Congestive heart failure
B. Mesothelioma
C. Parapneumonic effusion
D. Pulmonary embolism
E. Pulmonary metastases

B. Mesothelioma

This patient has evidence of pleural thickening along the borders of the right lung which is most clear when making a comparison to the left lung field. There is also an associated pleural effusion on the right side with a visible fluid level that abuts the right costophrenic and costocardiac angles. Overall, this significantly reduces the volume of the right lung. This is most likely in keeping with a diagnosis of **mesothelioma** a rare pleural malignancy that is largely associated with asbestos exposure. The age of onset is typically between 60-85 years of age and presents with dyspnoea, cough and chest pain. Chest x-ray findings of mesothelioma include unilateral pleural thickening, reduced lung volume size and lower zone interstitial fibrosis.

Congestive heart failure can present with pleural effusions. However, more common chest x-ray findings include cardiomegaly, pulmonary venous congestion and bilateral pulmonary infiltrates. The unilateral findings on this patient's chest x-ray are more suggestive of an alternative diagnosis.

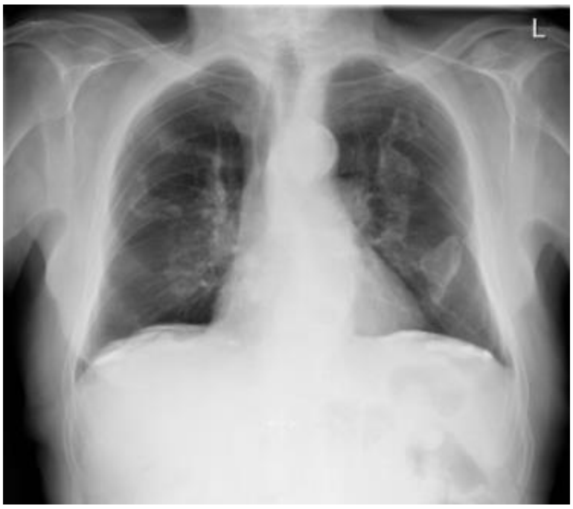
A **parapneumonic effusion** is a complication of viral or bacterial pneumonia when fluid collects in the pleural space due to increased permeability of the visceral pleura. Chest x-ray findings may be similar with signs of pleural effusion and an overall reduced lung volume. However, pleural thickening is not a feature of a parapneumonic effusion.

A **pulmonary embolism** (PE) does not frequently give rise to chest x-ray signs but can cause pleural effusions and Hampton hump sign (a wedge-shaped opacification secondary to lung infarction). However, the insidious onset of symptoms and pleural thickening make PE less likely.

Pulmonary metastases are relatively common but are not usually symptomatic unless advanced. Rather, the patient will experience constitutional symptoms and those of the primary malignancy. Chest x-ray findings of pulmonary metastases usually include multiple round nodules located in the peripheral lung fields. Unilateral pleural thickening with an associated effusion is unlikely to present pulmonary metastases

question 101 of 133

A 76-year-old retired boiler engineer presents with a 3-week history of a cough following a coryzal illness. He was a heavy smoker until around five years ago. An x-ray is performed:



What does the chest x-ray demonstrate?

A. Chronic obstructive pulmonary disease
B. Bronchiectasis
C. Asbestosis
D. Pleural plaques
E. Lung cancer

D. Pleural plaques

The correct answer is **Pleural plaques**. Pleural plaques are areas of fibrous thickening on the pleura (the lining of the lungs) and are often associated with exposure to asbestos. They are usually asymptomatic and do not typically affect lung function. The chest x-ray in the question shows bilateral pleural thickening, which is characteristic of pleural plaques. Their presence can indicate past exposure to asbestos, which would be consistent with the patient's history as a boiler engineer.

The first incorrect option is **Chronic obstructive pulmonary disease (COPD)**. Although the patient has a history of heavy smoking, which is a leading risk factor for COPD, this condition would not typically manifest as pleural plaques on an x-ray. Instead, one might expect to see hyperinflation of the lungs or potentially bullae (areas of emphysematous change), neither of which are evident in this image.

Next, we have **Bronchiectasis**, another incorrect choice. Bronchiectasis refers to permanent enlargement and damage to the airways (bronchi), often caused by recurrent infection or inflammation. On an x-ray, bronchiectasis may present as ring-like or tram-line shadows due to abnormally dilated bronchi; however, these signs are not visible in this case.

Asbestosis is also suggested but it is not correct either. Asbestosis refers specifically to interstitial lung disease caused by inhalation of asbestos fibres. While it shares a common cause with pleural plaques (asbestos exposure), its radiographic appearance differs significantly. Asbestosis typically presents as small irregular opacities predominantly in lower lobes and subpleural areas, rather than the discrete areas of pleural thickening seen here.

Lastly, **Lung cancer** was proposed as an option but it too does not fit with the findings on this xray. Lung cancers can take various forms on imaging - from solitary pulmonary nodules to larger masses - but they generally do not appear as bilateral pleural thickening like that seen in this image.

Question 102 of 133

A 68-year-old gentleman is presenting to the emergency department with a two month history of productive cough. He tells you that over the past week he is getting increasingly worse and feels feverish. He informs you that it is not unusual for him to cough copious amounts, but this time is much worse, and is wondering if he has an infection brewing. He also informs you that he is regularly under the care of the respiratory team, as he is suffering from a lung condition, however he cannot disclose further information. He is a non-smoker.

He appears sweaty and his temperature is 38.2ºC. He has a heart rate of 103/min and his capillary refill time is 3 seconds. Blood pressure is 102/54 mmHg. Examination indicates coarse crackles bilaterally and on the R mid-zone. Heart sounds are normal with no added sounds.

A chest x-ray is performed indicating increasing bronchovascular markings with bilateral consolidation.

You review his medicines and note that he is on salbutamol and ipratropium nebulisers, carbocisteine, aspirin and ramipril. You also notice that he is on ciprofloxacin, and when you enquire further he tells you that he has been on ciprofloxacin for 14 days so far as prescribed by the respiratory team because of an organism isolated in his sputum. You chase that up, and find out that he has previously grown Pseudomonas *aeruginosa*.

What antibiotic would you prescribe to this patient in this setting, given his status as penicillin allergic:

A. Intravenous meropenem
B. Intravenous co-amoxiclav
C. Intravenous tazocin
D. Intravenous metronidazole
E. Nebulised colomycin

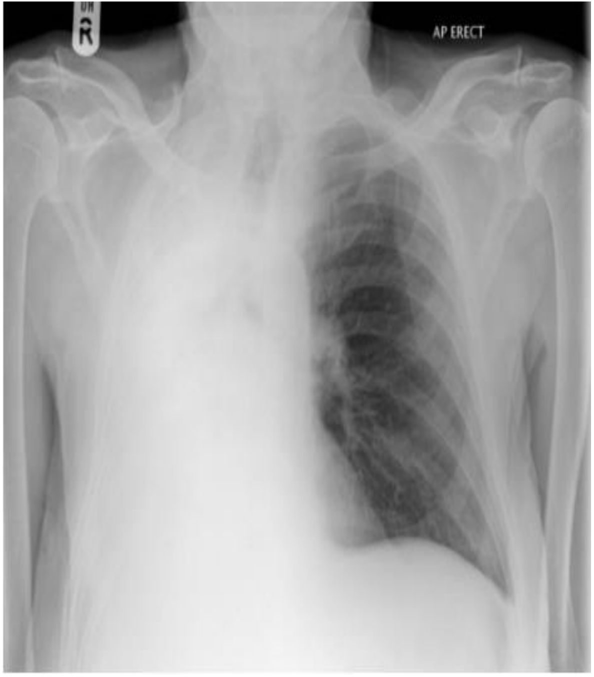
A. Intravenous meropenem

Appropriate treatment of *Pseudomonas* positive bronchiectasis failing to respond to ciprofloxacin includes IV tazocin, ceftazidime, aztreonam or meropenem

It is not uncommon in daily practice to meet patients that are aware of the fact that they suffer from a particular issue (in the case of this patient respiratory) without being able to exactly explain it (bronchiectasis). There are certain clues in this history pointing to a long term condition such as bronchiectasis (absence of smoking, bilateral crackles, copious productive cough, growth of pseudomonas, recent use of ciprofloxacin). As ciprofloxacin orally for 14 days has not worked for this patient the BTS guidelines suggest for the use of IV tazocin, ceftazidime, aztreonam or meropenem - given his penicillin allergic status, the safest choice is meropenem. Nebulised colomycin has a place in the treatment of these patients, not however in the acute setting

Question 103 of 133

An 82-year-old man is admitted with a three day history of increasing breathlessness, His past medical history includes ischaemic heart disease and rheumatoid arthritis. His current medications include aspirin, atorvastatin, bisoprolol, ramipril and methotrexate. On examination his heart rate is 80/min, blood pressure 111/80 mmHg, respiratory rate 24/min and oxygen saturations 89% on room air. There are reduced breath sounds on the right side of his chest. A chest x-ray is ordered:



What is the most likely diagnosis?

A. Mesothelioma
B. Methotrexate-induced pneumonitis
C. Multilobar pneumonia
D. Massive pleural effusion
E. Lung collapse

E. Lung collapse

The key to making the diagnosis, as with all white-outs, is to look at the position of the trachea. In this x-ray it is pulled towards the opacity making the diagnosis of a lung collapse likely.

Question 104 of 133

A 77-year-old active smoker with a 40 pack year history presents with his second non-infective exacerbation of COPD in 3 months. He was diagnosed with COPD two years ago and had been relatively well controlled using salbutamol as required prior to these two admissions. Two days after his admission, he reports that he is close to his baseline and would like to go home. His repeat pulmonary function tests reveal a forced expiratory volume in 1 second of 46%.

On reviewing his peak flow diary you note a significant (> 20%) diurnal variation in his peak flow.

Which would be most useful in optimising his COPD control?

A. No change to inhaler therapy, follow up in 3 months
B. Add beclometasone inhaler alone
C. Add salmeterol and fluticasone combination inhaler
D. Add salmeterol inhaler alone
E. Long term oxygen therapy

C. Add salmeterol and fluticasone combination inhaler

COPD - still breathless despite using SABA/SAMA and **asthma/steroid responsive features** → add a LABA + ICS

As this patient has a feature suggestive of asthma/steroid responsiveness (significant diurnal variation) the next step in management is a long-acting beta agonist (e.g. salmeterol) + an inhaled corticosteroid. NICE now recommend the use of combined inhalers where possible.

Question 105 of 133

A 46-year-old gentleman was referred by his GP to the respiratory clinic with deteriorating asthma control. He was diagnosed by his GP 2 years ago following complaints of feeling short of breath on exertion with wheeziness. Unfortunately, his symptoms deteriorated despite a trial of Clenil modulate 1000mcg BD, salmeterol 200mcg BD and montelukast 10mg OD. Upon further questioning, he stated that he had pins and needles in both hands as well as numbness of his fingertips for the last few weeks. His past medical history included hypertension, gout and sinusitis for which he was prescribed ramipril 5mg OD, allopurinol 100mg OD, salbutamol PRN and mometasone nasal spray 50mcg BD. He smoked 20 cigarettes per day and consumed 12 units of alcohol per week.

On examination, he appeared unwell. His heart rate was 98 bpm, respiratory rate 18/min, oxygen saturations 94% on air and blood pressure 146/82 mmHg. His heart sounds were normal with a normal JVP and examination of the chest revealed bilateral expiratory wheezes globally. Examination of the abdomen was normal. Examination of the peripheral nervous system demonstrated a peripheral neuropathy with a glove and stocking sensory loss of the upper limbs. Examination of the skin revealed the presence of purpura on his lower legs.

Initial investigations revealed the following results:

Hb	102 g/l
Platelets	342 * 10 /l9
WBC	12.1 * 10 /l9
Neutrophils 82% count	
Lymphocytes 7% count	
Eosinophils 10% count	
Monocytes 1% count	

ESR	42 mm/hr
Na +	141 mmol/l
K +	3.9 mmol/l
Urea	6.2 mmol/l
Creatinine	78 µmol/l

Chest x-ray: bilateral scattered pulmonary shadowing in lower zones

ECG: heart rate 97bpm, normal sinus rhythm, QRS interval 112ms, QTc interval 424ms

Which of the following tests is most likely to lead to the underlying diagnosis?

A. Anti glomerular basement membrane antibodies
B. p-ANCA antibodies
C. ANA antibodies
D. Anti Jo-1 antibodies
E. c-ANCA antibodies

B. p-ANCA antibodies

Churg-Strauss syndrome - positive pANCA serology

This gentleman presents with a combination of worsening asthma in combination with sinusitis. He has pulmonary infiltrates on his chest x-ray, as well as polyneuropathy and an eosinophilia. The diagnosis is strongly suggestive of Churg-Strauss syndrome, and of the following options, p-ANCA antibodies are the most sensitive and specific to this condition

Question 106 of 133

A 62-year-old gentleman is seen at the respiratory clinic after a mass lesion is identified by his GP on chest x-ray. He is a heavy smoker and has had a cough and weight loss for the past two months. On further questioning, he admits to feeling very weak and is having difficulty getting out of a chair. On examination, the consultant notices some proximal muscle wasting. Initial investigations reveal a blood pressure of 170/90mmHg and a low potassium. In addition to organising staging CT and bronchoscopy, he requests a 24-hour urinary cortisol test, which is raised.

Which of the following is the most likely underlying pathology?

A. Squamous cell bronchial carcinoma
B. Large cell bronchial carcinoma
C. Carcinoid tumour
D. Small cell bronchial carcinoma
E. Bronchial adenocarcinoma

D. Small cell bronchial carcinoma

The 24-hour urinary cortisol result here suggests a diagnosis of Cushing's syndrome. The coexistant presentation of lung cancer points to paraneoplastic Cushing's syndrome. The vast majority of paraneoplastic syndromes seen in lung cancer are caused by small cell bronchial carcinoma, a neuroendocrine tumour which can secrete a variety of hormones or antibodies. In paraneoplastic Cushing's syndrome, ectopic adrenocorticotrophic hormone (ACTH) production results in a syndrome characterised by muscle weakness, hypertension, hypokalaemia and oedema. The classical features of buffalo hump and moon face are often absent.

Question 107 of 133

A 29-year-old man presents to his GP with shortness of breath. This has been occurring for the past 6 months and is sometimes accompanied by wheezing, particularly later on during the day. He does not have any past medical history and occasionally takes over-the-counter antihistamines for hay-fever. His only recent travel history is a holiday to Greece, where he found he was less short of breath and he was less wheezy. His only family history of note is his mother who died of a heart attack aged 65. He works as a car mechanic and smokes 5 roll-up cigarettes per day and drinks on average 5-6 units of alcohol per week.

Examination of the chest is normal with good air entry bilaterally and no added sounds. Heart sounds I and II are present with no obvious murmurs.

What is the most useful investigation in determining this man's diagnosis?

A. Chest x-ray
B. Peak expiratory flow rate measurements at both home and at work
C. Pulmonary function testing
D. Serum IgE measurement
E. Skin prick testing

B. Peak expiratory flow rate measurements at both home and at work

The most likely diagnosis in this case is occupational asthma, as demonstrated by the timing of his breathlessness and wheeze as well as the improvement in symptoms when on holiday. The diagnosis of occupational asthma is best determined using peak expiratory flow rate measurements taken at home and work to determine the difference in the two - following this further investigation scan be performed to determine the allergen causing the occupational asthma

Question 108 of 133

A 55-year-old woman with poorly-controlled type 2 diabetes and a 50-pack-year smoking history presents to the Emergency Department with a three-day history of fever and productive cough. A chest radiograph shows right middle lobe pneumonia. She is prescribed a course of oral antibiotics and is discharged home.

She is reviewed in respiratory clinic two months later and has an ongoing dry cough.

What single investigation would be most appropriate to request during her consultation?

A. Arterial blood gas measurement
B. Pulmonary function tests
C. Referral for CT thorax
D. Repeat chest radiograph
E. Sputum culture

D. Repeat chest radiograph

All cases of pneumonia should have a repeat chest X-ray at 6 weeks after clinical resolution

This patient had lobar pneumonia and **a repeat chest radiograph should be arranged after about six weeks** for all patients with persistent symptoms, physical signs or at higher risk of malignancy such as smokers or older adults aged over 50 years old. Further investigation may be offered depending on their symptoms and radiology to rule out the presence of other conditions including malignancy. Although this patient does have a persistent cough, patients may take three to six months to feel back to normal and it would be most appropriate to request a chest radiograph first before any other radiological tests.

Arterial blood gas measurement is useful in patients with chronic lung disease to determine if they have chronic hypoxaemia. Such patients may be offered long-term oxygen therapy (LTOT) to improve survival. However, this patient does not have a diagnosis of chronic lung disease. Furthermore, if she continues smoking, it would not be appropriate to offer oxygen therapy as it is a fire hazard.

Pulmonary function tests such as spirometry should be offered to patients who smoke who have symptoms such as breathlessness, chronic cough, significant sputum production, wheeze and frequent chest infections. However, it would not be as important as ensuring her pneumonia has resolved or that lung cancer is not missed.

A **CT thorax** would be appropriate in patients with persistent changes on a repeat chest radiograph, or those with a normal chest radiograph but persistent symptoms. However, a chest radiograph should be repeated before a CT due to the high radiation dose involved.

Sputum culture may be helpful if the patient remains productive or if her symptoms fail to resolve with initial antibiotic therapy as she may have an atypical organism causing her chest infection, such as *Pseudomonas*. However, given that her cough is now dry this is unlikely to be feasible. A repeat chest radiograph would be more helpful in determining the possible causes of her symptoms in the first instance.

Question 109 of 133

A 45-year-old is referred to a respiratory clinic with 2 months of exertional shortness of breath. He has a past medical history of HIV. He is well controlled on Truvada. He has had an unprovoked pulmonary embolism three years ago and was treated with six months of warfarin. He smokes 15 cigarettes daily for the last 20 years. He recently completed his first mountaineering expedition and successfully reached the summit of Kilimanjaro.

On examination, there is a loud P2. The jugular venous pulse is raised and there is peripheral oedema. Chest auscultation is unremarkable and there are no murmurs.

A transthoracic echocardiogram is arranged, which demonstrates a mean pulmonary arterial pressure (PAPm) of 38 mmHg and mitral regurgitation with a regurgitant fraction of 14%.

An HRCT chest is normal. V/Q scanning demonstrates mismatched perfusion defects. The patient is referred for a right heart catheter, which confirms a PAPm of 38mmHg and also demonstrates a pulmonary arterial wedge pressure (PAWP) of 11 mmHg.

Given the likely diagnosis, what is the probable underlying aetiology?

A. COPD
B. Chronic thromboembolic disease
C. HIV
D. High altitude exposure
E. Mitral regurgitation

B. Chronic thromboembolic disease

Chronic pulmonary embolisms can lead to pulmonary hypertension

Chronic thromboembolic disease is the correct answer. The patient has pulmonary hypertension as evidenced by the clinical features and right heart catheter results (PAPm > =25mmHg). The patient has a history of pulmonary embolism and has been appropriately treated with warfarin for six months given his unprovoked VTE. However, the V/Q scan demonstrates mismatched perfusion defects suggesting chronic thromboembolic disease. This is therefore the most likely cause of pulmonary hypertension in this case.

COPD is incorrect. This is a cause of pulmonary hypertension. However, this is only seen in severe COPD. In a patient who has recently scaled a mountain and has a normal HRCT chest, COPD is unlikely.

HIV is incorrect. This is a cause of pulmonary hypertension. However, given he is well controlled on Truvada, this is slightly less likely in general. Additionally, it would not explain the mismatched perfusion defects on V/Q scanning and therefore it is a less likely cause than a chronic thromboembolic disease.

High altitude exposure is incorrect. He has only completed one mountaineering expedition. High altitude exposure can cause pulmonary hypertension but this is typically in people chronically exposed, living at high altitudes.

Mitral regurgitation (MR) is incorrect. The patient does not have clinically severe mitral regurgitation and the echocardiography findings are that of mild MR (regurgitant fraction < 30%). Therefore this is not the likely cause of pulmonary hypertension

Question 110 of 133

A 62-year-old man, with a 40-year heavy smoking history, presents to the emergency department. He's been troubled by a cough for the last five months associated with constant fatigue and weight loss. He reports coughing up blood on the morning of presentation.

On physical examination, the patient looks emaciated with notable finger clubbing. Auscultation reveals diminished air entry over the left lung with occasional wheezes. No murmurs are heard.

His vital signs are as follows:

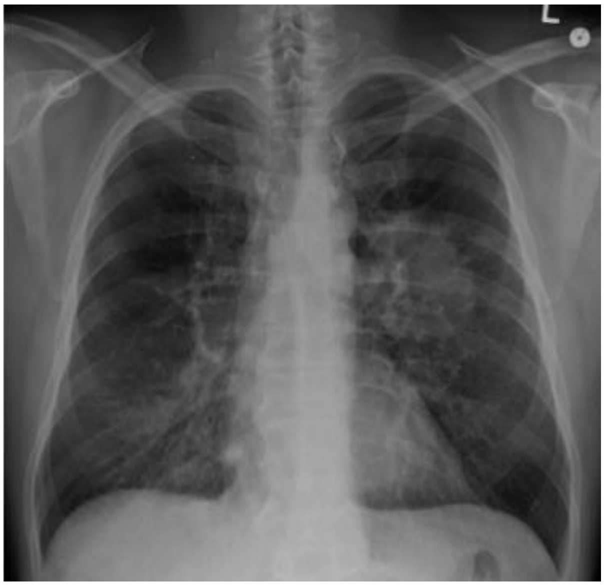
Temperature: 37.1°C.

Blood pressure: 115/75 mmHg. Heart rate: 101 beats/min, regular. Respiratory rate: 22 breaths/min. Oxygen saturation: 94 % on room air.

Labs are ordered and the results are the following:

Hb	125 g/L	Male: (135-180)
Platelets	680 * 10 /L	(150 - 400)
WBC	9.5 * 10 /L	(4.0 - 11.0)
Calcium	2.75 mmol/L	(2.1-2.6)

The chest x-ray is shown:



What is the most likely diagnosis?

A. Lung metastases
B. Sarcoidosis
C. Small cell lung cancer
D. Squamous cell lung cancer
E. Tuberculosis

D. Squamous cell lung cancer

Squamous cell lung cancer is the correct answer.

The patient's chest x-ray reveals a concerning mass in the left lung's hilar region. Considering the patient's extensive history of smoking coupled with respiratory symptoms, including haemoptysis, thrombocytosis, and the presence of hypercalcemia, a primary lung malignancy is the leading diagnostic consideration. While both small cell and squamous cell lung cancers could be potential culprits, hypercalcemia, which may be attributed to paraneoplastic secretion of parathyroid hormone-related protein, is more commonly associated with squamous cell lung cancer.

Lung metastases is not correct. While the x-ray findings and symptoms might suggest some form of lung disease, the patient's significant smoking history combined with hypercalcemia lean more towards a primary lung malignancy rather than metastatic disease.

Sarcoidosis is a viable differential diagnosis. The hypercalcemia and thrombocytosis associated with sarcoidosis make it a potential consideration. However, sarcoidosis typically presents with bilateral hilar lymphadenopathy. The unilateral presentation in this case, combined with the presence of haemoptysis, makes sarcoidosis less likely.

Small cell lung cancer is less likely. Although the patient's symptoms could be consistent with small cell lung cancer, the presence of hypercalcemia makes squamous cell lung cancer more likely.

Tuberculosis seems improbable in this case. While TB can indeed cause haemoptysis, chronic cough, weight loss, and hilar lymphadenopathy on imaging, other typical symptoms such as fever and night sweats are absent. The combination of a hilar mass and thrombocytosis with the patient's smoking history places lung cancer higher on the differential list.

Question 1 of 23

A 30-year-old man presents to the emergency department with a four-day history of subjective fevers, shortness of breath, non-productive cough and malaise.

On arrival to the emergency department, his observations revealed a temperature of 38.5ºC, heart rate 120/min, blood pressure 90/60 mmHg, respiratory rate 35/min and oxygen saturations of 80% on room air. An arterial blood gas revealed severe type 1 respiratory failure and the patient was intubated and placed on mechanical ventilation.

A post-intubation chest x-ray revealed bilateral pulmonary infiltrates and within the limits of a portable chest x-ray, a normal sized cardiac silhouette.

Intravenous 1g ceftriaxone, 500mg azithromycin and 200mg hydrocortisone were given in the emergency department for severe community acquired pneumonia prior to transfer to the intensive care unit (ICU).

The patient remained on invasive mechanical ventilation for the first 24 hours. During this time, he underwent computed tomography pulmonary angiography (CTPA) and bronchoalveolar lavage.

He made good progress and was extubated to high flow nasal cannulae after 24 hours. He is haemodynamically stable. He is stepped down to the high dependency unit (HDU), where you review the patient.

You speak with the patient's wife to obtain a collateral history. The patient has no known past medical, surgical, drug or family history of note. He works as a full-time chef and there have been no known sick contacts. He drinks 1-2 cans of standard strength beer per night and occasionally binge drinks at the weekends. He recently started smoking cigarettes to cope with increased stress at work and there is no history of illicit drug use. There has been no recent travel history.

On examination in the HDU, the temperature was 37.5ºC, heart rate 90/min, blood pressure 100/60 mmHg and respiratory rate 26/min. He is on high flow oxygen via nasal cannulae, with a flow rate of 30 litres per minute, the fraction of inspired oxygen is 30% and has oxygen saturations of 95%. There are fine inspiratory crepitations audible in the lower posterior chest bilaterally which do not change with coughing. The jugular venous pressure was not elevated and there was no pedal oedema.

Current medications are: intravenous ceftriaxone, azithromycin and as required paracetamol You review the latest available results for this patient:

WBC	11 * 10 /l 9	Urea	7 mmol/l
Neuts	8.5 * 10 /l 9	Creatinine	80 µmol/l
Lymphs	1.5 * 10 /l 9	CRP	80 mg/l
Eosin	1.0 * 10 /l9		

Bronchoalveolar lavage	Result
Neutrophils	5%
Lymphocytes	10%
Eosinophils	30%
Respiratory viruses PCR	Negative
Mycoplasma *pneumoniae* PCR	Negative
Microscopy, culture and sensitivities	Nil growth

Imaging	Report

CT pulmonary angiogram	No pulmonary embolism. Bilateral patchy areas of ground-glass opacities and small bilateral pleural effusions

Which of the following would be the most appropriate ongoing management for this patient?

A. Continue intravenous ceftriaxone and azithromycin
B. Cease intravenous ceftriaxone, azithromycin and observe
C. Cease intravenous ceftriaxone and azithromycin. Commence intravenous furosemide40mg twice daily
D. Continue intravenous ceftriaxone, azithromycin and commence intravenoushydrocortisone
E. Cease intravenous ceftriaxone, azithromycin and commence intravenous hydrocortisone

E. Cease intravenous ceftriaxone, azithromycin and commence intravenous hydrocortisone

Acute eosinophilic pneumonia is highly responsive to steroids

This man presents with clinical and laboratory features consistent with acute eosinophilic pneumonia, which is highly responsive to steroids.

Antibiotics can be ceased in this case for a number of reasons:

- There is no evidence of consolidation on the CTPA
- With bacterial pneumonia, you normally expect a neutrophil pleocytosis on the bronchoalveolar lavage fluid analysis

- Patients with bacterial pneumonia requiring intubation and ventilation rarely improve as rapidly as described in this case, which should make you question the diagnosis community acquired pneumonia.

The modified Philit criteria are used to diagnose acute eosinophilic pneumonia

- acute respiratory illness of less than or equal to 1-month duration
- pulmonary infiltrates on chest radiography or computed tomography (CT)
- pulmonary eosinophilia as demonstrated by more than 25% eosinophils in BAL fluid (can be accompanied by variably increased percentages of lymphocytes and neutrophils) or eosinophilic pneumonia on lung biopsy (bronchoscopic or surgical), and
- the absence of other specific pulmonary eosinophilic diseases e.g. eosinophilic granulomatosis with polyangiitis (previously known as Churg-Strauss syndrome)

Acute eosinophilic pneumonia can be caused by inhalation of chemicals, with tobacco being most frequently associated with developing the condition, especially those who have recently started smoking, as in this case.

There can be a spectrum of presentations, from mild symptoms that resolve spontaneously, to a clinical picture mimics acute respiratory distress syndrome (ARDS) and can be misdiagnosed as community acquired pneumonia.

The BAL is a key investigation. A marked eosinophilia (>25%) which may be accompanied by elevated neutrophils and lymphocytes, suggests an acute eosinophilic pneumonia. Whereas in ARDS, the BAL analysis is expected to show a neutrophilia.

Acute eosinophilic pneumonia responds within days with appropriate treatment but it can be fatal if not identified.

This patient received 200mg intravenous hydrocortisone in the emergency, most likely because the suspicion was severe community acquired pneumonia and you should be aware that there is a growing evidence base for the use of steroids for severe community acquired pneumonia. The hydrocortisone, albeit inadvertently, may have contributed to this patient's rapid improvement and extubation.

There is no consensus on the dose and duration of corticosteroid therapy for such patients. Some articles suggest methylprednisolone for those critically unwell, with a transition to oral prednisolone for up to 4 weeks once stabilised.

Option 1 - incorrect as this would treat bacterial pneumonia and no an acute eosinophilic pneumonia

Option 2 - stopping antibiotics is a correct management step but observing only would likely lead to this patient relapsing after only having one dose of corticosteroids

Option 3 - stopping antibiotics is a correct management step but there is no evidence of heart failure/fluid overload on examination. Acute eosinophilic pneumonia is frequently associated with pleural effusions, which may be unilateral or bilateral.

Option 4 - there is no indication for ongoing antibiotics

Option 5 - the correct answer

Question 2 of 23

A 59-year-old man is seen in the emergency department with abdominal pain. He is a type 2 diabetic with a body mass index of 40kg/m² and takes regular metformin and glibenclamide. He has previous episodes of pancreatitis and he says the pain is central and radiating to his back like on previous occasions. He has never smoked and worked as a loans manager for 38 years before taking early retirement.

On examination, he has a clear chest and respiratory rate is 15/min. His abdomen has guarding in the epigastric areas but bowel sounds are present. The surgical registrar asks you to perform an arterial blood gas and the results are as below.

pH	7.38
pCO2	7.8 kPa
pO2	8.4 kPa
HCO3	31 mmol/L
Base excess	+4.2
Glucose	5.3 mmol/L
Lactate	2.50mmol/L

Please explain what the likely cause of these findings are?

A. Obstructive sleep apnoea (OSA)
B. Chronic obstructive pulmonary disease (COPD)
C. Alpha-1-antitrypsin deficiency
D. Lactic acidosis
E. Sepsis

A. Obstructive sleep apnoea (OSA)

Untreated OSA -> compensated respiratory acidosis

This gentleman has a raised CO2 but a normal pH therefore this is a compensated respiratory acidosis. His lactate is raised but there is no acidosis so this is not lactic acidosis. COPD patients by definition almost always have some smoking history. Furthermore, he has no lung or liver pathology so alpha-1-antitrypsin deficiency is also unlikely. Patients who are obese often have lots of soft tissue in the neck that occlude the airway when asleep and the tone falls. Therefore, the carbon dioxide rises during sleep and they have daytime somnolence and headaches. Over long periods of time, the carbon dioxide can become compensated by renal retention of bicarbonate and in an analogous mechanism to COPD, patients with chronic OSA can become carbon dioxide retainers with normal pH. This gentleman would probably benefit from home CPAP after this result is repeated 6 weeks after discharge from hospital

Question 3 of 23

A 31-year-old male presented to the emergency department with a 1-day history of dyspnoea associated with a dry cough and palpitations.

On examination, his temperature is 37.3ºC. He has a respiratory rate of 28 breaths per minute, with an oxygen saturation of 94% on room air. His blood pressure is 125/80 mmHg with a heart rate of 100bpm. On auscultation, there is reduced air entry in the right lower zone and a chest radiograph shows a right pneumothorax. A thoracostomy tube is placed on the right side immediately.

During the morning ward round, 4 days later, it is noticed that whilst checking the chest drain there is an air leak of 50cc. A repeat chest radiograph shows the right pneumothorax is still present.

What is the next most appropriate step in this patient's management?

A. Apply supplemental oxygen therapy
B. Change the thoracostomy tube to a large bore catheter
C. Thoracic surgery consultation
D. Apply suction at -20 cmH2O using a high pressure-low volume system
E. Observe for a further 48 hours

C. Thoracic surgery consultation

When draining a pneumothorax, if after 3-5 days there is persistent air leak (e.g. bubbling chest drain) or failure of the lung to re-expand, a thoracic surgical opinion should be sought

All patients with a primary spontaneous pneumothorax (PSP) should receive resuscitation with a focus on airway stabilisation (if indicated) as well as supplemental oxygen to treat hypoxaemia (if present) and to facilitate absorption of air from the pleural space. Subsequent management is directed at deciding whether air needs to be removed from the pleural space. If so, then options include observation with or without supplemental oxygen, needle aspiration of intrapleural air, and chest tube or catheter thoracostomy.

An air leak is considered prolonged if it continues over the next three to seven days. One should check that the leak is not coming from the drainage chamber, from the tubing, or from around the chest tube at the skin entry site; this is classically done by clamping the tube at different locations to isolate the site of the leak. A thoracic surgery consultation is necessary for patients with a persistent air leak. These patients require a more aggressive approach for closure since the longer an air leak persists, the less likely it is that the leak will close spontaneously requiring a definitive intervention (e.g. pleurectomy, blebectomy and/or pleurodesis).

Applying supplemental oxygen is of no benefit here as the patient is currently saturating well. Observation for a further 48 hours is not indicated in patients with a persistent air leak.

Applying suction will not be the right answer here as the thoracostomy tube is already on suction once inserted.

Changing the tube will not be of benefit as it has been inserted 4 days ago with a persistent air leak and without any changes in the chest radiograph or clinical examination

Question 4 of 23

A 66-year-old gentleman with a diagnosis of chronic obstructive pulmonary disease (COPD) is reviewed in clinic. He complains of persistent breathlessness on exertion. There is no significant history of cough.

Pulmonary function testing reveals:

SpO2	90%
FVC	2.8L
FEV1	1.47 (40% predicted)
FEV1/FVC ratio	53%

Looking back at his notes you note that significant (> 20%) diurnal variation has been noted in his peak flows.

The patient is currently prescribed a short-acting beta-2 agonist along with a combination inhaler containing a long-acting beta-2 agonist and corticosteroid.

What is the most appropriate additional medication?

A. Inhaled tiotropium
B. Oral modified-release aminophylline
C. Short-acting muscarinic agonist
D. Oral prednisolone
E. Oral roflumilast

A. Inhaled tiotropium

COPD - still breathless despite using SABA/SAMA and a LABA + ICS → add a LAMA

The correct answer in this case is **inhaled tiotropium**. Tiotropium is a long-acting muscarinic antagonist (LAMA) that is commonly used as an additional bronchodilator in the management of COPD. According to the National Institute for Health and Care Excellence (NICE) guidelines, adding a LAMA such as tiotropium is recommended when patients with COPD continue to experience breathlessness or exacerbations despite treatment with a long-acting beta-2 agonist (LABA) and inhaled corticosteroid (ICS).

Oral modified-release aminophylline is not the most appropriate choice because it is a methylxanthine bronchodilator that has a narrow therapeutic index and requires regular monitoring of serum levels. It is generally reserved for cases where other treatments have failed or are contraindicated.

Short-acting muscarinic agonist would be an incorrect choice because these medications are not used in the treatment of COPD. In fact, short-acting muscarinic antagonists (SAMA), such as ipratropium, may be used as bronchodilators in COPD but are not the most appropriate option here given that the patient already has LABA/ICS therapy and adding a LAMA like tiotropium would provide better symptom control.

Oral prednisolone would not be the best option in this scenario, as it is typically used for acute exacerbations of COPD rather than as part of routine maintenance therapy. Long-term oral corticosteroid use has significant side effects and should be avoided unless absolutely necessary.

Finally, **oral roflumilast** could be considered for patients with severe COPD who have chronic bronchitis symptoms and frequent exacerbations, but it is not the first choice for managing persistent breathlessness in this patient. Adding a LAMA like tiotropium would be more appropriate according to the NICE guidelines.

In summary, the most appropriate additional medication for this patient with COPD and persistent breathlessness despite LABA/ICS therapy is inhaled tiotropium.

Question 5 of 23

A 25-year-old woman presents with a 2 month history of increasing shortness of breath and feeling that he chest is tight. This has built up gradually with no obvious cause and it has reached the point where she struggles to breathe when climbing a flight of stairs. She has occasional episodes of sweating profusely at night with her having to change pillows during the night,

She has a dry cough and a past medical history only significant for recurrent UTIs. She takes the combined oral contraceptive pill and prophylactic nitrofurantoin. She does not smoke, keeps a pet dog and was abroad in the USA 6 months ago.

On examination, her saturations are 94% in air and she is tachypnoeic as she walks down the corridor. Her chest has a mixture of crepitations and wheeze. There is no JVP and no murmurs. She has some red scaly areas across the back of her hands. There is no clubbing and her current peak flow is 320.

Hb	140 g/l	Na +	139 mmol/l
Platelets	397 * 10 /l 9	K +	3.9 mmol/l
WBC	6.2 * 10 /l 9	Urea	5.6 mmol/l
Neuts	3.4 * 10 /l 9	Creatinine	89 µmol/l
Lymphs	0.3 * 10 /l 9	CRP	32 mg/l
Eosin	1.5 * 10 /l9		

Chest x-ray	bilateral patchy shadows in the mid zones
High resolution CT scan	bilateral patchy areas of ground-glass opacity and interlobular septal thickening
Bronchoalveolar lavage	raised leucocytes with eosinophil predominance

What is the likely diagnosis?

A. Eosinophilic polyangiitis with granulomatosis
B. Eosinophilic asthma
C. Pulmonary eosinophilia
D. Idiopathic pulmonary fibrosis
E. Bronchiectasis

C. Pulmonary eosinophilia

Nitrofurantoin causes a pulmonary eosinophilia

This lady has a reduced peak flow, shortness of breath and wheeze. This immediately excludes pulmonary fibrosis which is a restrictive airway disease and peak flow is relatively preserved. Asthma would not have ground glass changes. Bronchiectasis would have dilated airspaces on CT that are not seen here. Eosinophilic polyangiitis with granulomatosis will have renal involvement also. Nitrofurantoin is a recognised cause of pulmonary eosinophilia and should be stopped.

Question 6 of 23

A 64-year-old woman was brought in by ambulance following an out-of-hospital cardiac arrest. Return of spontaneous circulation was achieved with 2 cycles of resuscitation. She has spent the following 3 days in the intensive care unit, intubated and ventilated.

On the morning round, it is noted that she is requiring a higher fraction of inspired oxygen to maintain normal saturations and is bringing up copious amounts of secretions. A sample of sputum is sent for microscopy, culture, and sensitivity (MC&S). These results are shown below:

Sputum MC&S	Bacteroides fragilis

What is the most likely explanation for these results?

A. Aspiration pneumonia
B. Contaminated sample
C. Hospital-acquired pneumonia
D. Seeding of bacteraemia
E. Ventilator-associated pneumonia

A. Aspiration pneumonia

Anaerobic bacteria in sputum culture suggest aspiration pneumonia

This woman is suffering from **aspiration pneumonia**, likely secondary to her cardiac arrest. Not only does this fit the clinical history, but the anaerobic growth (*Bacteroides fragilis*) on MC&S provides further evidence that this is the case. This is therefore the most likely answer.

A **contaminated sample** may be suspected if the MC&S grows a bacteria that does not fit the clinical picture, however, *Bacteroides fragilis* is rarely a contaminant and its presence is consistent with the clinical picture here. The most common contaminants are coagulase-negative staphylococci species in blood cultures.

A **hospital-acquired pneumonia** could explain this clinical picture however the distinguishing feature is the sputum MC&S. *Bacteroides fragilis* is an anaerobe associated with aspiration pneumonia, making hospital-acquired pneumonia less likely.

Secondary seeding of a *Bacteroides fragilis* bacteraemia is less likely than aspiration in this instance given the clinical history of cardiac arrest and no mention of further systemic features on infection. Further blood cultures to investigate this possibility would be a reasonable further step to take.

As with hospital-acquired pneumonia, **ventilator-associated pneumonia** is less likely than aspiration pneumonia here due to the species grown. *Bacteroides fragilis* is an anaerobe associated with aspiration pneumonia.

Question 7 of 23

A 71-year-old retired bus driver is referred to the respiratory fast-track clinic. He saw his GP after 2 months of right-sided chest pain and cough. He received a course of amoxicillin with some improvement in the cough and chest pain but has since started getting short of breath when just walking around the house. He is an ex-smoker of 30 pack-years.

On assessment, he has dullness to percussion and reduced air entry on the right lung base. His saturations are 96% on air. Chest x-ray confirms a right pleural effusion. Pleural ultrasound shows a moderate sized simple effusion. A pleural aspirate is performed:

Appearance	Serosanguineous
pH	7.32
Protein	45 g/l
LDH	450 IU/l
Glucose	4.0mmol/l
Gram stain	No organisms or malignant cells seen
Culture	No growth

Given the clinical scenario and results so far, what is the next best step for investigation of this patient?

A. Surgical VATS for pleural biopsy
B. CT thorax with contrast
C. Further course of antibiotics
D. Intercostal drain
E. Observe and repeat chest x-ray and pleural ultrasound in 4 weeks

B. CT thorax with contrast

This patient has been investigated according to the algorithm supplied in the British Thoracic Society guidelines 2010: Investigation of a unilateral pleural effusion in adults. He has a unilateral pleural effusion that is an exudate. Investigations have yielded neither a diagnosis nor an indication for chest drain(purulent fluid, pH<7.2, organisms on Gram stain or culture). There is no clear evidence of infection and malignancy needs to be excluded. The next step in the algorithm is CT with contrast. Pleural biopsy may be required after that

Question 8 of 23

A 45-year-old patient undergoing R-CHOP (Rituximab, Cyclophosphamide, Doxyrubicin, Vincristine and Prednisolone) chemotherapy for Non-Hodgkins Lymphoma presents with a progressive cough, rigors and fevers up to 39.4 degrees on his home thermometer. His GP had tried 2 courses of antibiotics that had no beneficial effect on his symptoms. He attended to the Medical Assessment Unit after a particularly bad episode of coughing having experienced blood stained sputum.

Investigations are as follows

Hb	10.4 g/dl
Platelets	460 * 10 /l9
WBC	16.4 * 10 /l9
Neutrophils	13.5 * 10 /l9
Lymphocytes	2.7 * 10 /l9
Na +	134 mmol/l
K +	4.2 mmol/l
Urea	7.4 mmol/l
Creatinine	106 µmol/l

Chest X-Ray

- Cavitating lesion in the right upper zone.
- No evidence of pleural effusion
- No other focal consolidation

CT Thorax

- Cavitating lesion with halo sign

Broncho-alveolar lavage induced sputum

- Hyphae seen on silver staining

What is the most likely diagnosis?

A. Invasive Aspergillosis
B. Allergic Bronchopulmonary Aspergillosis
C. Aspergilloma
D. Tuberculosis
E. Post atypical pneumonia lung abscess

A. Invasive Aspergillosis

This patient is likely to have had a prolonged period of neutropenia post chemotherapy that has left him at risk from fungal infections. Fungal infections are the preserve of the immunosuppressed. They should be part of a differential diagnosis of patients undergoing chemotherapy, stem cell transplant, patients on immunosuppressive medications for autoimmune conditions and those that are HIV positive.

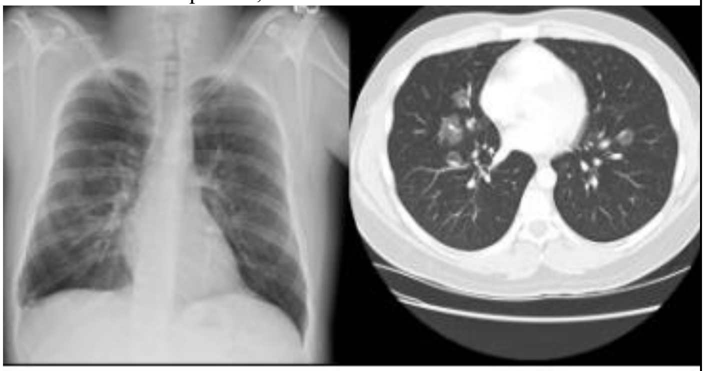
The patient above describes symptoms of active infection and also has the worrying symptom of haemoptysis. Investigations have revealed the classical signs on CT scanning the 'halo sign' and hyphae have been seen on silver staining. Specific blood tests for aspergillous can be done in some centres although the reliability of these are variable. The mortality for invasive aspergillosis vary from 40-90% in the literature. As such it is important to identify it and treat quickly. Fluconazole is of no benefit in these patients. The first line treatment is voriconazole which should be started intravenously before oral dosing is considered. This is due to oral voriconazole taking at least 10 days to get to therapeutic levels. If voriconazole is not tolerated, then lipsomal amphotericin should be used.

Of the other options listed above, aspergilloma may be asymptomatic and only be noted incidentally when scanning for other conditions is undertaken. It can form in cavities left behind from treated TB. Allergic bronchopulmonary Aspergillosis (ABPA) presents commonly in patients with asthma or cystic fibrosis. Typically ABPA presents like poorly controlled asthma, with wheeze and exercise intolerance. It can progress to a bronchecstasis type illness with or without chronic sputum production.

Question 9 of 23

A 55-year-old man presents to the emergency department with a 2-day history of haemoptysis. In the past 3-4 weeks he also reports worsening shortness of breath with mild central chest pain. His past medical history includes hypertension for which he takes amlodipine 5mg daily.

On examination, his saturations are 90% on air. His heart rate is 72/min and regular with a blood pressure of 115/85mmHg. There is reduced air entry at the right base. During the examination, he coughs up approximately 100mls of fresh blood. Imaging of his chest is requested, as shown below.



What is the most likely diagnosis?

A. Eosinophilic granulomatosis with polyangiitis
B. Granulomatosis with polyangiitis
C. Pulmonary embolism
D. Rheumatoid arthritis
E. Sarcoidosis

B. Granulomatosis with polyangiitis

This patient's chest x-ray and CT scan show multiple small nodules throughout the lung fields. Additionally, on the CT scan, there are a few well-defined regions of airspace opacification (as demonstrated by the grey-coloured, round lung markings) which can represent consolidation or pulmonary haemorrhage. In keeping with the history of haemoptysis and shortness of breath, pulmonary haemorrhage is a likely cause of this finding. Further inspection of the chest x-ray hows blunting of the right costophrenic angle demonstrating a likely pleural effusion. Granulomatosis with polyangiitis (GPA) is a type of small vessel vasculitis that is responsible for causing respiratory symptoms such as sinusitis, nasal crusting, shortness of breath and pulmonary haemorrhage. The disease can also cause multiple nodules (with or without cavitation) to arise in the lung parenchyma similarly to that seen in this patient. Pleural effusions are seen in up to 20% of chest x-rays of GPA patients. The disease can initially manifest around middle-age.

Eosinophilic granulomatosis with polyangiitis also causes respiratory symptoms. Pulmonary nodules and pleural effusions may also be seen on imaging, however, pulmonary haemorrhage is not a typical symptom. Instead, wheeze and sinusitis tend to feature.

Pulmonary emboli are capable of causing shortness of breath, hypoxia and haemoptysis (as seen in this patient). They are diagnosed using a CT pulmonary angiogram to assess for filling defects in the pulmonary arteries. Normally, chest x-ray appearances are completely normal. However, in some cases (and also on CT imaging), there is evidence of pulmonary infarction. Pulmonary infarctions create wedge-shaped opacifications without the presence of air bronchograms. Instead, this patient's imaging shows multiple lung nodules, pleural effusion and airspace opacification suggestive of pulmonary haemorrhage.

Rheumatoid arthritis has multiple respiratory features including lower zone fibrosis, pulmonary nodules, pleural effusions and bronchiolitis obliterans. However, rheumatoid arthritis does not cause haemoptysis, making it the incorrect answer.

Sarcoidosis causes a range of pulmonary features from hilar lymphadenopathy to diffuse pulmonary infiltrates and fibrosis. The disease is most common in young Afro-Caribbean women and although shortness of breath and a dry cough are common symptoms, haemoptysis is not seen.

Question 10 of 23

A 59-year-old man is admitted with fever, chest pain, and breathlessness. Chest x-ray confirms a large right-sided pleural effusion.

Thoracic ultrasound confirms a large volume pleural effusion and a suitable site is marked. A chest drain is inserted without complication and the drain yields frank pus. Post-procedure, the patient is well and observations are unchanged.

Twenty-four hours later, the effusion is much improved on x-ray and minimal fluid is draining. This remains mildly purulent but non-bloody. However, you note bubbling in the underwater seal. On review of the chart, this has been the case since insertion. The patient is much improved and the drain is still swinging.

What is the most likely diagnosis?

A. Bronchopleural fistula
B. Iatrogenic diaphragmatic injury
C. Iatrogenic lung injury
D. Infection with gas-producing organism
E. Re-expansion pulmonary oedema

A Bronchopleural fistula

Bubbling of a chest drain inserted to drain an empyema may indicates that a bronchopleural fistula has occurred

The correct answer is **bronchopleural fistula**. This refers to abnormal communication between the pleural space and the bronchi. Complicated infection such as empyema is a well-recognised cause. Unexpected bubbling when a chest drain is inserted for empyema should raise the suspicion of this complication.

Iatrogenic diaphragmatic injury is incorrect. This is a very rare complication of chest drain insertion, made less likely by the use of ultrasound guidance and the large volume of fluid. It also would not lead to bubbling in the drain as there would be no leakage of air **Iatrogenic lung injury** is incorrect. While this could lead to bubbling of the drain, it is an unusual complication of chest drain insertion that is even less likely given the use of ultrasound guidance and the large volume of fluid here. We are also told that the procedure has gone ahead without complication. In the case of lung injury, we would expect increased pain and breathlessness during the procedure.

Infection with gas-producing organism is incorrect. An empyema may contain a variety of organisms but it is very rare to see gas in the fluid as a result of the infection alone. In the case described, there is also minimal fluid after 24 hours but ongoing bubbling, suggesting ongoing leakage of air into the pleural space. This suggests a structural problem such as a bronchopleural fistula.

Re-expansion pulmonary oedema is incorrect. This tends to present at the time of chest drain insertion with increased breathlessness, cough, and often chest pain. In the case described, the patient is well post-procedure with none of these features. It also would not cause bubbling in the drain.

Question 11 of 23

A 25-year-old man is reviewed prior to discharge. He was admitted to hospital one day ago with a small pneumothorax. As it was <1cm in size he was treated with high flow oxygen and he admitted for observations over 24 hours. His symptoms have resolved and a repeat chest x-Ray shows complete resolution of the pneumothorax.

What advice should he be given before discharge?

A. He should not dive for a year
B. He should never dive unless he undergoes pleurodesis
C. He should not drive for four weeks
D. He should have no contact sport for four weeks
E. He should not fly for four weeks

B. He should never dive unless he undergoes pleurodesis

'The correct answer is that he should be advised to never dive unless he undergoes pleurodesis. The British Thoracic Society (BTS) advises that a patient who has developed a pneumothorax should never dive again unless the undergo a pleurodesis procedure and are investigated following the procedure to ensure its success. He should also be advised not to fly, but this restriction is generally only for two weeks. There are no restrictions on driving or contact sport with pneumothorax.

Question 12 of 23

A 35-year-old man who lives in long-term residential care presents with drowsiness and increased respiratory effort. There is new consolidation on his chest x-ray and he is diagnosed with pneumonia. His care workers reported that he had some vomiting episodes 5 days previously.

A sputum sample is analysed in the microbiology laboratory. On microscopy of the sample, a mixture of Gram-positive cocci and Gram-negative rods is visible. At 48 hours there is growth on both the blood agar plate and the anaerobic agar plate.

What finding on sputum culture would provide evidence that his pneumonia is due to aspiration?

A. Bacteria which are sensitive to metronidazole
B. Bacteria which are sensitive to optochin
C. Bacteria which are sensitive to penicillin
D. Catalase negative organisms
E. Growth of Gram negative cocci

A. Bacteria which are sensitive to metronidazole

Anaerobic bacteria in sputum culture suggest aspiration pneumonia

Anaerobic bacteria often contribute to aspiration pneumonia as these bacteria are part of the microflora of the mouth and digestive tract, and may enter the respiratory system via aspiration of oral or gastrointestinal fluids. They are commonly found to cause infection alongside other aerobic bacteria. It is important to consider whether a patient's pneumonia may be due to aspiration, as not all the commonly used antibiotic options for community-acquired pneumonia will treat anaerobic infections.

Most **anaerobic bacteria** are sensitive to metronidazole, and conversely, metronidazole does not have any activity against non-anaerobes. Therefore the presence of metronidazole-sensitive organisms suggests that there are anaerobes present, which are often found in aspiration pneumonia.

Optochin is used as a test to differentiate *Streptococcus pneumoniae* from other alpha-haemolytic Streptococci. *Strep pneumoniae* is a common cause of community-acquired pneumonia and would not suggest aspiration pneumonia.

Most **anaerobic bacteria** are not sensitive to penicillin (with a few exceptions). A penicillinsensitive organism grown from sputum would be most likely to be a typical organism such as *Strep pneumoniae*.

The **catalase test** is used to differentiate Gram-positive cocci into *Staphylococci* or *Streptococci*. Streptococci are catalase negative.

The most important **Gram-negative cocci** are *Neisseria* and *Moraxella*. *Moraxella catarrhalis* can cause respiratory tract infections but is not associated with aspiration pneumonia.

Question 13 of 23

A 65-year-old gentleman who is visiting is family and usually resides in Chile attends the emergent department with shortness of breath of two days worsening.

Through translation by a relative, you understand that he is retired and formerly worked on coal extraction and in silica extraction but retired a few years ago due to health issues.

He usually has problems with feeling short of breath and can just about manage a flight of stairs but he has worsened and is now short of breath at rest. He has a cough which is productive of black sputum but he reports this is normal for him. He has lost 5 stone in weight over the last year.

He has a history of rheumatoid arthritis, ischaemic heart disease and hypertension. He is a smoker of 10 pack years and does not recall any exposure to tuberculosis or foreign pets.

On examination, he is in respiratory distress with use of the accessory muscles. No JVP can be seen and there are crepitations throughout the mid and upper zones. He looks drowsy and has peripheral cyanosis. There is clubbing and he is a febrile. He is saturating at 92% on 10 litres of oxygen via a face mask. There are no murmurs or signs of endocarditis.

ABG now pH	7.31
PaO2	7.92 kPa

PaCO2	6.44 kPa		
HCO3	21 mmol/l		
Chest X-Ray	upper zone reticulonodular changes with large cavity on right apex		
High resolution CT	ground glass opacities in upper zones with a modular appearance and centroacinar emphysema in the lower zone		
Hb	110 g/l	Na	139 mmol/l
Platelets	310* 10 /l	K	4.5 mmol/l
WBC	12.3 * 10 /l	Urea	9.9 mmol/l
Neuts	11.9 * 10 /l	Creatinine	178 µmol/l
Lymphs	0.2 * 10 /l	CRP	156 mg/l
c-ANCA	pending	p-ANCA	pending
Rheumatoid factor	pending		

What is the likely diagnosis?

A. Silicotuberculosis
B. Idiopathic pulmonary fibrosis
C. Granulomatosis with polyangiitis
D. Caplan Syndrome
E. pneumocystis jirovecii

D. Caplan Syndrome

Rheumatoid arthritis causes a severe form of coal workers pneumoconiosis called Caplan's syndrome

A complex background in a gentleman with severe respiratory failure. He has a background lung disease with smoking and possible coal and silica exposure. He is also immunosuppressed and so is at risk of PCP.

The key features are melanoptysis the coughing up of black sputum which is pathognomonic of coal workers pneumoconiosis. This disease is typically leads to the severe progressive massive fibrosis in those with rheumatoid arthritis which is named eponymously as Caplan's syndrome.

Question 14 of 23

A 32-year-old woman presents to the emergency department with shortness of breath. This has been worsening over the past 24 hours and was preceded by coryzal symptoms. She has a past medical history of asthma for which she takes regular montelukast and a Seretideµ inhaler.

On examination, she is alert but unable to speak in full sentences. Her temperature is 37.1ºC, oxygen saturation 92% on air, heart rate 83 beats per minute, respiratory rate 32 breaths per minute and blood pressure 130/83 mmHg. Auscultation of the chest reveals bilateral wheeze and poor air entry in the bases.

Her initial arterial blood gas on room air shows the following:

pH	7.46 (7.35-7.45)
PaO	7.5 kPa (10-13 kPa)
PaCo	3.1 kPa (4.7-6.0 kPa)
Lac	2.0 (<2.0)
HCo	24 (22-26)
BE	+3 (-2-2)

You initiate management with high flow oxygen, back-to-back salbutamol nebulisers, an ipratropium bromide nebuliser, intravenous hydrocortisone and intravenous magnesium sulphate.

On reassessment, in the subsequent hour, she appears more comfortable with a temperature of 37.3ºC, oxygen saturation 100% on high flow oxygen, heart rate 92 beats per minute, respiratory rate of 24 breaths per minute and blood pressure 126/78mmHg.

You repeat an arterial blood gas:

pH	7.34 (7.35-7.45)
PaO	43 kPa (10-13 kPa)
PaCo	3.2 kPa (4.7-6.0 kPa)
Lac	6.4 (<2.0)
HCo	19 (22-26)
BE	-5 (-2-2)

What is the most likely cause for the hyperlactatemia seen on the subsequent arterial blood gas?

A. Sepsis
B. Magnesium sulfate
C. Salbutamol
D. Pulmonary embolism
E. Stress response

C. Salbutamol

Excessive salbutamol use in acute asthma can lead to hyperlactatemia

Salbutamol is a cause of type B lactic acidosis (that is not caused by tissue hypoperfusion or hypoxia resulting in anaerobic metabolism). This should be kept in mind along with the clinical picture - in this case, the patient was orientated and not clinically toxic, hypoxic or hypotensive.

Magnesium sulfate is not reported to cause lactic acidosis.

The clinical picture is not in keeping with sepsis nor a pulmonary embolism. The symptoms, examination findings, observations and response to treatment are in keeping with a lifethreatening attack of asthma.

There is evidence that high levels of lactatemia in acute asthma are due to the effects of β- adrenergic agonists rather than a physiological response to the stress.

Question 15 of 23

A 32-year-old businessman presented with a 1-week history of a productive cough of yellow sputum, mild wheeze, and mild dyspnoea. He has a 2 pack year history of smoking but has been otherwise well. On examination, his blood pressure is 120/90 mmHg, heart rate is 80 beats/minute, oxygen saturation 98% on room air. On auscultation, there is a mild wheeze but his chest is otherwise clear.

Hb	140 g/l
Platelets	350 * 10 /l9
WBC	13 * 10 /l9

Chest x-ray is clear. How should he be managed?

A. Amoxicillin
B. Amoxicillin with clavulanic acid
C. Amoxicillin and doxycycline
D. Cough medicine and adequate hydration

E. Adequate hydration

E. Adequate hydration

The clinical scenario is of acute bronchitis in an otherwise well man. This is usually a self-limiting condition which resolves without antibiotics within 3 weeks and therefore the best management strategy is adequate analgesia and hydration. Cough medicines are not routinely recommended by NICE as they suppress natural mechanisms to keep the airways clear.

In someone who has a chronic chest condition, antibiotics may be prescribed but a delayed prescription strategy should be considered.

Finally, it is important to always remember to provide smoking cessation advice

Question 16 of 23

A 68-year-old woman presents to the emergency department with shortness of breath. A CT pulmonary angiogram is undertaken, which excludes a pulmonary embolism. However, it demonstrates a 4.5mm solid nodule in the right midzone.

What is the appropriate management of this lesion?

A. Biopsy immediately
B. CT PET immediately
C. MRI chest in 3 months
D. No further investigation required
E. Traditional CT chest in 12 months

D. No further investigation required

Solitary, solid and non-calcified lung nodules of less than 5mm in size do not warrant further investigations or monitoring

No further investigation required is the right answer. Solitary, solid and non-calcified lung nodules of less than 5mm in size do not warrant further investigations or monitoring.

Biopsy immediately is incorrect. If a lesion is determined to be sufficiently sinister to require a biopsy to exclude malignancy, a PET scan should be undertaken prior to biopsy. If the PET scan is positive the patient should proceed to biopsy.

CT PET immediately is incorrect. This is the investigation undertaken if a nodule is > 8mm and high risk. High-risk features include older age, female sex, family history of lung cancer, emphysema, spiculation and larger nodule size.

MRI chest in 3 months is incorrect. MRI scanning is not typically used in the investigation and monitoring of lung nodules.

Traditional CT chest in 12 months is incorrect. Undertaking a repeat CT in 3 rather than 12 months would be an appropriate option if the nodule was 5-6mm, or =>8mm and low-risk.

Question 17 of 23

A 63-year-old man presents to his general practitioner complaining of frequent wheezy exacerbations. He has a past medical history of chronic obstructive pulmonary disease. There is no history of asthma or atopy. He takes salbutamol as required. He is an ex-smoker with 35 pack-year history. He does not drink alcohol.

On examination, he is comfortable at rest. There is a mild expiratory wheeze audible in the midzones. The jugular venous pulse is not raised and there is no peripheral oedema.

Blood tests:

Hb	138 g/L	Male: (135-180)Female: (115 - 160)
Platelets	189 * 10 /L 9	(150 - 400)
WBC	4.8 * 10 /L 9	(4.0 - 11.0)
Neuts	3.1 * 10 /L 9	(2.0 - 7.0)
Lymphs	1.5 * 10 /L 9	(1.0 - 3.5)
Mono	0.2 * 10 /L 9	(0.2 - 0.8)
Eosin	0.0 * 10 /L 9	(0.0 - 0.4)
Na +	138 mmol/L	(135 - 145)
K +	4.2 mmol/L	(3.5 - 5.0)
Urea	5.2 mmol/L	(2.0 - 7.0)
Creatinine	93 µmol/L	(55 - 120)

Previous investigations did not demonstrate substantial variation in FEV1 over time or substantial diurnal variation in peak expiratory flow.

What is the most appropriate addition to his medication regimen?

A. Long acting beta agonist (LABA)
B. Long acting beta agonist (LABA) + long acting muscarinic antagonist (LAMA)
C. Long acting beta agonist (LABA) and inhaled corticosteroid (ICS)
D. Long acting muscarinic antagonist (LAMA)
E. LABA + LAMA + ICS

B. Long acting beta agonist (LABA) + long acting muscarinic antagonist (LAMA)

COPD - still breathless despite using SABA/SAMA and **no** asthma/steroid responsive features → add a LABA + LAMA

Long-acting beta2-agonist (LABA) + long-acting muscarinic antagonist (LAMA) is the correct answer. This patient has poorly controlled COPD. He has no features to suggest steroid responsiveness and therefore the next step should be the addition of a LABA+LAMA. He does not warrant inhaled corticosteroids on this basis.

Long-acting beta2-agonist (LABA) is incorrect. The patient needs a combination of LABA/LAMA.

Long-acting muscarinic antagonist (LAMA) is incorrect. The patient needs a combination of a LABA/LAMA.

LABA + LAMA + ICS is incorrect. This combination would be warranted in those patients with steroid-responsive features that fail a LABA + ICS combination.

Long-acting beta2-agonist (LABA) and inhaled corticosteroid (ICS) is incorrect. This would be a suitable choice if the patient had features of steroid-responsiveness.

Question 18 of 23

A 55-year-old HIV-positive American woman develops fever, cough and fatigue after returning from her Thanksgiving holiday. Her chest radiograph is normal. Her symptoms persist despite treatment with oral co-amoxiclav. She undergoes a bronchoscopy and her bronchial washings grow *Histoplasma capsulatum*.

What single treatment is most likely to resolve her symptoms?

A. Amphotericin
B. Caspofungin
C. Fluconazole
D. Ivermectin
E. Oral prednisolone

A. Amphotericin

Amphotericin or itraconazole are the pharmacological agents of choice for histoplasmosis

Histoplasma capsulatum is a rare fungus that can be found in the United States of America that can cause respiratory symptoms in immunocompromised patients. It should be treated with **amphotericin** or itraconazole.

Caspofungin is also an antifungal drug, but it is more appropriate to treat patients with *Aspergillus* infections.

Fluconazole is also an antifungal drug, but it is more appropriate to treat patients with *Candida* infections.

Ivermectin is an anti-parasitic drug, but it is more appropriate to treat other infections such as *Strongyloides* and Norwegian scabies.

Oral prednisolone is used to treat many respiratory diseases including acute asthma. However, it is not appropriate for the initial treatment of a fungal infection such as *Histoplasma* and appropriate anti-fungal medication should be prescribed first

Question 19 of 23

A 63-year-old man was admitted two hours ago with an infective exacerbation of COPD. He had become rapidly unwell following one day of a productive cough, developing worsening fever, shortness of breath and chest tightness. Arterial blood gas demonstrated that he was acidotic in type two respiratory failure despite treatment with back-to-back nebulisers, IV hydrocortisone and IV antibiotics. He was started on non-invasive ventilation (NIV) with IPAP of 10cmH O and EPAP of 4cmH O. This has been increased to IPAP of 16cmH O. During a review of this patient, the nurses ask what should be done about his nebulisers, prescribed 3 hourly, during NIV treatment.

How should he be treated with nebulisers whilst having NIV?

A. Add nebulisers into NIV mask
B. Take off mask to administer nebulisers
C. Hold nebulisers whilst on NIV
D. Give IV salbutamol instead of nebulised
E. Stop NIV whilst on 2 hourly nebulisers

B. Take off mask to administer nebulisers

The correct answer is to take off the mask to administer nebulisers. This is a patient with a severe exacerbation of COPD with type two respiratory failure with acidosis not responding to medical therapy and needing NIV. Whilst it is possible to give nebulisers into some NIV masks it is preferable to administer nebulisers off NIV as a far greater proportion of the drug reaches the airways, necessary for bronchodilation. Giving nebulised medication into ongoing NIV is not recommended by the BTS as the pressures reduce the effective dose reaching the airways. Stopping nebulisers or NIV are both inappropriate as both are necessary.

Question 20 of 23

An 81-year-old man is admitted to the geriatric ward with confusion.

His past medical history is significant for hypertension, hyperlipidaemia, and a TIA 3 years ago. There is no history of any cognitive impairment. He has never smoked and consumes minimal alcohol.

Urine sent for culture and sensitivity is positive for *Escherichia coli*, so the patient is treated for a presumed delirium triggered by a UTI.

As part of the delirium workup, a routine chest x-ray is also performed. The chest x-ray is reported as follows:

There is a 3 cm right-sided apical pneumothorax.

On review, the patient does not complain of any shortness of breath or chest pain. His observations are shown below:

- Heart rate: 66 bpm
- Respiratory rate: 18 breaths/minute
- Oxygen saturation: 98%
- Temperature: 37.0ºC
- Blood pressure: 124/76 mmHg

What is the most appropriate management?

A. Ambulatory device insertion
B. Chest drain insertion
C. Conservative management
D. Needle aspiration
E. Pleurodesis

C. Conservative management

Minimally symptomatic pneumothorax, regardless of size, can be treated with conservative treatment / regular follow-up

Conservative management is correct. This patient has an asymptomatic pneumothorax of 3 cm. Since the 2023 BTS guidelines update the determinants of the management strategy have changed, and the focus is no longer on the size of the pneumothorax but on the level of symptoms experienced by the patient. It is recommended that any asymptomatic pneumothorax, regardless of size, be managed conservatively.

Ambulatory device insertion is incorrect. This would be an option if the patient was symptomatic from their pneumothorax and did not have any of the 'high risk' characteristics (these include haemodynamic compromise, hypoxia, pre-existing lung disease, bilateral pneumothoraces, age over 50 years with a significant smoking history, or haemopneumothorax). The patient in this case is asymptomatic, so conservative management is the correct answer.

Chest drain insertion is incorrect. A chest drain would be indicated if the patient was symptomatic from their pneumothorax and also had any of the 'high risk' characteristics mentioned above or if a symptomatic patient without 'high risk' features underwent unsuccessful needle aspiration. The patient in this question has neither symptoms nor any of the 'high risk' characteristics; this answer is therefore incorrect.

Needle aspiration is incorrect. Needle aspiration would be indicated if the patient was symptomatic from their pneumothorax and had none of the 'high-risk' features mentioned above. This patient is asymptomatic, so this answer is incorrect.

Pleurodesis is incorrect. This procedure prevents recurrent pneumothoraces. This patient does not report having had a previous pneumothorax; thus this answer is incorrect.

Question 21 of 23

A 78-year-old female presents to the Emergency Department with confusion, nausea and vomiting. She has been generally unwell with fatigue, weakness and fevers for 4 weeks. On examination her respiratory rate is 26/min, oxygen saturations are 98% on 4 litres of oxygen, blood pressure 100/65 mmHg, pulse 120/min and temperature is 36.3 C. Her airway is patent and crepitations are present at both bases. There are crusting lesions beneath both nostrils, the pulse is thready and regular, heart sounds are normal and her abdomen is soft non-tender. Electrocardiogram shows a sinus tachycardia and urine dip showed 3+ blood and protein. Arterial blood gas on 4 litres of oxygen is as follows:

pH	7.35
pO2	7.79 kPa
pCO2	3.52 kPa
Bicarbonate	17 mmol/l
Base Excess	-6.9 mmol/l
Lactate	4.5 mmol/l

Venous blood analysis is as follows:

Hb	119 g/l	Na +	129 mmol/l
Platelets	511 * 10 /l 9	K +	6.2 mmol/l
WBC	19.1 * 10 /l 9	Urea	42.1 mmol/l
Neuts	17.2 * 10 /l 9	Creatinine	497 µmol/l
Lymphs	1.8 * 10 /l 9	CRP	241 mg/l
Eosin	0.04 * 10 /l9		

The patient was resuscitated with fluids and antibiotics although the full septic screen was negative and renal function remained poor. ANA and cANCA pattern were positive with PR3 antibodies found and the renal team were involved.

What is the most likely underlying diagnosis?

A. Lymphoma
B. Churg-Strauss syndrome
C. Granulomatosis with polyangiitis
D. Acute myeloid leukaemia
E. Goodpasture's syndrome

C. Granulomatosis with polyangiitis

Consider granulomatosis with polyangiitis when a patient presents with ENT, respiratory and kidney involvement

This patient presents with systemic inflammatory response syndrome (SIRS) as evidence by her tachycardia, tachypnoea and leucocytosis. The most common cause, in this case, would be sepsis, however, history, examination and investigations have not demonstrated a source. The raised lactate suggests end organ hypo-perfusion. Routine biochemistry demonstrates severe renal impairment which must be assumed to represent stage 3 acute kidney injury (AKI) without previous values for comparison. Arterial blood gas demonstrates a compensated metabolic acidosis consistent with acute kidney injury, sepsis or SIRS.

With a negative septic screen we must consider alternative causes of SIRS and in the context of stage 3 AKI and heavy blood and protein on the urine dip, intrinsic renal pathology must be suspected. Granulomatosis with polyangiitis (previously known as Wegener's Granulomatosis) typically presents with non-specific symptoms, an unwell patient and AKI with active urinary sediment. Nasal crusting due to granulomas and a collapsed nasal septum may also be found. The diagnosis is also suggestive by a positive cANCA with PR3 antibodies although renal biopsy is required to confirm the diagnosis

Question 22 of 23

A 72-year-old man is admitted to hospital for an elective knee replacement. The surgery is completed successfully and induction, maintenance and emergence of anaesthesia proceeds without any problem. Several hours after the operation however, the patient's saturations start to drop, reaching a level of 91% when the patient is started on oxygen via face mask. He also starts coughing up small mucous plugs.

His background history includes mild COPD that is treated with tiotropium and hypertension which is treated with ramipril and amlodipine. There is no relevant family history and he has smoked 15 cigarettes a day for 30 years.

What is the most likely diagnosis?

A. Basal atelectasis
B. Pneumococcus pneumoniae
C. Streptococcus pneumoniae
D. Pulmonary embolism
E. Pneumothorax

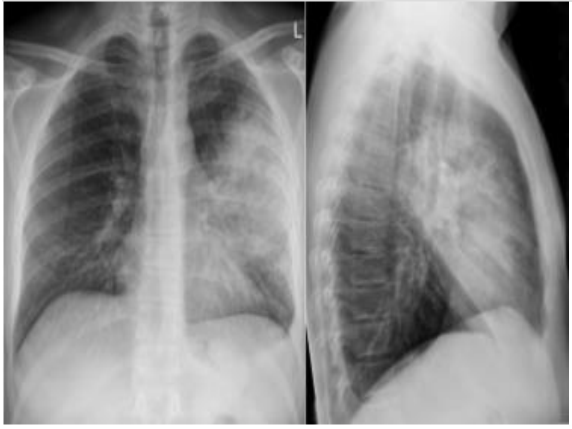
A. Basal atelectasis

The most likely diagnosis in this scenario is basal atelectasis - a condition where mucous trapped in the bronchial tree results in small airway obstruction. There is often segmental lung collapse due to the basal atelectasis. Management is with chest physiotherapy and saline nebulisers to encourage production of the mucous.

Question 23 of 23

A 40-year-old male smoker presents with a 10-day history of shortness of breath and pleuritic chest pain. He has been acutely short of breath for the last 24 hours. His respiratory rate is 34 breaths per minute and his blood pressure is 80/45 mmHg. His oxygen saturations are 84% on arrival in triage. Initial blood results and chest-x-ray are as below.

Hb	118 g/L	Male: (135-180)
Platelets	564 * 10 /L	(150 - 400)
WBC	24 * 10 /L	(4.0 - 11.0)
Na	136 mmol/L	(135 - 145)
K	5.6 mmol/L	(3.5 - 5.0)
Urea	11 mmol/L	(2.0 - 7.0)
Creatinine	154 µmol/L	(55 - 120)
CRP	250 mg/L	(< 5)



What is his risk of mortality based on a scoring system used in the management of this condition?

A. 10%
B. 15%
C. 3%
D. 30%
E. <3%

B. 15%

This patient has severe community-acquired pneumonia. The x-ray shows dense consolidation in a lobar distribution, affecting the left middle zone. His CURB-65 score is 3 (scoring for respiratory rate, urea, and blood pressure). This equates to an in-hospital mortality risk of greater than **15%**. These patients should be considered for referral to high dependency or intensive care.

<3% is incorrect. This is the mortality associated with a CURB-65 score of 0.

3% is incorrect . This is the mortality associated with a patient with a CURB-65 score of 1. **10% is incorrect**. A CURB-65 score of 2 is associated with a mortality risk of 5-10%. **30% is incorrect**. This is the mortality associated with a CURB-65 score of 5

Alrajhi Jad

PASSMEDICINE

GASTROENTEROLOGY

Internal medicine

EDITED BY
DR. JAMIL ALRAJHI

2024

773-555-493
jameelalrajhi@gmail.com

GASTROENTEROLOGY

passmedicine PART 2

2024

Question 1 of 259

A 65-year-old man is seen in the gastroenterology clinic with bloating and loose stool for the last 8 months. This is associated with vague abdominal discomfort. He has a normal diet and has not lost any weight.

Past medical history includes type 2 diabetes and psoriasis. He also has IgA deficiency and suffers from intermittent chest infections, most recently 2 months ago. After treatment for the infection, his diarrhoea improved slightly for a short while.

He is referred for an oesophago-gastro-duodenoscopy and colonoscopy which are normal. Several biopsies are taken which return showing normal mucosa.

What treatment is most likely to be effective?

A. Avoid gluten
B. Creon
C. Mesalazine
D. Omeprazole
E. Rifaximin

E. Rifaximin

Rifaximin is the treatment of choice for small bowel bacterial overgrowth syndrome

The normal endoscopic and histological findings help to narrow our list of differentials in this patient. A diagnosis of irritable bowel syndrome (IBS) could be considered, but, he has several risk factors for small bowel bacterial overgrowth. Risk factors are:

- Motility delay, such as diabetes, scleroderma
- Anatomical, such as strictures
- Immune-related, such as IgA deficiency

Rifaximin is correct as this is the treatment of choice for small bowel bacterial overgrowth syndrome. A trial of treatment can be used as a diagnostic tool. The alternative is using the hydrogen breath test.

Avoiding gluten would be correct if celiac disease was the diagnosis, however, the lack of characteristic histological findings makes this unlikely.

Creon is a pancreatic enzyme replacement therapy used in pancreatic insufficiency. This would be more likely if there were steatorrhoea or weight loss due to malabsorption.

Mesalazine is used in the management of inflammatory bowel disease (IBD). The normal endoscopic and histological findings make IBD unlikely.

Omeprazole can be used in gastro-oesophageal reflux disease (GORD) which typically presents with heartburn and regurgitation symptoms. These are not present in this case and hence omeprazole is not of benefit.

Small bowel bacterial overgrowth syndrome

Small bowel bacterial overgrowth syndrome (SBBOS) is a disorder characterised by excessive amounts of bacteria in the small bowel resulting in gastrointestinal symptoms.

Risk factors for SBBOS

- neonates with congenital gastrointestinal abnormalities
- scleroderma
- diabetes mellitus

It should be noted that many of the features overlap with irritable bowel syndrome:

- chronic diarrhoea
- bloating, flatulence
- abdominal pain

Diagnosis

- hydrogen breath test
- small bowel aspiration and culture: this is used less often as invasive and results are often difficult to reproduce
- clinicians may sometimes give a course of antibiotics as a diagnostic trial

Management

- correction of the underlying disorder
- antibiotic therapy: rifaximin is now the treatment of choice due to relatively low resistance. Co-amoxiclav or metronidazole are also effective in the majority of patients.

Question 2 of 259

A 54-year-old woman presents to her general practitioner with distressing abdominal symptoms. She complains of a combination of bloating, diarrhoea, abdominal discomfort and abdominal distension. She has tried a gluten-free diet, without significant improvement. She has a past medical history of limited systemic sclerosis. She does not take any regular medications.

On examination, her abdomen is mildly distended but otherwise unremarkable. She has skin thickening extending to the dorsum of her hands. Microstomia and telangiectasia are noted on her face.

Blood tests:

Bilirubin	12 µmol/L	(3 - 17)
ALP	89 u/L	(30 - 100)
ALT	32 u/L	(3 - 40)
Î³GT	44 u/L	(8 - 60)
Albumin	40 g/L	(35 - 50)
Hb	136 g/L	Male: (135-180)Female: (115 - 160)
Platelets	187 * 10 /L 9	(150 - 400)
WBC	4.5 * 10 /L 9	(4.0 - 11.0)
Na +	136 mmol/L	(135 - 145)
K +	4.1 mmol/L	(3.5 - 5.0)
Urea	5.2 mmol/L	(2.0 - 7.0)
Creatinine	89 µmol/L	(55 - 120)
CRP	4 mg/L	(< 5)
Vitamin B12	120 pg/ml	(160-950)
Serum folate	21.2 ng/mL	(2.7-17)

What is the most likely diagnosis?

A. Coeliac disease
B. Pernicious anaemia
C. Irritable bowel syndrome
D. Small bowel bacterial overgrowth
E. Whipple's disease

D. Small bowel bacterial overgrowth

A combination of low vitamin B12 and high folate levels favours small bowel bacterial overgrowth

Small bowel bacterial overgrowth is the correct answer. Obtaining a diagnosis in someone with vague abdominal symptoms is often difficult. However, this patient has a diagnosis of scleroderma, which is a risk factor for small bowel bacterial overgrowth. Furthermore, the low B12 and high folate levels are characteristic of this condition. This is because bacteria in the bowel absorb vitamin B12 and convert it to its inactive analogue, which then competes for normal B12 absorption in the gut, resulting in low levels. Conversely, bacteria are able to synthesise folate, resulting in normal or elevated levels. Taken together, the most likely diagnosis is small bowel bacterial overgrowth.

Irritable bowel syndrome is incorrect. While this can also give rise to symptoms such as bloating, diarrhoea, abdominal discomfort and abdominal distension, the pre-existing diagnosis of scleroderma and abnormal vitamin B12 and folate levels, make a functional diagnosis less likely.

Coeliac disease is incorrect. We would expect a gluten-free diet to improve her symptoms. Additionally, this disease is often associated with folate deficiency rather than a raised folate level, due to an inability to absorb in the upper part of the gastrointestinal tract.

Whipple's disease is incorrect. Joint pain is characteristic of this condition, which is not mentioned in this case. Additionally, folic acid deficiency is the expected finding, rather than excess.

Pernicious anaemia is incorrect. This is a cause of B12 deficiency.

However, the patient does not present with symptoms and signs of significant B12 deficiency but rather abdominal symptoms. Symptoms and signs of significant B12 deficiency include cognitive changes, shortness of breath, headache, indigestion, loss of appetite and palpitations. The presentation of this case suggests the B12 deficiency itself is not the principal cause of symptoms.

Small bowel bacterial overgrowth syndrome

Small bowel bacterial overgrowth syndrome (SBBOS) is a disorder characterised by excessive amounts of bacteria in the small bowel resulting in gastrointestinal symptoms.

Risk factors for SBBOS

- neonates with congenital gastrointestinal abnormalities
- scleroderma
- diabetes mellitus

It should be noted that many of the features overlap with irritable bowel syndrome:

- chronic diarrhoea
- bloating, flatulence
- abdominal pain

Diagnosis

- hydrogen breath test
- small bowel aspiration and culture: this is used less often as invasive and results are often difficult to reproduce
- clinicians may sometimes give a course of antibiotics as a diagnostic trial

Management

- correction of the underlying disorder
- antibiotic therapy: rifaximin is now the treatment of choice due to relatively low resistance. Co-amoxiclav or metronidazole are also effective in the majority of patients.

Question 3 of 259

A 44-year-old woman is referred to the gastroenterology outpatient with intractable bloating and flatulence. She has a past medical history of diffuse cutaneous systemic sclerosis. She takes mycophenolate mofetil.

The examination reveals mild abdominal bloating but a soft abdomen. Her skin is diffusely thickened.

Given the probable diagnosis, what is the best treatment?

A. Amoxicillin + metronidazole + omeprazole
B. Creon
C. Hyoscine butylbromide
D. Metronidazole
E. Rifaximin

E. Rifaximin

Rifaximin is the treatment of choice for small bowel bacterial overgrowth syndrome

Rifaximin is correct. The patient presents with chronic bloating and flatulence. Given her history of systemic sclerosis, the likely cause is small bowel bacterial overgrowth (SBBO). The diagnosis can be confirmed with a hydrogen breath test. In this test, hydrogen exhaled in the breath is measured using a gas chromatograph. Bacteria colonizing the large bowel in health and small bowel (in disease) produce hydrogen by fermentation of unabsorbed carbohydrates. Small amounts of hydrogen are produced normally from unabsorbed carbohydrates reaching the colon. Large amounts are produced if there is malabsorption of carbohydrates in the small intestine (allowing larger amounts to reach the colon) or if there are excess bacteria in the small bowel. The hydrogen produced by bacteria is exhaled in the breath and can then be measured.

Amoxicillin + metronidazole + omeprazole is incorrect. This is a treatment regimen for *H. pylori* eradication. This condition typically presents with dyspepsia rather than bloating and flatulence.

Creon is incorrect. This treatment is used for patients with pancreatic exocrine insufficiency. Typically, these patients have steatorrhoea (characterized by pale, difficult-to-flush stools). Stool abnormalities are not mentioned here.

Hyoscine butylbromide is incorrect. This is an anti-spasmodic, which sometimes provides pain relief for irritable bowel syndrome (IBS). While bloating and flatulence may be features of IBS, buscopan (as it is more commonly known) will mostly help with pain. Additionally, given her underlying diagnosis of systemic sclerosis, a significant pathology is more likely.

Metronidazole is incorrect. This treatment may well be effective for SBBO but rifaximin is preferred as it demonstrated a significantly higher decontamination rate when compared with metronidazole.

Question 4 of 259

A 54-year-old woman presents to the gastrointestinal clinic with a 4-month history of abdominal bloating and cramps associated with loose stools. She denies vomiting but does recall feeling nauseated with a reduced appetite. Her past medical history includes type 2 diabetes mellitus for which she takes metformin, gliclazide and long-acting insulin.

On examination, she is afebrile with a heart rate of 82 beats/min and a blood pressure of 132/77mmHg. Her abdomen is soft and non-tender with no palpable masses. Her BMI is 25kg/m².

Investigations are requested:

Hydrogen breath test	20 ppm within 1 hour of oral glucose ingestion

Given the likely diagnosis, what is the recommended management for this patient?

A. Amoxicillin, metronidazole and omeprazole
B. Ciprofloxacin
C. Hydrocortisone
D. Rifaximin
E. Vancomycin

D. Rifaximin

Rifaximin is the treatment of choice for small bowel bacterial overgrowth syndrome

Rifaximin is correct. This patient has symptoms and signs of small bowel bacterial overgrowth syndrome given the history of prolonged diarrhoea and abdominal pain and bloating. Symptoms can often overlap with irritable bowel syndrome. However, small bowel bacterial overgrowth syndrome is caused by excessive bacteria within the small bowel. Risk factors include diabetes mellitus, as seen in this patient's past medical history. A hydrogen breath test is used in the diagnosis of small bowel bacterial overgrowth syndrome with a positive result indicating the diagnosis. The raised reading seen here further supports the diagnosis. Rifaximin is used in the management of small bowel bacterial overgrowth syndrome. It is currently the preferred treatment recommended by the British Society of Gastroenterology.

Amoxicillin, metronidazole and omeprazole is incorrect. This is used in the management of infection with *Helicobacter pylori*. Unlike a positive hydrogen breath test, *Helicobacter pylori* infection is associated with a positive urea breath test. This combination of antibiotics and omeprazole is not used in the management of small bowel bacterial overgrowth syndrome.

Ciprofloxacin is incorrect. This is used in the management of urinary tract infections, diverticulitis and prostatitis. However, it plays no role in the management of small bowel bacterial overgrowth syndrome.

Hydrocortisone is incorrect. Corticosteroids are used in the management of acute flares of inflammatory bowel disease. However, they are not used in the management of small bowel bacterial overgrowth syndrome.

Vancomycin is incorrect. This is a second-line option recommended by the British Society of Gastroenterology. However, it would not be the initial choice of antibiotic to use.

Question 5 of Question 259

A 24-year-old woman who is known to have haemochromatosis comes to the antenatal clinic for review. She is 12 weeks pregnant and wishes to keep the baby. On examination her blood pressure is 105/70 mmHg, her pulse is 67 beats per minute and regular. Cardiac function is normal and a liver biopsy from 6 months earlier shows no evidence of hepatic fibrosis.

Hb	120 g/l
Platelets	191 * 10 /l9
WBC	7.1 * 10 /l9
Ferritin	600 mcg/l

Which of the following is the most appropriate way to manage the haemochromatosis in pregnancy?

A. Observation only
B. Oral deferiprone
C. Venesection once every two months
D. Iron supplementation
E. Vitamin C supplementation

A. Observation only

Where the patient's cardiac function is within the normal range, and as here there is no evidence of hepatic fibrosis on liver biopsy, there is no need for intervention in pregnancy for haemochromatosis.

Once the pregnancy has been completed, both cardiac and liver function should be reassessed.

Deferiprone is an iron chelating agent normally used in the treatment of haemochromatosis.There is limited evidence to support its safe use in pregnancy, and the ferritin is only moderately elevated. In the presence of no significant end-organ damage, chelation therapy isn't indicated, nor is venesection. Iron supplementation and vitamin C supplementation may risk a potentially damaging further elevation in ferritin.

There is current debate as to whether patients without cirrhosis ever require phlebotomy at all. Discussion points out that the major benefit has been demonstrated in patients with documented cirrhosis, unlike the patient described here. The lack of randomised controlled trial evidence in this regard is unsurprising given the prolonged nature of haemochromatosis with respect to progression. Post pregnancy this patient may be able to manage her ferritin levels with dietary measures alone.

Haemochromatosis: investigation and management

Haemochromatosis is an autosomal recessive disorder of iron absorption and metabolism resulting in iron accumulation. It is caused by the inheritance of mutations in the HFE gene on both copies of chromosome 6.

Screening for iron overload

There is continued debate about the best investigation to screen for haemochromatosis.

- general population
    - transferrin saturation is considered the most useful marker
    - ferritin should also be measured but is not usually abnormal in the early stages of iron accumulation
- testing family members
    - genetic testing for HFE mutation

These guidelines may change as HFE gene analysis become less expensive

Typical iron study profile in patient with haemochromatosis

- transferrin saturation > 55% in men or > 50% in women
- raised ferritin (e.g. > 500 ug/l) and iron
- low TIBC

Further tests and diagnosis

Further tests

- liver function tests
- molecular genetic testing for the C282Y and H63D mutations
- MRI is generally used to quantify liver and/or cardiac iron
- liver biopsy is now generally only used if suspected hepatic cirrhosis

Management

Outline

- venesection is the first-line treatment
    - monitoring adequacy of venesection: transferrin saturation should be kept below 50% and the serum ferritin concentration below 50 ug/l
    - desferrioxamine may be used second-line

Question 6 of 259

A 42-year-old lady with known longstanding Crohn's disease was referred to the gastroenterology clinic. She presented to her GP with an 8-month history of tiredness. Since then she has developed loose stools with vague persistent abdominal pains with bloating and weight loss of approximately 4kg over the corresponding period. Her symptoms were initially attributed to irritable bowel syndrome but despite a trial of mebeverine 135mg TDS and loperamide 2mg PRN her symptoms persisted. She stated that her Crohn's disease was well controlled for several years with mesalazine 500mg TDS. In addition to Crohn's disease, she also had a past medical history of asthma and eczema for which she was prescribed mesalazine, Clenil modulite 200mcg BD, salbutamol PRN and Diprobase cream. She consumes 28 units of alcohol per week and is a non-smoker.

On examination, she appeared pale but otherwise well. She had a temperature of 37.3ºC, a heart rate of 88/min and a blood pressure of 122/78 mmHg. Examination of the cardiovascular and neurological system was unremarkable. Examination of the gastrointestinal system likewise was unremarkable with no organomegaly and a soft non-tender abdomen.

Investigations revealed the following results:

Hb	86 g/l
MCV	112 fl
Platelets	342 * 10 /l9
WBC	7.2 * 10 /l9
TSH	1.96 u/l
B12	64 ng/l (160-900)
Folate	28 mcg/l (3-20)

Gastroscopy: normal appearance

Colonoscopy: normal appearance

Capsule endoscopy: jejunal strictures and fistulae

Schilling test: Prior to administration of intrinsic factor 1% B12 isotope

excreted

Post-administration of intrinsic factor 1% B12 isotope excreted What is the most likely diagnosis?

A. Bacterial overgrowth
B. Blind loop syndrome
C. Pernicious anaemia
D. Terminal ileal disease
E. Vegan diet

A. Bacterial overgrowth

The schilling test here shows an initial abnormally low excretion of B12 isotope which is not normalised by administration of intrinsic factor, excluding the presence of an intrinsic factor deficiency. This lady has presented with chronic malabsorptive symptoms and a macrocytic anaemia. The capsule endoscopy did not reveal any ileum disease and in this context, the most likely diagnosis is one of bacterial overgrowth.

Bacterial overgrowth affects the small intestine, and by definition is diagnosed by jejunal aspirate revealing the presence of >105 bacteria/ml. It often presents with non-specific abdominal symptoms such as nausea, abdominal pain, bloating, diarrhoea, steatorrhoea and weight loss. It also presents with symptoms of malabsorption of various nutrients. Predisposing factors include anatomical disturbances of the intestine (such as fissuring and strictures in this instance secondary to Crohn's disease) dysmotility and various medications such as proton pump inhibitors.

Question 7 of 259

A 65-year-old man attends the clinic with a 12-month history of chronic diarrhoea, bloating, and abdominal pain. He has a past medical history of poorly controlled diabetes mellitus.

Hb	115 g/L	Male: (135-180)Female: (115 - 160)
Platelets	158 * 10 /L 9	(150 - 400)
WBC	6.2 * 10 /L 9	(4.0 - 11.0)
Na +	136 mmol/L	(135 - 145)
K +	4.2 mmol/L	(3.5 - 5.0)
Urea	6.2 mmol/L	(2.0 - 7.0)
Creatinine	74 µmol/L	(55 - 120)
CRP	4 mg/L	(< 5)
Hydrogen breath test	Rise in breath hydrogen by 12 ppm above the basal rate	

What treatment is indicated?

A. Co-amoxiclav
B. Faecal transplant
C. Loperamide
D. Metronidazole
E. Rifaximin

E. Rifaximin

Rifaximin is the treatment of choice for small bowel bacterial overgrowth syndrome

The clinical features and positive hydrogen breath test are suggestive of small bowel bacterial outgrowth (SBBO). It would be important to correct any underlying risk factors if possible (e.g. better-controlled diabetes in this case).

Rifaximin is correct. Rifaximin is now the treatment of choice due to its relatively low resistance.

Co-amoxiclav is incorrect. Co-amoxiclav is also effective in the majority of patients with SBBO, however, higher rates of resistance are present making rifaximin the treatment of choice.

Metronidazole is incorrect. Metronidazole is also effective in the majority of patients with SBBO, however, higher rates of resistance are present making rifaximin the treatment of choice.

Loperamide is incorrect. Antimotility agents such as loperamide have been shown to promote bacterial overgrowth and should therefore be avoided.

Faecal transplant is incorrect. Faecal transplantation had previously been considered as a possible solution, but, in fact, seems to increase the risk of SBBO.

Question 8 of 259

A 53-year-old man presents to his general practitioner complaining of fatigue and joint pains. He has no significant past medical history and does not take any regular medications. He does not smoke or drink alcohol.

On examination, a 'slate-grey' appearance of the skin is noted and there is tenderness at the 2nd and 3rd MCP joints on his left hand but no obvious swelling. Cardiovascular, respiratory and abdominal examinations are unremarkable.

Blood tests:

Hb	138 g/L	Male: (135-180)Female: (115 - 160)
Platelets	189 * 10 /L 9	(150 - 400)
WBC	4.6 * 10 /L 9	(4.0 - 11.0)
Na +	136 mmol/L	(135 - 145)
K +	4.2 mmol/L	(3.5 - 5.0)
Urea	6.2 mmol/L	(2.0 - 7.0)
Creatinine	89 µmol/L	(55 - 120)
CRP	4 mg/L	(< 5)
9am cortisol	400 nmol/L	(170-420)
TSH	2.2 mU/L (0.5-5.0)	
HbA1c	42 mmol/L (<48)	

Plain radiography of the hand demonstrates hook-like osteophytes at the 2nd and 3rd MCP joints on the left hand.

Given the likely diagnosis, what is the best screening testing for this condition in the general population?

A. Ferritin
B. HFE genetic testing
C. Short synacthen test
D. Transferrin saturation
E. Insulin like growth factor - 1 (IGF-1)

D. Transferrin saturation

Screening for haemochromatosis

- general population: transferrin saturation > ferritin
- family members: HFE genetic testing

Transferrin saturation is correct. This man presents with characteristic skin and x-ray (hook-like osteophytes at 2nd and 3rd MCP joints) of haemochromatosis. Fatigue is a common symptom. The best test to screen for this condition in the general population is transferrin saturation.

Ferritin is incorrect. This may be normal in early disease and is inferior to the transferrin saturation for screening purposes.

HFE genetic testing is incorrect. Genetic testing is more appropriate if there is a relevant family history of haemochromatosis, which is not mentioned here.

Insulin-like growth factor - 1 (IGF-1) is incorrect. This can be used to screen for acromegaly, which can also cause fatigue and joint pain. However, the skin and radiographic appearances are more characteristic of haemochromatosis.

Short synacthen test is incorrect. Addison's can cause skin changes and fatigue. However, the 9am cortisol and electrolytes are normal, making this diagnosis unlikely.

Question 9 of 259

A 45-year-old woman is referred to gastroenterology with reports of six months of chronic diarrhoea, bloating, flatulence and episodic abdominal pain. Her stools are a normal colour and do not float. She has a past medical history of limited systemic sclerosis. She does not take any regular medications. There is no recent history of antibiotic use.

On examination, she has mild skin thickening affecting the hands. Abdominal examination is unremarkable.

Hb	122 g/L	Male: (135-180)Female: (115 - 160)
Platelets	189 * 10 /L $_9$	(150 - 400)
WBC	4.2 * 10 /L $_9$	(4.0 - 11.0)
Na $_+$	137 mmol/L	(135 - 145)
K $_+$	4.2 mmol/L	(3.5 - 5.0)
Urea	5.2 mmol/L	(2.0 - 7.0)
Creatinine	88 µmol/L	(55 - 120)
CRP	4 mg/L	(< 5)

Given the likely diagnosis, which is the most appropriate diagnostic test?

A. Hydrogen breath testing
B. Oesophageal manometry
C. SeHCAT
D. Serology
E. Stool antigen testing

A. Hydrogen breath testing

Hydrogen breath testing is an appropriate first line test for diagnosis of small bowel overgrowth syndrome

Hydrogen breath testing is the correct answer. The patient presents with chronic diarrhoea, bloating, flatulence and abdominal pain. Small bowel overgrowth syndrome is common in systemic sclerosis and in this context, these symptoms are highly suggestive of the condition. It is diagnosed by hydrogen breath testing. In those patients with overgrowth of bacteria in the small bowel, sugar is converted to hydrogen by excess bacteria before it has a chance to be absorbed. The increased hydrogen is noted in the breath of affected individuals.

SeHCAT is incorrect. This is a scan used to diagnose bile acid malabsorption. This can be a primary or secondary problem (secondary to gastrointestinal disease). While this condition can also cause many of the symptoms outlined above, it is not as strongly associated with systemic sclerosis as small bowel overgrowth syndrome. Additionally, it also often causes steatorrhoea, and we are told her stools do not float. and are normal in colour.

Oesophageal manometry is incorrect. This can be used to diagnose gastro-oesophageal reflux disease. This is not associated with diarrhoea.

Serology is incorrect. This can be used to diagnose some *Helicobacter pylori* infections but diarrhoea tends not to be a feature of this condition.

Stool antigen testing is incorrect. This can be used to diagnose *Helicobacter pylori* infection or may provided indirect evidence of *Clostridium difficile* infection. However, diarrhoea is not a feature of the former and the history is too long for the latter, which tends to cause a more acute problem.

Question 10 of 259

An 18-year-old lady works as a nursery teaching assistant and has noticed her right arm always shakes as she stretches it out to write on the blackboard at school. She has also started dropping cups of tea because of shaking on picking them up. Her balance has become poor and she now falls over easily when playing hopscotch with the children. These problems have developed over 14 months slowly. Her neurological examination reveals a postural tremor of the upper limbs; right worse than left, as well as a broad-based ataxic gait. She has some co-existent rigidity of the right upper limb. Slit lamp examination reveals browngreen deposits in Descemet's membrane of the cornea. The remainder of the clinical examination is unremarkable.

What is the most appropriate treatment for the underlying condition?

A. Dimercaprol
B. Levodopa
C. Beta-interferon
D. D-penicillamine
E. Calcium EDTA

D. D-penicillamine

Treatment for Wilson's disease is currently penicillamine

The diagnosis is Wilson's disease: an autosomal-recessive condition that produces neurological, psychiatric, and hepatic dysfunction. Presentation is usually in children and young adults in the form of a postural tremor that may look like essential tremor although may also be much larger in amplitude (wing-beating tremor). Some patients present with rigidity and bradykinesia or ataxia rather than tremor. Others present with an acute hepatitis. Clearly in a young adult multiple sclerosis (MS) is the other differential to exclude neurologically (MS plaques in the cerebellum are not uncommon), and the give-away here is the corneal findings described (they could give you a picture of them), which are typical of Wilson's. The rigidity is also an extrapyramidal feature that might steer you away from multiple sclerosis (although of course, basal ganglia plaques would not be impossible).

The pathophysiology is that there is a failure of hepatic excretion of copper into bile, therefore copper accumulates in the liver and secondarily suppresses the synthesis of caeruloplasmin (a coppercarrying blood protein). Eventually, copper spills over into the circulation and deposits also in the:

- basal ganglia - with neurological sequelae
- kidneys - causing tubular degeneration and thus a Fanconi syndrome (aminoaciduria, glycosuria, phosphaturia, renal tubular acidosis type 2 (proximal), rickets (children) or osteomalacia (adults))
- cornea - with Kayser-Fleischer rings
- bones and joints – erosions
- parathyroid glands - altered calcium metabolism

Look out for any of these features in a stem.

The first test to pick in an exam stem if you suspect Wilson's disease should be caeruloplasmin, which will be low (<20 mg/dl), and then an elevated urine 24-hour copper excretion (>40 micrograms/ml). If the diagnosis is still not certain, the liver biopsy can be useful: showing increased hepatic copper levels. However, in reality, these changes can be non-specific on their own therefore interpretation is made on their combination alongside the clinical features.

Treatment is with the copper-chelating trientine or penicillamine.

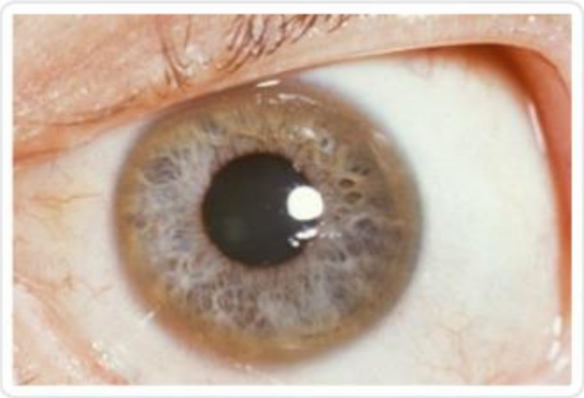
Wilson's disease.

Wilson's disease is an autosomal recessive disorder characterised by excessive copper deposition in the tissues. Metabolic abnormalities include increased copper absorption from the small intestine and decreased hepatic copper excretion. Wilson's disease is caused by a defect in the ATP7B gene located on chromosome 13.

The onset of symptoms is usually between 10 - 25 years. Children usually present with liver disease whereas the first sign of disease in young adults is often neurological disease

Features result from excessive copper deposition in the tissues, especially the brain, liver and cornea:

- liver: hepatitis, cirrhosis
- neurological:
    - basal ganglia degeneration: in the brain, most copper is deposited in the basal ganglia, particularly in the putamen and globus pallidus
    - speech, behavioural and psychiatric problems are often the first manifestations
    - also: asterixis, chorea, dementia, parkinsonism
- Kayser-Fleischer rings
    - green-brown rings in the periphery of the iris
    - due to copper accumulation in Descemet membrane
    - present in around 50% of patients with isolated hepatic Wilson's disease and 90% who have neurological involvement
- renal tubular acidosis (esp. Fanconi syndrome)
- haemolysis
- blue nails



Investigations

- slit lamp examination for Kayser-Fleischer rings
- reduced serum caeruloplasmin
- reduced total serum copper (counter-intuitive, but 95% of plasma copper is carried by ceruloplasmin)
    - free (non-ceruloplasmin-bound) serum copper is increased
- increased 24hr urinary copper excretion
- the diagnosis is confirmed by genetic analysis of the ATP7B gene

Management

- penicillamine (chelates copper) has been the traditional first-line treatment
- trientine hydrochloride is an alternative chelating agent which may become first-line treatment in the future
- tetrathiomolybdate is a newer agent that is currently under investigation

Question 11 of 259

A 44-year-old woman was referred to the gastroenterology clinic with a 6-month history of diarrhoea, weight loss, and fatigue. She was diagnosed with scleroderma 10 years ago, but took no medications for this. She ate a varied diet, and no particular foods exacerbated her symptoms. On examination you noted skin bruising and a peripheral neuropathy in a glove-and-stocking distribution.

Investigations:

Hb	108g/l (115-165)
MCV	103fl (80-96)
WBC	10.2 * 10 /l (4.0-11.0)9
Serum vit B12	140ng/l (160-760)
Serum folate	10.5µg/l (2.0-11.0)
CRP	130mg/l (<10)
Anti TTG antibodies	negative

Which would be the most appropriate first line investigation to make a diagnosis?

A. Faecal calprotectin
B. Hydrogen breath testing
C. Flexible sigmoidoscopy
D. SeHCAT scan
E. Faecal elastase test

B. Hydrogen breath testing

Hydrogen breath testing is an appropriate first line test for diagnosis of small bowel overgrowth syndrome

When there is an increase in the bacterial colonization of the small bowel proximal to the distal ileum, it is referred to as small bowel bacterial overgrowth (SBBO). Patients can present with symptoms which include weight loss, abdominal pain, diarrhoea, and malnutrition. They may also exhibit neurological symptoms associated with vitamin B12 malabsorption and impaired absorption of fat-soluble vitamins (A, D, E, and K). Hydrogen breath testing is a more commonly used first-line investigation for making diagnosis of small bowel bacterial overgrowth syndrome. Treatment of SBBO includes surgical correction of the underlying abnormality, antibiotics (metronidazole is commonly used), and treatment of dysmotility.

The other options'faecal calprotectin, flexible sigmoidoscopy, and SeHCAT scan'may be useful to exclude other diagnoses, such as inflammatory bowel disease, microscopic colitis, and bile acid malabsorption, respectively. The faecal elastase test is used along with other tests, such as fecal fat and/or fecal chymotrypsin, to evaluate both children and adults for pancreatic insufficiency.

Question 12 of 259

A 52 year-old man presents with a 3 month history of impotence, joint pains and malaise. His past medical history includes type 2 diabetes mellitus and hypertension. His current medication includes metformin, ramipril and amlodipine. He has a 30 pack-year history and drinks 15-20 units per week. His occupation is as a prison warden.

On examination of his abdomen, he has a palpable 2cm non-tender live edge. Otherwise heart sounds 1 and 2 are presents with no added sounds, his pulse in regular and his lung fields are clear.

Blood tests reveal:

Bilirubin	20 µmol/l
ALP	462 u/l
ALT	79 u/l
γGT	98 u/l
Albumin	37 g/l

What is the most appropriate diagnostic investigation?

A. Serum ferritin
B. Transferrin saturation index
C. Caeruloplasmin
D. Urinary copper
E. MRCP

B. Transferrin saturation index

Screening for haemochromatosis

general population: transferrin saturation > ferritin

family members: HFE genetic testing

The most likely diagnosis in this patient is haemochromatosis, an autosomal recessive disease that presents with bronzed pigmentation, joint pain, impotence and fatigue. Late signs can include cirrhosis of the liver and diabetes. A fasting transferrin saturation of greater than 55% indicates iron accumulation and along with genotyping for the HFE gene, this would be the most appropriate investigation to establish the diagnosis of the disease.

Question 13 of 259

A 42-year-old man attends gastroenterology clinic after being referred by his GP for assessment of deranged liver function tests. The patient denied experiencing any specific symptoms, but on close questioning revealed that he had been feeling increasingly lethargic and sleepy during his daily activities. He also reported suffering from intermittent generalised joint pains. The patient had not drawn a connection between these symptoms, attributing them all to getting older and the demands of his busy life at work and at home. Nevertheless, the patient had attended his GP for a 'health check' and abnormalities were noted on a set of screening blood tests.

The patient's past medical history was generally unremarkable, significant only for a laparoscopic cholecystectomy performed 4 years previously as a treatment for recurrent biliary colic. The patient also stated that he had been told by his GP that his recent of blood tests showed that he suffered from 'borderline diabetes', but that he had not started any treatment for this at the present time. The patient took no regular or over the counter medications and had no known drug allergies.

The patient had been adopted as a young child and reported to not having much information about the medical history of his biological relatives. As an adult, he had briefly made contact with his biological father until his father had died as a result of complications of liver disease, but the patient stated he did not know the cause of his father's condition. The patient was a former cigarette smoker who occasionally consumed moderate amounts of alcohol. He had never injected drugs, had a tattoo or received a blood transfusion. The patient lived with his wife and 3 young children and ran his own building company.

General examination of the patient indicated a caucasian middle-aged man who appeared tanned and slightly under-weight. Abdominal examination was significant only for 5 possible spider naevi on the patient's upper chest. There was no palpable hepatomegaly or splenomegaly. Examination of the cardiovascular and respiratory systems was unremarkable. Please see below for investigation results arrange before and after the patient's clinic attendance.

Haemoglobin	136 g / dL
Mean cell volume	89.1 fl
White cell count	6.5 x 10>3 / microlitre
Platelets	385 x 10>3 / microlitre
Urea	6.4 mmol / L
Creatinine	91 micromol / L
eGFR	89 ml / min / 1.73 m2
Sodium	145 mmol / L
Potassium	4.3 mmol / L
Albumin	48 g / L (reference 35-50)
Alkaline phosphatase	124 U / L (reference 35-100)
ALT	275 U / L (reference 3-36)
Bilirubin	23 micromol / L (reference < 26)
Total protein	68 g / L (reference 60-80)
Ferritin	1,290 microgram / L (30-300)
Transferrin saturation	87 % (15-45)

HbA1C	46 mmol / mol (reference < 42)
HFE gene analysis	C282Y homozygous

What is the appropriate initial management of the patient's condition?

A. Phlebotomy: 500 ml every 2 days
B. Phlebotomy: 500 ml every 2 week
C. Phlebotomy: 500 ml every 2 months
D. Watchful waiting: phlebotomy once ferritin > 2,000 microgram / L
E. Watchful waiting: phlebotomy once ALT > 1,000 U / L

B. Phlebotomy: 500 ml every 2 week'

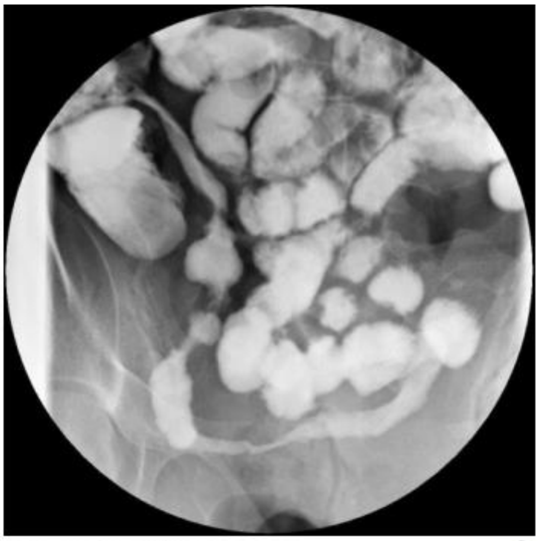
The patient has a non-specific clinical presentation typical for hereditary haemochromatosis. In particular, note the suggestion of the early stages of classical 'bronze diabetes': diabetes, skin pigmentation and liver cirrhosis. The clinical picture is supported by the elevated ferritin levels and elevated transferrin saturation. The diagnosis is confirmed by the patient being homozygous for the C282Y mutation of the HFE gene on chromosome 6 (the most common genotype associated with iron overload). The patient would also require additional investigation to assess for endorgan damage; for example, electrocardiography, echocardiography and liver biopsy or liver MRI.

Phlebotomy is the mainstay of treatment in hereditary haemochromatosis and is indicated in all patients with a ferritin level greater than 1,000 microgram / L. Typical initial phlebotomy regimes are 400-500 ml every 1-2 weeks. Once ferritin level falls to the range 50-100 microgram / L, phlebotomy regime can transition to a maintenance schedule every 2-4 months. Please note, that the incorrect answer of 500 ml phlebotomy every 2 days would lead to the patient becoming dangerously anaemic!

Question 14 of 259

A 27-year-old man is referred to the gastroenterology clinic due to a 4 month history of diarrhoea. He is now passing around 6 loose non-bloody stools per day. This has been associated with weight loss of 5 kg. He suffers from colicky abdominal pains, particularly after eating.

A barium study is shown below:



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What is the most likely diagnosis?

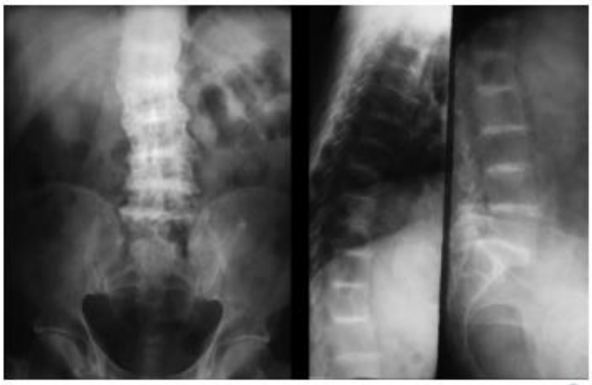
A. Volvulus
B. Coeliac disease
C. Crohn's disease
D. Ulcerative colitis
E. Irritable bowel syndrome

C. Crohn's disease

The barium study is consistent with a diagnosis of Crohn's disease. There is a long segment of narrowed terminal ileum in a 'string like' configuration in keeping with a long stricture segment. Termed 'Kantor's string sign'.

Question 15 of 259

A 41-year-old man presents with lower back pain. The pain has been getting gradually worse over the past nine months and is located in the lower lumbar spine. Other than a slightly pigmented sclera no other abnormalities are found on examination. A number of x-rays are obtained:



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What is the most likely diagnosis?

A. Fabry's disease
B. Alkaptonuria
C. Homocystinuria

D. Osteopetrosis
E. Ankylosing spondylitis

B. Alkaptonuria

The x-rays show multi-level intervertebral disc calcification with disc space narrowing.

Question 16 of 259

A 22-year-old man presents with a tremor and the feeling of 'slowing down'. Lately, the patient has noticed that he is feeling more nauseated, has pain in his abdomen, worse around the right upper quadrant. His friends have commented that his eyes have changed colour and are turning yellow.

Which investigation result is most supportive of a diagnosis of Wilson's?

A. Elevated ceruloplasmin
B. Elevated 24-hour urinary copper excretion
C. Elevated serum copper
D. Elevated transaminases
E. Elevated pro-thrombin time

B. Elevated 24-hour urinary copper excretion

The correct answer is elevated 24-hour urinary copper excretion. Whilst serum copper and serum ceruloplasmin levels are useful in the diagnosis of Wilson's disease they are expected to be lowered rather than elevated. It would be very likely that the transaminases would be elevated in this patient as he has right upper quadrant pain and jaundice, but this is not specific for Wilson's disease. It would be unlikely that hepatic function would be sufficiently impaired to affect prothrombin time for this patient, and even if this was the case it is not specific for Wilson's disease.

Question 17 of 259

A 42-year-old man presents to the emergency department due to bleeding from hair follicles on his head. He has also noticed that he has been off his food and losing weight. He is currently homeless after having been made redundant from his job one year ago. He is alcohol dependent with 30-units per week of hard liquor. He is not confused.

He has a past medical history of tuberculosis, for which he completed treatment six months. This treatment finished two months ago. On examination, he appears unkempt with poor oral hygiene and gingivitis. He has global muscle weakness rated at 4+/5 on the MRC scale. There is no sensory impairment.

What is the most likely diagnosis?

A. Pellagra
B. Scurvy
C. Beriberi
D. Wernicke-Korsakoff syndrome
E. Recurrence of tuberculosis

B. Scurvy

The correct answer is scurvy. He is a homeless and alcohol-dependent patient who is very likely to have a very poor diet. The combination of bleeding hair follicles and gingivitis is highly suggestive of vitamin C (ascorbic acid) deficiency. Pellagra is associated with diarrhoea and dementia. Beriberi can be associated with peripheral neuropathy with sensory involvement, or heart failure and oedema. Neither of these cases is described here. The classical triad of nystagmus, ophthalmoplegia and ataxia with confusion is not described, making Wernicke-Korsakoff syndrome less likely. The absence of a cough, haemoptysis and night sweats makes TB recurrence less likely.

Question 18 of 259

A 60-year-old female patient is receiving etanercept for long term rheumatoid arthritis. She attends the general medical clinic with increasing shortness of breath on exertion. She has received treatment for arthritis for many years and has in the past received methotrexate. This was stopped due recurrent infections. She has had no recent courses of steroids. Her symptoms of arthritis are well controlled on her current treatment regimen.

On examination the patient had a blunted affect. Her tongue appeared mildly swollen and red with some area of soreness at the corners of her mouth. She did not exhibit finger clubbing but the nails appeared brittle. Her pulse was 92 bpm and her blood pressure 106/68 mmHg. Examination of her cardiorespiratory system revealed normal heart sounds with no added sounds and the occasional fine crepitation in her lung fields.

Basic investigations such as blood tests and a chest x-ray were undertaken. Results are shown below.

Hb	8.2 g/dl
MCV	109fl
Haematocrit	0.39
Platelets	380 * 10 /l9
WBC	9.4 * 10 /l9
Renal function	normal
Liver function	normal
CXR	normal

What is the most likely diagnosis?

A. Pernicious anaemia
B. Sideroblastic anaemia
C. Anaemia of chronic disease
D. Iron deficiency anaemia
E. Aplastic anaemia

A. Pernicious anaemia

Autoimmune diseases, in this case pernicious anaemia, are more likely in the presence of another autoimmune disease (rheumatoid arthritis). Pernicious anaemia is an autoimmune condition that targets the parietal cells of the gastric lining, the role of which is to produce intrinsic factor. Intrinsic factor is responsible for the absorption of vitamin B12. In this case, a lack of B12 has led to a macrocytic anaemia as seen with the high MCV. To diagnose pernicious anaemia blood can be tested for parietal cell antibodies and intrinsic factor antibodies. These supersede the classical test Schilling Test.

The patient in this case demonstrated glossitis and angular chelitis with brittle nails that are associated with this condition. Her blunted affect is a sign of mild cognitive impairment that can also be present. The signs and symptoms of pernicious anaemia are vast and there frequency in clinical practice can be varied. Often the only presentation is with the fatigue associated with the anaemia.

It should be noted that another treatment for rheumatoid arthritis, Methotrexate, can induce another type of macrocytic anaemia. This is due to it inhibiting DNA replication in the same way as heavy metals such as lead.

Question 19 of 259

A 30-year-old woman is referred to haematology clinic by her GP for assessment for possible hereditary haemochromatosis. The patient reports that her 37-year-old brother has recently been diagnosed with the condition after developing progressive fatigue and joint pains over the previous few months. The patient is very concerned that she too may have inherited the condition, especially as at the time of his diagnosis her brother has been found to have significant damage to his liver and diabetes secondary to iron overload. With her brother's consent, the patient had brought a copy of his medical records to the clinic appointment and details of his investigations are given below.

Aside from the anxiety induced by her brother's recent illness, the patient reported being in generally good health. She reported leading a busy life, working full time as a primary school teacher in addition to raising her 4-year-old daughter. In the context of these commitments and activities, the patient did not feel she was unreasonably tired or lethargic and did not experience significant joint pains.

The patient had a history of psoriasis that was well controlled with topical treatments. The pregnancy and delivery of her daughter had been uncomplicated. The patient reported a tendency to a heavy menstrual flow and had previously been prescribed tranexamic acid for this by her GP although she had not found significant benefit from this treatment. Aside from topical treatments for her psoriasis, the patient took no regular, prescribed medications and had no known allergies to medications.

Physical examination of the patient was unremarkable; in particular, no signs of chronic liver disease were identified on gastrointestinal system examination.

A summary of the patient's brothers investigations for hereditary haemochromatosis at the time of his diagnosis is given below.

Ferritin	1,527 microgram / L (30-300)
Transferrin saturation	78 % (15-45)
HFE gene analysis	homozygous for C282Y mutation
HbA1C	53 mmol / mol (reference < 42)
Liver magnetic resonance imaging	Generalised cirrhosis; low signal seen on all sequences, especially T2 weighted

The patient had undergone basic blood tests arranged by her GP at the time of referral, with results given below.

Haemoglobin	136 g / dL
Mean cell volume	84.1 fl
Ferritin	190 microgram / L (30-300)
Transferrin saturation	43 % / (15-45)

What is the appropriate next investigation to confirm or exclude the patient suffering from hereditary haemochromatosis?

A. Pituitary hormone panel
B. HFE gene analysis
C. Liver magnetic resonance imaging (T2 weighted)
D. Patient asymptomatic and normal iron studies, no further investigations indicated
E. Liver biopsy

B. HFE gene analysis

Screening for haemochromatosis

general population: transferrin saturation > ferritin

family members: HFE genetic testing

Hereditary haemochromatosis presents with nonspecific and insidious symptoms, with the result that in some cases, the diagnosis is not suspected until after damage to end-organs has occurred. In such individuals, the risk of death from liver cancer, cirrhosis, cardiomyopathy and diabetes are increased compared to the general population. However, detection and treatment of the condition in a susceptible individual prior to the development of cirrhosis or diabetes results in normal survival. Therefore, genetic testing for HFE gene mutations (specifically, the C282Y and H63D mutations) is indicated for an individuals meeting one of the following criteria:

Elevated serum ferritin (> 300 microgram / L in males; > 200 microgram / L in females)

Elevated transferrin saturation (> 45 %) First degree relative with haemochromatosis

Therefore, in the case of this patient, HFE gene analysis should be requested to confirm or exclude hereditary haemochromatosis. The patient's iron studies are at the upper end of normal, which is surprising for this patient in the context of her menorrhagia and raises suspicion of iron overload developing in the future. Women with haemochromatosis do not normally develop significant iron overload until after their menopause. In addition, the clinical penetrance of C282Y homozygosity is variable, with haemochromatosis associated morbidity developing in only 10-33 % of individuals with this genotype. Therefore, even in the absence of iron overload, it is helpful to be aware of the presence of HFE mutations to enable informed testing of an individual's children.

The other available answers are investigations to assess damage to end-organs that may be appropriate for an individual with a confirmed diagnosis of haemochromatosis. Of note, liver MRI is the most useful imaging technique for assessing hepatic iron overload and can quantify the degree of iron overload to guide phlebotomy regimes and avoid repeated liver biopsies.

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A 23-year-old student presented with a tremor in his right arm. He had also noticed increasing difficulty with speech, clumsiness in both hands and unsteadiness walking. He reported being very stressed and anxious. His relationship with his long-term girlfriend had come to an end and his younger brother had recently been diagnosed with bipolar affective disorder.

This student had a past medical history of depression and was born prematurely at 29 weeks gestation requiring ventilatory support in the neonatal intensive care unit. He took no regular medications and denied recreational drug use. He was, however, a binge drinker, consuming at least 30 units of alcohol per week. He had a penicillin allergy.

On examination there was a bilateral resting tremor more marked in the right arm than the left. The tone of the right arm was slightly increased. The movements of his upper limbs and hands were slow with reduced dexterity although there was no weakness. Reflexes were present and symmetrical. There was no sensory disturbance. On walking this patient had a broad-based, slow, ataxic gait with small steps. Rombergs test was negative. Power, tone, reflexes and sensation in the lower limbs were normal.

Investigations:

Hb	11.0 g/dl
Platelets	110 * 10 /l9
WBC	10.5 * 10 /l9
International Normalised Ratio	1.3
Na +	134 mmol/l
K +	4.0 mmol/l
Urea	5.8 mmol/l
Creatinine	78 µmol/l
Bilirubin	80 µmol/l
ALP	211 u/l
ALT	94 u/l
Amylase	72 u/l

Given the most likely underlying diagnosis which of the following would be the most appropriate management plan?

A. Start penicillamine as an inpatient and prescribe an alcohol detoxification regimen
B. Prescribe an alcohol detoxification regimen and refer to a geneticist
C. Start pentoxifylline and prescribe an alcohol detoxification regimen
D. Start trientine as an inpatient and prescribe an alcohol detoxification regimen
E. Refer to the psychiatric team

D. Start trientine as an inpatient and prescribe an alcohol detoxification regimen

This presentation (ataxia, movement disorder, psychiatric disturbance and deranged liver function tests in a young patient) is highly suggestive of Wilsons disease. Wilsons disease is an autosomal recessive disorder of copper metabolism. Deposition of copper in the liver, brain and eyes results in hepatic, neurological, psychiatric and ophthalmological manifestations as outlined below:

- Hepatic: acute hepatitis, steatosis, severe chronic liver disease, fulminant hepatic liver disease +/- haemolytic anaemia
- Neurological: asymmetrical tremor (can be postural or resting), dysarthria, pseudobulbar palsy, ataxia and gait disturbances
- Psychiatric: personality change, severe depression, neurotic behaviours
- Ophthalmological: Kayser-Fleischer rings, sunflower cataracts, night blindness, optic neuritis
- Renal: Fanconi syndrome, nephrocalcinosis
- Cardiac: arrhythmias

A diagnosis of Wilsons disease is supported biochemically by a low serum caeruloplasmin, elevated 24 hour urinary copper excretion and increased hepatic parenchymal copper concentration on liver biopsy.

First line pharmacological treatment aims to prevent copper absorption by using chelating agents. Penicillamine is a potential first line treatment however, in this case, there is a high risk of a hypersensitivity reaction given this patients history of penicillin allergy.

Trientine is now a popular first line treatment of neurological and hepatic disease in Wilsons as it has a better side-effect profile than penicillamine. In this patient with a penicillin allergy it would certainly be the treatment of choice.

Zinc can be used for maintenance treatment following initial chelation therapy or during pregnancy.

This patient should be advised to eat a low copper diet and avoid alcohol as this worsens hepatotoxicity.

Referral to geneticist may be required later on but does not contribute to the acute management of this patient. There are several mutations that can cause Wilsons disease, all of which are of low prevalence, and therefore genetic testing is not routinely used in clinical practice.

Pentoxifylline is a treatment for alcoholic hepatitis not Wilsons disease.

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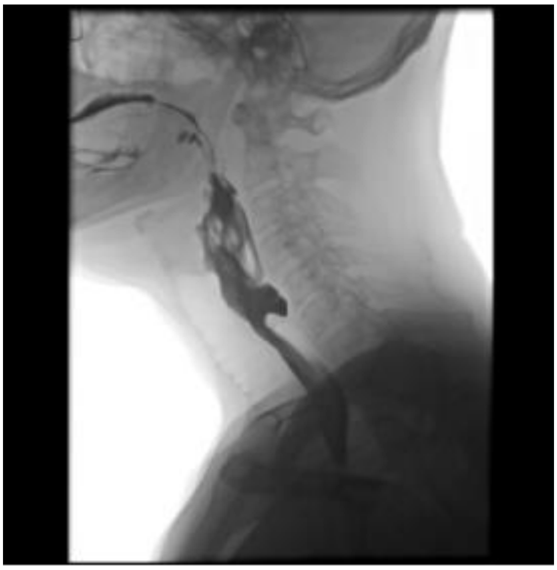
A 71-year-old man presents with dysphagia which has been getting progressively worse for the past few months. He is generally fit and well other than a history of chronic obstructive pulmonary disease. On occasions he has regurgitated some food after eating a large meal.

On examination of his abdomen, no masses are noted. A barium swallow with fluoroscopy is arranged:



What is the most likely diagnosis?

A. Cystic hygroma
B. Pharyngeal pouch
C. Thyroglossal cyst
D. Oesophageal cancer
E. Achalasia

B. Pharyngeal pouch

During swallowing an outpouching of the posterior hypopharyngeal wall is visualised at the level C5-C6, right above the upper oesophageal sphincter.

Question 22 of 259

An 18-year-old male is brought into the Emergency Department by police with facial injuries following an altercation with a work colleague. They are accompanied by his tearful mother, who reports that her son has developed violent mood swings and become increasingly aggressive over the last 6 months. There is no relevant past medical history.

On examination, the patient is mildly dysarthric and tearful but denies drinking alcohol. His observations are normal and examination of the precordium is unremarkable. His abdomen is soft and non-tender. Peripheral neurological examination reveals the presence of mildly increased tone, although there is no weakness. A 'wing-beating tremor' is also observed.

Blood tests are as follows:

Hb	132 g/l	Na +	143 mmol/l	Bilirubin	23 µmol/l
Platelets	189* 10 /l 9	K +	4.2 mmol/l	ALP	189 u/l
WBC	7.6* 10 /l 9	Urea	4.1 mmol/l	ALT	342 u/l
Neuts	5.3* 10 /l 9	Creatinine	76 µmol/l	γGT	122 u/l
Lymphs	2.1* 10 /l 9	Albumin	43 g/l		
Eosin	0.3* 10 /l9				

Given the likely diagnosis, which of the following investigations should be performed next?

A. 24-hour urinary copper
B. Serum caeruloplasmin
C. Serum ferritin and transferrin saturations
D. Anti-LKM antibody
E. Viral hepatitis screen

B. Serum caeruloplasmin

The combination of neuropsychiatric features and deranged LFTs should alert you to a possible diagnosis of Wilson's disease. Caeruloplasmin is the most appropriate investigation as extremely low levels provide strong evidence of Wilson's disease and are considered diagnostic if associated with Keyser-Fleischer rings. 24-hour urinary copper is useful in confirming the diagnosis but is more difficult to perform and would not normally be requested first-line.

Serum ferritin and transferrin saturations are used to diagnose haemochromatosis, whilst anti-LKM antibodies are found in autoimmune hepatitis type 2. None of the final 3 options are likely

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A 24-year-old Greek man presented after his partner reported a personality change over the past three weeks. She reported that he had been very low in mood, and had been behaving strangely. He reported being a little fatigued over the past couple of weeks. Neurological examination was unremarkable other than a rest tremor in his right hand. There was evidence of scleral icterus, and the liver edge was just palpable below the costal margin.

Initial blood results are shown below:

Haemoglobin	98 g/L
Platelets	156 x 10^9/l
Mean corpuscular volume	89 fl
White cell count	6.8 x 10^9/l
Reticulocyte count	5%
Alkaline phosphatase	142 U/L
Bilirubin	55 mol/L
Alanine transaminase	105 U/L
Albumin	35 g/L

Which investigation is most likely to reveal the underlying cause of his anaemia?

A. Direct antiglobulin test
B. 24-hour urinary copper excretion
C. Vitamin B12 level
D. Iron profile
E. Haemoglobin electrophoresis

B. 24-hour urinary copper excretion

The combination of neurological and hepatic disease in young adults raises suspicion of Wilson's disease. Personality change and movement disorders (such as tremor) are common presenting features of Wilson's disease.

Haemolytic anaemia occurs in 10 to 15% of patients with Wilson's disease and accounts for the anaemia in this case, as indicated by the normocytic anaemia with reticulocytosis. As such 24-hour urinary copper is the investigation most likely to reveal the underlying diagnosis in this case. The direct antiglobulin test (DAT) is positive in antibody-mediated haemolytic anaemia (such as warm and cold haemolytic anaemia, haemolytic disease of the new born, and haemolytic transfusion reactions). In Wilson's disease haemolysis occurs due to excess copper rather than antibodies, so the DAT does not give a positive result.

Vitamin B12 and iron deficiency would not account for the combination of liver and neurological features seen in this case, and haemoglobin electrophoresis is used in the diagnosis of haemoglobinopathies, of which there is no evidence.

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A 45-year-old man is reviewed routinely in the hepatology clinic. He has a past medical history of chronic hepatitis B infection. He takes tenofovir. He does not smoke or drink alcohol.

The examination is unremarkable. Blood tests:

Bilirubin	12 µmol/L	(3 - 17)
ALP	88 u/L	(30 - 100)
ALT	88 u/L	(3 - 40)
Î³GT	44 u/L	(8 - 60)
Albumin	36 g/L	(35 - 50)

Further testing:

Hepatitis B DNA	2500 IU/ml	(not detected)
Hepatitis B surface antigen	positive	(not detected)
Hepatitis B core antibody	positive	(not detected)
Hepatitis B e antigen	positive	(not detected)
Hepatitis B surface antibody	negative	(negative)

Given the clinical context, which parameter is the best marker in determining the risk of developing cirrhosis?

A. ALT level
B. Hepatitis B DNA level
C. Hepatitis B core antibody
D. Hepatitis B e antigen
E. Hepatitis B surface antigen

B. Hepatitis B DNA level

Hepatitis B DNA levels are the best marker for determining the risk of developing cirrhosis

Hepatitis B DNA level is the correct answer. The patient has chronic hepatitis B. The viral load i.e. hepatitis B DNA level is positive and suggests ongoing viral activity. The viral load is the best marker in determining the risk of developing cirrhosis.

ALT is incorrect. This is a marker of liver inflammation but is inferior to the HBV DNA level in determining the risk of cirrhosis.

Hepatitis B core antibody is incorrect. This suggests that the patient has caught hepatitis B at some point in time. It is not used to quantify the risk of developing cirrhosis.

Hepatitis B e antigen is incorrect. This is a marker of infectivity and correlates with HBV DNA levels but is less accurate in predicting the risk of cirrhosis than HBV DNA levels.

Hepatitis B surface antigen is incorrect. HBsAg being positive implies ongoing infection, either acute or chronic (if present > 6 months). It is less accurate than HBV DNA in predicting those who will develop cirrhosis.

Hepatitis B serology

Interpreting hepatitis B serology is a dying art form which still occurs at regular intervals in medical exams. It is important to remember a few key facts:

- surface antigen (HBsAg) is the first marker to appear and causes the production of anti-HBs
- HBsAg normally implies acute disease (present for 1-6 months)
- if HBsAg is present for > 6 months then this implies chronic disease (i.e. Infective)
- Anti-HBs implies immunity (either exposure or immunisation). It is negative in chronic disease
- Anti-HBc implies previous (or current) infection. IgM anti-HBc appears during acute or recent hepatitis B infection and is present for about 6 months. IgG anti-HBc persists
- HbeAg results from breakdown of core antigen from infected liver cells as is, therefore, a marker of infectivity. Marker of HBV replication and infectivity

Example results

- previous immunisation: anti-HBs positive, all others negative
- previous hepatitis B (> 6 months ago), not a carrier: anti-HBc positive, HBsAg negative
- previous hepatitis B, now a carrier: anti-HBc positive, HBsAg positive

Question 1 of 235

You review a 58-year-old woman in gastroenterology clinic who has had 3 months of diarrhoea and weight loss. She complains of going to the toilet up to 7-8 times a day and has lost 1 stone in weight over this 3 month period. She denies any blood or mucus in her stools. Her past medical history includes osteoarthritis of the hands and anxiety. Her drug history includes sertraline which was started 4 months ago by her general practitioner and ibuprofen with paracetamol taken when required for pain.

Examination of the abdomen reveals mild diffuse tenderness but with no guarding or rigidity and no mass palpable.

Investigations have been undertaken revealing the following:

Hb	117 g/L	Female: (115 - 160)
Platelets	375 * 10 /L 9	(150 - 400)
WBC	10.2 * 10 /L 9	(4.0 - 11.0)
Na +	137 mmol/L	(135 - 145)
K +	3.6 mmol/L	(3.5 - 5.0)
Urea	5.8 mmol/L	(2.0 - 7.0)
Creatinine	102 µmol/L	(55 - 120)
CRP	32 mg/L	(< 5)
Colonoscopy	Good views of the large bowel up to the normal appearance of the large colon, no polyps identified, no cause for symptoms. Biopsies were taken and sent for histology	
Histology	Lymphocytic infiltrate within the intestinal villi.	

What is the most important management step in view of the likely underlying condition?

A. Stopping the sertraline
B. Gluten free diet
C. Loperamide
D. Oral prednisolone
E. IV hydrocortisone

A. Stopping the sertraline

SSRIs are linked to the development of microscopic colitis

Based on the symptoms, normal macroscopic appearance of the colon and biopsy results, this lady has likely developed microscopic colitis which has been known to be linked to selective serotonin reuptake inhibitors and non-steroidal anti-inflammatory analgesia.

The most important management step would thus be to cease the use of these medications, as the onset of sertraline has seemed to correlate to her symptoms.

A gluten-free diet would be appropriate if coeliac disease was suspected and only once it had been diagnosed. Coeliac disease is a possibility and additional investigation may be indicated to exclude this. However, in this case, we are told that the symptoms are correlating with the onset of sertraline and the histology supports a diagnosis of microscopic colitis.

Loperamide would only be of symptomatic benefit and not resolve the underlying cause of the diarrhoea.

Oral prednisolone and IV hydrocortisone are indicated in acute flares of macroscopic colitis such as ulcerative colitis or Crohn's colitis. In this case, the most important management step would be to stop the offending agent which could lead to a complete resolution of the microscopic colitis.

Question 2 of 235

A 16-year-old male presents to his GP with a four-month history of yellowing of the eyes. He reports no other associated symptoms and is otherwise well. He has a family history of jaundice but is unsure which family members are affected and whether said family members have been investigated. He takes no regular medications, herbal or over the counter remedies and has had no recent antibiotics. He is a non-smoker and occasionally drinks up to two pints of beer in an evening. He denies any recreational drug use and reports one previous female sexual partner. He has recently travelled to Spain.

Clinical examination is unremarkable aside from scleral jaundice. His GP performs a set of routine blood tests to investigate, results show:

Hb 145 g/L	Male: (135-180)
Platelets 200 * 10 /L 9	(150 - 400)
WBC 6.5 * 10 /L 9	(4.0 - 11.0)
Na 136 mmol/L +	(135 - 145)
K 4.0 mmol/L +	(3.5 - 5.0)
Urea 3.5 mmol/L	(2.0 - 7.0)
Creatinine 87 µmol/L	(55 - 120)
CRP 4 mg/L	(< 5)
Bilirubin 32 µmol/L	(3 - 17)
Conjugated Bilirubin 15 µmol/L	(1.0-5.1)
ALP 50 u/L	(30 - 100)
ALT 21 u/L	(3 - 40)
γGT 45 u/L	(8 - 60)
Albumin 37 g/L	(35 - 50)

An ultrasound abdomen shows no pathological features. What is the most likely underlying diagnosis?

A. Hepatitis A
B. Drug induced hepatitis
C. Dubin-Johnson syndrome
D. Gilbert syndrome
E. Crigler-Najjar syndrome

C. Dubin-Johnson syndrome

Dubin-Johnson Syndrome is characterised by a raised conjugated bilirubin

This patient has Dubin-Johnson syndrome, characterised by a raised conjugated bilirubin but otherwise normal liver function tests.

Drug-induced hepatitis and hepatitis A would both cause transaminitis in addition to raised bilirubin.

Gilbert syndrome and Crigler-Najjar syndrome both cause a raised unconjugated bilirubin.

Question 3 of 235

A 55-year-old man attends with lethargy and itch. He has a past medical of cirrhosis secondary to alcoholic liver disease. Observations are as follows: heart rate 95 beats per minute, blood pressure 125/75 mmHg, respiratory rate 20 breaths per minute, SpO2 95% on air, and temperature 37.2ºC. On examination he is euvolemic. Urinalysis is negative.

Blood results are as follows:

Hb	110 g/L	Male: (135-180)Female: (115 - 160)
Platelets	82 * 10 /L 9	(150 - 400)
WBC	5.9 * 10 /L 9	(4.0 - 11.0)
Neuts	4.8 * 10 /L 9	(2.0 - 7.0)
Lymphs	0.8 * 10 /L 9	(1.0 - 3.5)
Mono	0.2 * 10 /L 9	(0.2 - 0.8)
Eosin	0.1 * 10 /L 9	(0.0 - 0.4)
Bilirubin	48 µmol/L	(3 - 17)
ALP	142 u/L	(30 - 100)
ALT	82 u/L	(3 - 40)
Î³GT	168 u/L	(8 - 60)
Albumin	18 g/L	(35 - 50)
Na +	132 mmol/L	(135 - 145)
K +	3.5 mmol/L	(3.5 - 5.0)
Bicarbonate	18 mmol/L	(22 - 29)
Urea	24.2 mmol/L	(2.0 - 7.0)
Creatinine	268 µmol/L	(55 - 120)

Blood results from 4 weeks ago were as follows:

Na +	129 mmol/L	(135 - 145)
K +	3.6 mmol/L	(3.5 - 5.0)
Bicarbonate	20 mmol/L	(22 - 29)
Urea	22.2 mmol/L	(2.0 - 7.0)
Creatinine	242 µmol/L	(55 - 120)

What is the most likely diagnosis?

A. Acute interstitial nephritis
B. Acute tubular necrosis
C. Glomerulonephritis
D. Type 1 hepatorenal syndrome
E. Type 2 hepatorenal syndrome

E. Type 2 hepatorenal syndrome

Slowly worsening creatinine suggests a diagnosis of type 2 hepatorenal syndrome

Type 2 hepatorenal syndrome is correct. The history of cirrhosis and renal dysfunction suggests hepatorenal syndrome. The patient has not had a significant deterioration in their renal function over a 4-week period making type 2 hepatorenal syndrome the most likely diagnosis.

Acute interstitial nephritis is incorrect. This condition is characterised by acute inflammation of the renal tubulo-interstitium, commonly mediated by a hypersensitivity reaction to medications. The classic triad of clinical features includes rash, joint pain, and eosinophilia. In this case, the absence of these features makes it a less likely diagnosis.

Acute tubular necrosis is incorrect. Acute tubular necrosis (ATN) is common in cirrhotic patients, often as a consequence of sepsis, dehydration or drugs. The patient does not have any of these features to support ATN.

Glomerulonephritis is incorrect. The negative urinalysis result makes glomerulonephritis unlikely. This condition would tend to present with acute renal failure, hypertension, haematuria, and proteinuria.

Type 1 hepatorenal syndrome is incorrect. Type 1 hepatorenal syndrome is characterised by a rapid decline in renal function making this a less likely diagnosis in this case.

Question 4 of 235

A 64-year-old man comes to your clinic. He recently attended an endoscopy due to persistent symptoms of reflux.

The gastroscopy report is as follows:

Oesophagus: 3cm pink mucosa arising from the gastroesophageal junction. Biopsy taken. Stomach: patchy antral erythema. Normal body and fundus. CLO negative.

Duodenum: normal to second part. **Plan**: Review in clinic with histology

The histology is back and reported as follows:

Squamous epithelium and squamocolumnar junctional mucosal with reflux oesophagitis. Some goblet cells. Basal portions of the crypts show nuclei limited to the basal half of cell cytoplasm without loss of polarity in keeping with low-grade dysplasia.

Given the diagnosis, what is the preferred management option?

A. Endoscopic mucosal resection
B. Endoscopic surveillance
C. Oesophagectomy
D. Radiofrequency ablation
E. Regular low-dose omeprazole

D. Radiofrequency ablation

Barrett's oesophagus: radiofrequency ablation is the preferred treatment for low-grade dysplasia

The biopsy findings are in keeping with Barrett's oesophagus. The first stage is metaplasia, where there is intestinal epithelium lining the lower part of the oesophagus. The intestinal epithelium is made up of simple columnar cells and goblet cells. In this case, this epithelium type is found within the oesophagus and is indicative of intestinal metaplasia here. The report also details features of dysplasia - cytological changes, loss of polarity etc. You wouldn't be expected to know the specific features of high-grade vs low-grade dysplasia for MRCP, and so the stem clarifies that the findings are in keeping with low-grade dysplasia.

Radiofrequency ablation is the correct answer. This is the preferred choice of treatment for Barrett's oesophagus with low-grade dysplasia.

Endoscopic mucosal resection is incorrect. Although this can be used as a treatment option for Barrett's oesophagus with low-grade dysplasia, it would not be the first-line option.

Oesophagectomy is incorrect. This treatment option may be appropriate in cases of oesophageal cancer, however, there is no evidence of this on the histology for this patient.

Regular low-dose omeprazole is incorrect. High dose proton-pump-inhibitors can be used in the management of Barrett's oesophagus where there is metaplasia, but low-dose omeprazole is not specifically indicated.

Endoscopic surveillance is incorrect. This would be appropriate if the histology had shown metaplasia only, however, in this case, as there is evidence of dysplasia, it would not be the most appropriate option.

Question 5 of 235

A 28-year-old man presents to his general practitioner complaining of 2 years of diarrhoea and fatigue. His stools are pale, and difficult to flush away. He has recently returned from a 4-month holiday in Uganda. He does not take any regular medications.

On examination, he is of thin body habitus. The abdomen is soft. Blood tests:

Hb	124 g/L	Male: (135-180)Female: (115 - 160)
Platelets	189 * 10 /L $_9$	(150 - 400)
WBC	4.8 * 10 /L $_9$	(4.0 - 11.0)
Na $_+$	138 mmol/L	(135 - 145)
K $_+$	4.2 mmol/L	(3.5 - 5.0)
Urea	5.2 mmol/L	(2.0 - 7.0)
Creatinine	66 µmol/L	(55 - 120)
CRP	4 mg/L	(< 5)
Bilirubin	12 µmol/L	(3 - 17)
ALP	189 u/L	(30 - 100)
ALT	32 u/L	(3 - 40)
Î³GT	44 u/L	(8 - 60)
Albumin	35 g/L	(35 - 50)
Calcium	2.06 mmol/L	(2.20-2.60)
Vitamin D	13 nmol/L	(> 50)

What is the most likely diagnosis?

A. Coeliac disease
B. Giardiasis
C. Primary biliary cirrhosis
D. Ulcerative colitis
E. Vitamin D deficiency

A. Coeliac disease

Diarrhoea, fatigue, osteomalacia → ?coeliac disease

Coeliac disease is correct. The patient has chronic diarrhoea with likely steatorrhoea (pale stools that are difficult to flush) along with fatigue and biochemical evidence of osteomalacia (low vitamin D, raised ALP, low calcium). The unifying diagnosis is coeliac disease. Osteomalacia is thought to occur in this condition secondary to impaired intestinal absorption of vitamin D.

Giardiasis is incorrect. This is more common in tropical areas (but can occur worldwide) and is caused by infection with the microscopic parasite *Giardia lamblia* . It tends to cause explosive diarrhoea and is not classically associated with osteomalacia. Additionally, while it can occur worldwide, it is usually associated with recent travel. Symptoms for two years but travel only in the last 4 months for this patient suggests the travel and the symptoms are unlikely to be related.

Primary biliary cirrhosis is incorrect. This can cause a raised ALP and fatigue. However, it tends to occur in middle-aged women and does not cause diarrhoea and therefore is a less likely diagnosis based on this scenario.

Ulcerative colitis is incorrect. Vitamin D is principally absorbed in the small bowel and so ulcerative colitis (which affects the large bowel) is less implicated in impaired intestinal absorption. Furthermore, ulcerative colitis tends to cause abdominal pain and bloody diarrhoea, which are not mentioned here.

Vitamin D deficiency is incorrect. While this patient has biochemical evidence of osteomalacia, the unifying diagnosis is coeliac disease. Vitamin D deficiency alone does not cause diarrhoea.

Question 6 of 235

A 75-year-old male is admitted to ICU with severe pneumonia. He has a previous history of asthma. His regular medications include a beclometasone inhaler 2 puffs BD and salbutamol inhaler PRN. He has never smoked and drinks 10 units of alcohol per week. His CURB-65 score was 5 and he required intubation and sedation. On his 3rd post admission day in ICU, he is started on enteral feeding. His BMI on admission was 19. His serum electrolytes on admission were normal. The dietician reviews him and recommends checking his electrolytes every day to avoid refeeding syndrome.

Which of the following, if present, will be considered high risk for refeeding syndrome in this patient?

A. BMI of 19 kg/m2
B. Unintentional weight loss of 15% over 3-6 months
C. No nutritional intake for 2 days
D. Hyperphosphataemia prior to refeeding
E. Hyperkalaemia prior to refeeding

B. Unintentional weight loss of 15% over 3-6 months

Unintentional weight loss of more than 15% over 3-6 months: high risk of refeeding syndrome

B is the correct answer according to NICE guidelines.

Patients with a BMI of <16 kg/m2 and no nutritional intake for >10 days are considered a high risk for refeeding syndrome.

Patients with hypophosphataemia and hypokalemia are high risk as compared to those with hyperphosphataemia and hyperkalemia.

Question 7 of 235

A 45-year-old man presents to the gastroenterology clinic with a flare of ulcerative colitis. He reports that for the three weeks he has been experiencing bloody diarrhoea, with bowels opening up to 5 times per day. He denies any fevers, but has been feeling nauseous with reduced appetite and feels he has lost around 3kg in weight whilst unwell. He experienced a similar episode three months ago which settled with oral steroids and he is keen to try this again. He has been compliant with mesalazine orally and has continued his rectal mesalazine most nights. He has type 2 diabetes mellitus and takes metformin.

On examination, he is afebrile, basic observations are within normal limits and his abdomen is soft with moderate lower abdominal tenderness.

Blood tests show:

Hb	150 g/L	Male: (135-180)Female: (115 - 160)
Platelets	340 * 10 /L 9	(150 - 400)
WBC	12.0 * 10 /L 9	(4.0 - 11.0)
ESR	31 mm/hr	(<10)

An abdominal X-ray is unremarkable.

A flexible sigmoidoscopy is arranged for the following day and shows mild-moderate inflammation.

Given the clinical history, which of the following interventions should be considered for this patient in his acute flare?

A. Azathioprine
B. Prednisolone
C. Hydrocortisone
D. Surgical intervention
E. Infliximab

B. Prednisolone

If a mild-moderate flare of ulcerative colitis does not respond to topical or oral aminosalicylates then oral corticosteroids are added

This patient has a mild-moderate flare of ulcerative colitis and would benefit from a trial of oral steroids i.e. prednisolone.

If he had features of a moderate-severe flare e.g. tachycardia, fevers, drop in haemoglobin, bowels opening >6 times per day, ESR > 30 or changes on abdominal X-ray, then hydrocortisone would be preferable to prednisolone.

Azathioprine may be useful in the maintenance of remission but is not indicated in an acute flare.

Surgical intervention is not indicated until steroid therapy has failed or in severe flares with radiological changes e.g. toxic megacolon.

Infliximab is not indicated unless steroid therapy has failed.

Question 8 of 235

A 62-year-old woman was admitted to the hospital as the first presentation of decompensated liver disease. Her daughter confirmed excessive alcohol consumption. She was initially treated with lactulose, pabrinex, chlordiazepoxide and was commenced on diuretics.

An abdominal ultrasound was performed which showed evidence of liver cirrhosis with gross ascites. An aspirate of the ascitic fluid showed no evidence of spontaneous bacterial peritonitis.

She has been advised to reduce her alcohol intake with a plan to stop and has been informed that she will require ongoing screening due to the risks of developing liver cancer.

How often should screening take place?

A. Every 6 month
B. Every 12 months
C. Every 2 years
D. Every 3 years
E. Every 5 years

A. Every 6 month

Adults with cirrhosis should be screened for hepatocellular carcinoma by ultrasound (with or without measurement of serum alpha-fetoprotein) every 6 months

For patients diagnosed with liver cirrhosis, it is recommended to screen for hepatocellular carcinoma (HCC) every 6 months.

Annual screening is not appropriate for HCC in liver cirrhosis. An example of an annual screening programme instead would be diabetic eye screening patients over the age of 12 years.

The national bowel screening program is an example of a screening programme that takes place every 2 years, however, this would not be appropriate for HCC screening in the context of liver cirrhosis.

Again, 3 yearly is not an adequate frequency for HCC screening in this case. Note that cervical screening is offered 3 yearly for women aged 24 to 49 years of age.

For women aged 50 to 64 years of age, cervical screening is offered every 5 years. This is not frequent enough for screening HCC in liver cirrhosis.

Question 9 of 235

A 21-year-old female presents for the first time with crampy right iliac fossa pain and diarrhoea. Colonoscopy shows moderate ileocaecal disease, with the appearance of linear ulcerations and patchy erythema. She responds well to oral prednisolone and is discharged home with follow-up. On tapering the steroid dose, she feels that her abdominal pain gets worse and the frequency of diarrhoea increases. Her gastroenterologist performs a blood test showing:

Thiopurine methyltransferase (TPMT)	<10Mu/L
Normal range	68 - 150mu/L

What is the next best agent to offer?

A. Methotrexate
B. Azathioprine
C. Mercaptopurine
D. Infliximab
E. Increase prednisolone

A. Methotrexate

This patient presents with Crohn's disease. The initial monotherapy of prednisolone is effective at high dose but symptoms return on tapering its dose. An add-on treatment is therefore needed. The next agent to think about is azathioprine or mercaptopurine, in accordance with NICE guidance. Before either is commenced, a TPMT level must be done. If it is deficient, very low or absent, neither azathioprine or mercaptopurine should be offered.

Methotrexate is the next most suitable agent to add if the patient cannot tolerate mercaptopurine or azathioprine.

Infliximab is used only when patients have failed conventional therapy, including immunosuppressives and corticosteroids.

Question 10 of 235

A 55-year-old man presents to the emergency department with shortness of breath.

He reports several months of worsening diarrhoea and is now passing over 10 watery motions per day.

On examination he is dyspnoeic at rest. The blood pressure is 104 / 77mmHg and the pulse rate is 90bpm and regular. Oxygen saturations are 93% on room air. Chest auscultation reveals widespread mild polyphonic wheeze and bibasal crackles. Jugular venous pressure is elevated and C-V waves are present. There is a pansystolic murmur at the left lower sternal border louder in inspiration. There is a palpable liver edge 2cm below the costal margin.

Which is the most likely underlying heart valve pathology?

A. Infected platelet / fibrin deposits
B. Fibrous endocardial thickening
C. Sterile vegetations
D. Annular dilatation
E. Calcification

B. Fibrous endocardial thickening

A long history of diarrhoea along with signs consistent with tricuspid regurgitation is suggestive of carcinoid syndrome. This is a a paraneoplastic syndrome that is caused by endogenous secretion of serotonin (chiefly) from carcinoid tumours.

The vast majority of patients with cardiac involvement present with signs of right heart failure secondary to severe dysfunction of the tricuspid and / or pulmonary valves.

The characteristic pathological findings of carcinoid heart disease are endocardial plaques of fibrous tissue that may involve the tricuspid valve, pulmonary valve, cardiac chambers, venae cavae, pulmonary artery, and coronary sinus.

Sterile vegetations are associated with non-bacterial thrombotic endocarditis (NBTE), formerly known as marantic endocarditis.

Infected platelet / fibrin deposits are associated with infective endocarditis.

Annular dilatation is associated with 'functional' regurgitation caused by a distorted or dilated ventricle, displacing the papillary muscles.

Calcification is related to age-related valve disease.

Question 11 of 235

A 33-year-old female presents to urgent care centre complaining of 4 days of increasing lethargy and reduced exercise tolerance. In addition, she reports dark urine but no other symptoms. She normally works as a clerical member of staff in the same hospital. Her only past medical history if ulcerative colitis, which before a flare 2 weeks ago requiring the addition of sulphasalazine, had been well controlled with no immunosuppressants. On examination, you note conjunctival pallor bilaterally. Her abdomen is soft and non-tender. Her respiratory, cardiovascular and neurological examinations are unremarkable except for a mild systolic murmur and sinus tachycardia of 104 beats/ minute. Rectal examination was empty, no oral ulcers were noted.

Her blood tests are as follows:

Hb	89 g/l
MCV	85fl
Platelets	356 * 10 /l9
WBC	12.1 * 10 /l9
Blood film	Heinz bodies, reticulocytosis
CRP	30 mg/l
LDH	2400 u/l

What is the most appropriate immediate treatment?

A. Transfuse 2 units of packed red blood cells
B. Start intravenous hydrocortisone, stop sulphasalazine
C. Start intravenous hydrocortisone, continue sulphasalazine
D. Stop sulphasalazine only
E. Urgent colonoscopy

D. Stop sulphasalazine only

The diagnosis of the underlying cause of anaemia is in the blood film: the patient has Heinz bodies, small inclusion bodies in red blood cells that are a consequence of usually oxidative damage to haemoglobin components. Note the close temporal association of the anaemia with starting sulphasalazine. Drugs such as sulphasalazine, dapsone, ribavirin and poisoning with paraquat ingestion leads to the oxidation of Fe2+ (ferrous) components of haem to Fe3+ (ferric), forming methaemoglobin. When the intrinsic antioxidation mechanism of NADPH and glutathione is overwhelmed, red cell components undergo oxidative damage and cell death, leading to haemolysis, explaining the significantly raised LDH. Methaemoglobin is converted to hemichromes and eventually precipitated to Heinz bodies.

The treatment of oxidative haemolytic anaemia is by withdrawal of the offending drug alone. There is no role for steroids in this setting: the symptoms are not consistent with an ulcerative colitis flare. Blood abnormalities should normalise within weeks of drug withdrawal: transfusion of further red blood cells prior to stopping the drug would only result in further haemolysis.

Question 12 of 235

A 56-year-old male presents to the clinic with a 6 month history of progressive nausea and lethargy. He has a past medical history of diabetes which is poorly controlled. On examination you note jaundiced sclera and moderate ascites. He drinks approximately 20 units of alcohol per week.

Investigation results are as follows:

Hb	125 g/l	Na +	138 mmol/l	Bilirubin	96 µmol/l
Platelets	355 * 10 /l 9	K +	3.8 mmol/l	ALP	110 u/l
WBC	9.6 * 10 /l 9	Urea	6.8 mmol/l	ALT	210 u/l
Neuts	6.4 * 10 /l 9	Creatinine	76 µmol/l	AST	95 u/l
Lymphs	3.1 * 10 /l 9	Albumin	28 g/l		
Eosin	0.1 * 10 /l9				

An enhanced liver fibrosis (ELF) test was performed with the following results:

Enhanced liver fibrosis score	11.62 (high)

Current guidelines recommend that a PPAR-gamma agonist can be used to prevent further liver fibrosis.

What treatment will you consider prescribing?

A. Pioglitazone
B. Metformin
C. Fenofibrate
D. Atorvastatin
E. Prednisolone

A. Pioglitazone

Patients with NAFLD who have evidence of advanced fibrosis may benefit from pioglitazone

This patient has presented with decompensated liver failure as demonstrated by the low albumin, high bilirubin, and ascites. The history of diabetes and ALT>AST make non alcoholic fatty liver disease (NAFLD) the most likely diagnosis.

The advanced fibrosis tests is a new blood test which can quantify the degree of fibrosis via measurement of 3 markers which are elevated in fibrosis. A score of > 10.51 suggests severe fibrosis.

As per NICE guidelines for the management of NAFLD, pioglitazone or vitamin E can be used in cases of severe fibrosis, whether the patient has diabetes or not. Pioglitazone is a PPAR-gamma agonist.

Prednisolone may be of use for acute alcoholic hepatitis in severe cases with a Maddrey discriminant function (MDF) score ≥ 32. However, in this case, the modest weekly alcohol intake makes alcoholic hepatitis unlikely.

Fibrates are PPAR alpha agonists.

Question 13 of 235

A 48-year-old female presents with abdominal pain. She states that the pain is at the top of her abdomen. It is excruciatingly painful, sharp in character and radiates through to her back. On examination she is haemodynamically stable and has epigastric tenderness. Her past medical history includes ulcerative colitis and osteoarthritis. She cannot remember the medicines that she takes, however she states that she takes an oral drug for inflammatory bowel disease, and an over the counter medicine for arthritis.

Blood results are as follows:

Hb	136 g/l	Na +	138 mmol/l
Platelets	582 * 10 /l 9	K +	3.6 mmol/l
WBC	18.2 * 10 /l 9	Urea	8.6 mmol/l
Neuts	14.2 * 10 /l 9	Creatinine	62 µmol/l
Lymphs	2.2 * 10 /l 9	CRP	52 mg/l
Eosin	0.2 * 10 /l 9	Amylase	800 U/L (normal < 160)

What drug is most likely responsible for this presentation?

A. Naproxen
B. Ibuprofen
C. Mesalazine
D. Sulfasalazine
E. Paracetamol

C. Mesalazine

Mesalazine can cause drug-induced pancreatitis

Both mesalazine and sulfasalazine can cause drug induced pancreatitis, however the risk is much higher with mesalazine, making this the better option.

Naproxen and ibuprofen are NSAIDs which predispose to gastric ulceration. The history is more in keeping with pancreatitis rather than peptic ulcer disease. Paracetamol is not known to cause pancreatitis.

Question 14 of 235

A 78-year-old man comes to the GP with complaints of tiredness and epigastric pain. The symptoms began 9 months ago and have progressively worsened. The pain is intermittent, diffuse and rated 4 out of 10 in intensity. He appears pale and reports passing dark stools.

On examination, his pulse is 75/min, and blood pressure is 120/70 mmHg. There is epigastric tenderness. Upper GI endoscopy reveals a mass in the gastric antrum. The biopsy of the mass shows infiltrates of lymphoid cells. A CT scan of the chest and abdomen reveals normal lymph nodes.

Which of the following is the best management for this patient?

A. Amoxicillin, clarithromycin, and omeprazole
B. Biopsy of regional lymph nodes
C. Combination chemotherapy
D. Gastrectomy with gastrojejunostomy
E. Radiation therapy

A. Amoxicillin, clarithromycin, and omeprazole

Gastric MALT lymphoma - eradicate *H. pylori*

This patient's presentation and biopsy findings are indicative of gastric MALT lymphoma. Gastric MALT lymphoma is associated with *Helicobacter pylori* in most cases. It is treated by eradicating *Helicobacter pylori* with a triple-therapy regimen of a proton pump inhibitor (omeprazole), plus amoxicillin, and either clarithromycin or metronidazole. The triple therapy is taken orally for seven days.

Gastric MALT lymphoma is a slow-growing tumour. Most people have early-stage (localised) MALT lymphoma when they are diagnosed. Biopsy of regional lymph nodes does not provide essential information for the management of this condition.

MALT lymphoma has an excellent prognosis and responds well to antibiotic treatment. If antibiotic therapy fails, radiotherapy, rituximab therapy, chemotherapy, or surgery may be considered.

Question 15 of 235

A 64-year-old woman undergoes a gastroscopy for persistent dyspepsia over several months. She has had a few episodes of vomiting over this time and lost approximately 6kg over the past six months but her bowel movements are unchanged and she is otherwise asymptomatic. She has no relevant past medical history.

The histopathology report of an area biopsied during her gastroscopy is displayed below:

Histopathology of endoscopic gastric biopsy	Presence of dense diffuse infiltrate of marginal-zone cells in lamina propria with prominent lymphoepithelial lesions

What is the most appropriate treatment regimen given the diagnosis?

A. Endoscopic ablation
B. Endoscopic ligation
C. Gastrectomy
D. Omeprazole only
E. Omeprazole, clarithromycin, and amoxicillin

E. Omeprazole, clarithromycin, and amoxicillin

Gastric MALT lymphoma - eradicate *H. pylori*

This woman has biopsy-proven gastric MALT lymphoma. The first-line treatment is eradication of *Helicobacter pylori* which is associated with 95% of cases. The only option here that consists of a viable *Helicobacter pylori* eradication regimen is omeprazole, clarithromycin, and amoxicillin and therefore this is the correct answer.

Endoscopic ablation involves removing tissue as a therapeutic procedure during endoscopy. This is a common treatment for Barrett's oesophagus however is not used first-line for gastric MALT lymphomas and therefore is incorrect.

Endoscopic ligation is a therapeutic procedure for the treatment of varices. This involves applying bands to veins to reduce the risk of bleeding. Ligation is not used in the treatment of gastric MALT lymphomas.

A gastrectomy is a significant surgical procedure and is not usually required to treat gastric MALT lymphomas. Most show a good response to *Helicobacter pylori* eradication and therefore this is a more appropriate initial management plan.

Omeprazole alone may reduce the symptoms of a gastric MALT lymphoma but will not result in *Helicobacter pylori* eradication or the resolution of the lymphoma. Therefore, the addition of clarithromycin and amoxicillin is indicated in this instance and omeprazole alone is an incorrect answer.

Question 16 of 235

A 72-year-old man presents with increasing fatigue and generalised bone pain, worsening over the past few months. He is generally frail and housebound. He has a background of COPD and osteoarthritis.

On examination, he has moderate proximal muscle weakness and a waddling gait.

A number of blood tests are taken and the results are pending. X-rays of the femurs are taken and pseudofractures are noted.

Given the likely underlying cause, what is most likely?

A. High calcium, high phosphate, high alkaline phosphatase
B. Low calcium, high phosphate, high alkaline phosphatase
C. Low calcium, low phosphate, high alkaline phosphatase
D. Normal calcium, normal phosphate, high alkaline phosphatase
E. Normal calcium, normal phosphate, normal alkaline phosphatase

C. Low calcium, low phosphate, high alkaline phosphatase

In Vitamin D deficiency, there is usually low serum calcium, low serum phosphate and high serum alkaline phosphatase

The scenario here is most likely to be vitamin D deficiency, presenting with osteomalacia. The patient has a number of risk factors - his age, and being housebound and thus not receiving any sunlight exposure. The symptoms and examination findings support the likely diagnosis of vitamin D deficiency. With this deficiency, **calcium is therefore low, phosphate is also low, and alkaline phosphatase is usually raised**.

High calcium, phosphate and alkaline phosphatase is not referring to any specific pathology.

Low calcium, high phosphate and high alkaline phosphatase refers to secondary hyperparathyroidism. Whilst this is often due to vitamin D deficiency as well, the primary findings in the scenario above are those of osteomalacia.

Normal calcium, normal phosphate and high alkaline phosphatase is suggestive of Paget's disease. This would also present with bone pain, but may also lead to vision and hearing problems as skull bone thickens. Given that the history above mentions being housebound and a lack of sunlight exposure, as well as proximal weakness and waddling gait, osteomalacia due to vitamin D deficiency is more likely.

Normal calcium, normal phosphate and normal alkaline phosphatase may be seen in osteopenia, where bone mineral density is low. The history above is more suggestive of osteomalacia.

Question 17 of 235

A 51-year-old Italian farmer was referred to your clinic, having been found by his GP to have mildly abnormal LFTs during a routine health check. He is well in himself, with no change in bowel habit, nor any abdominal pain. On examination, you note mild scleral icterus.

His observations are as follows: blood pressure 130/66mmHg, heart rate 71 bpm, respiratory rate 14/min, pulse oximetry 97%, temperature 37.2ºC.

An ultrasound of his liver showed a cyst with a 'sand sign'. You requested a CT abdomen, the report for which is below.

CT abdomen: A cyst measuring 5cm in diameter can be seen in the right lobe of the liver. At the periphery of this cyst are two smaller cysts each measuring 1cm in diameter.

Which of the following would be the most useful in confirming the diagnosis?

A. Amoebic serology
B. Echinococcus granulosus serology
C. HIV test
D. Percutaneous aspiration
E. Tuberculin skin test

B. Echinococcus granulosus serology

Serology for *Echinococcus granulosus*can be useful in the investigation of suspected hydatid cysts

This man has developed multiple hepatic cysts. It is worth considering other causes, but the most likely in this situation is hydatid disease. The ultrasound showed 'sand sign', debris within the cyst moving freely and the CT scan suggests sister cysts. Both of these findings are characteristic of hydatid disease. The Mediterranean basin is an endemic area, plus he is a farmer. Both of these would be considered risk factors. Western blot serology for *Echinococcus granulosus* is the confirmatory test and is 80-100% sensitive and 88-96% specific. The cyst may be having a mass effect on the liver, causing obstructive jaundice in this man, and hence scleral icterus. Amoebic abscesses usually present as a single mass in the right lobe, and though there may be multiple cysts, this is less common. In addition to this, patients with amoebic abscesses typically have a fever and are more unwell. It is important to differentiate between amoebic abscesses and hydatid cysts as the management is very different. Hydatid cysts are treated with albendazole and usually require surgical excision; whereas amoebic abscesses are typically treated medically with metronidazole and tinidazole, followed by a luminal amoebicide to eradicate the cystic stage.

An HIV test might be suggested if you suspect fungal microabscesses, as these typically occur in immune-compromised individuals. However, the CT scan does not indicate microabscesses, and he has no other features to suggest that he may be immunocompromised.

There is little to suggest tuberculosis.

The percutaneous aspiration would in fact be contraindicated. The hydatid cyst contains allergens that can precipitate a type 1 hypersensitivity reaction, such as anaphylaxis. Plus, there is a risk of seeding the daughter cysts through the abdomen. During surgical excision, the cyst walls must not be ruptured.

Question 18 of 235

A 45-year-old man attends your clinic with a 7-month history of dysphagia. He describes food getting stuck in his throat and recurrent regurgitation of undigested food within 15 minutes of eating. This is associated with pain and weight loss.

His past medical history includes Coeliac disease and he does not take any regular medications.

Hb	111 g/L	Male: (135-180)Female: (115 - 160)
Platelets	400 * 10 /L 9	(150 - 400)
WBC	10.0 * 10 /L 9	(4.0 - 11.0)
Neuts	5.6 * 10 /L 9	(2.0 - 7.0)
Lymphs	2.2 * 10 /L 9	(1.0 - 3.5)
Mono	0.8 * 10 /L 9	(0.2 - 0.8)
Eosin	1.4 * 10 /L 9	(0.0 - 0.4)

A gastroscopy is undertaken and shows a proximal moderate-grade stricture and stacked circular rings in the oesophagus.

A biopsy is taken and reported as showing subepithelial and lamina propria fibrosis and inflammation with eosinophil microabscesses.

Given the likely diagnosis, what is the most appropriate first-line treatment?

A. Oesophageal stenting
B. Ranitidine
C. Sucralfate
D. Systemic steroids
E. Topical steroids

E. Topical steroids

Dietary modification and topical steroids are used in the management of eosinophilic oesophagitis

The most likely diagnosis here is eosinophilic oesophagitis (EO) as evidenced by the symptomatology, eosinophilia, gastroscopic and histological findings.

Topical steroids such as fluticasone and budesonide are the first-line treatments for EO. EO is poorly understood but thought to be a form of an allergic reaction occurring within the oesophagus in response to certain food types.

Ranitidine is incorrect. Acid suppression does form part of the first-line management of EO, however, omeprazole and lansoprazole would be the preferred initial options.

Sucralfateis a complex of aluminium and sulphated sucrose, it can be used in the management of duodenal and gastric ulcers as it forms a protective layer over the mucosa. It is not commonly used in the management of EO.

Systemic steroids can be considered in patients with very severe disease refractory to first-line treatment.

Oesophageal stenting is incorrect. This can be considered if there is no symptomatic improvement in dysphagia after 12 months of budesonide induction therapy. Stenting can be used as initial therapy in very high-grade strictures.

Question 19 of 235

A 32-year-old man presents with a two-week history of progressively worsening fatigue, anorexia, and joint pains. He reports itching but denies any past medical issues, illicit drug use, or drinking.

On examination, he is alert and oriented. Scleral icterus is noted. There are no spider naevi or palmar erythema present. His vital signs reveal a blood pressure of 100/76 mmHg, pulse rate of 88 bpm, and temperature of 37 ºC.

Laboratory investigations show the following results:

Hb	140 g/L	Male: (135-180)
Platelets	180 * 10 /L 9	(150 - 400)
WBC	7.4 * 10 /L 9	(4.0 - 11.0)
Bilirubin	125 µmol/L	(3 - 17)
ALP	490 u/L	(30 - 100)
ALT	384 u/L	(3 - 40)
γGT	102 u/L	(8 -60)
Albumin	41 g/L	(35-50)
INR	1.8	
ASMA	positive	
AMA	negative	
ANA	positive	
HBsAg	negative	
HCB Ab	negative	
Creatinine	84 µmol/L	(55-120)

A liver biopsy has been scheduled.

What is the most appropriate initial treatment for this patient?

A. Liver transplantation
B. Mycophenolate mofetil (MMF)
C. Prednisolone
D. Prednisolone and azathioprine
E. Ursodeoxycholic acid

C. Prednisolone

Steroids are part of the first-line recommended treatment for severe autoimmune hepatitis

Prednisolone is the correct answer. The clinical presentation, alongside abnormal liver biochemistry and positive antinuclear antibody (ANA) and anti-smooth muscle antibody (ASMA), strongly suggests autoimmune hepatitis as the most likely diagnosis. This patient presents with severe acute autoimmune hepatitis, characterised by jaundice, an elevated international normalised ratio (INR), and no hepatic encephalopathy. Prednisolone is the cornerstone of treatment in such scenarios. Following initial management with steroids, maintenance therapy typically includes azathioprine, which is commenced once cholestasis has resolved.

Liver transplantation is not the correct choice. While liver transplantation must be considered for any individual with severe acute autoimmune hepatitis, current indications include acute liver failure, decompensated cirrhosis or a lack of response to conventional medical treatment. As this case involves a recent diagnosis without prior therapeutic intervention and lacks evidence of chronic liver disease or malignancy, this option is inappropriate.

Mycophenolate mofetil (MMF) acts by inhibiting inosine monophosphate dehydrogenase (IMPDH), crucial for T and B lymphocyte proliferation, making it a second-line agent when azathioprine cannot be used due to contraindications or intolerance. At this stage, however, induction therapy with steroids is required; therefore, MMF is not an appropriate choice.

The combination of **Prednisolone and azathioprine** may be employed for maintenance therapy in certain cases. Nevertheless, initiation of azathioprine should only occur after cholestasis resolution indicated by higher alkaline phosphatase (ALP) relative to alanine transaminase (ALT) and increased gamma-glutamyl transferase (GGT). The rationale for this approach is that azathioprine can cause idiosyncratic liver injury and potentially worsen existing cholestasis.

Ursodeoxycholic acid does not represent the correct answer in this context. It serves as the firstline treatment for primary biliary cholangitis; however, while overlap syndromes between autoimmune hepatitis and primary biliary cholangitis occur, negative antimitochondrial antibodies (AMA) rule out primary biliary cholangitis in this patient's case. Therefore, ursodeoxycholic acid would not be a suitable therapeutic option here.

Question 20 of 235

A 34-year-old patient presents to the endocrinology clinic. He has had difficulties with erections and sexual desire, as well as low energy. He was referred by the GP after a morning serum testosterone was low. His problems started six months ago, following increasing symptoms of acid reflux. He also has T1DM. His current medications include Levemir, Novorapid, omeprazole and metoclopramide. Capillary monitoring of sugar levels has been within normal range, but prolactin has found to be elevated.

What is the most appropriate action?

A. Start testosterone replacement
B. Stop metoclopramide
C. Prescribe testosterone replacement
D. MRI of pituitary
E. Stop omeprazole

B. Stop metoclopramide

Metoclopramide can cause drug-induced hyperprolactinaemia presenting as hypogonadism in men

This patient has developed symptoms of hypogonadism after being diagnosed with acid reflux. The connecting element is metoclopramide; it is a commonly used anti-emetic, especially in patients with type 1 diabetes, due to its pro-kinetic effects. However, it can also cause elevated prolactin which has a negative feedback effect on the hypothalamus-pituitary-gonadal axis, causing low testosterone. Stopping it may relieve the effects. If this did not work, then replacing the testosterone would be appropriate.

Question 21 of 235

A 45-year-old woman is referred to the endocrinology clinic. She has been experiencing episodes of feeling faint, light-headed and nauseous approximately two hours after eating meals. She has measured her blood glucose at these points and found herself to have capillary blood glucose around 2mmol/l. She has found that having a sugar containing food or beverage helps at this point. She has a history of morbid obesity for which she underwent a Roux en Y reconstruction. Since then she has gone from a BMI of 45 to 29 over one year. She has also a diagnosis of type 2 diabetes mellitus which was treated with metformin and gliclazide, but has now manage to come off all medication.

What is the most likely diagnosis?

A. Insulinoma
B. Early dumping syndrome
C. Late dumping syndrome
D. Gliclazide abuse
E. Liver failure

C. Late dumping syndrome

Dumping syndrome can cause post-prandial hypoglycaemia following gastric bypass or bariatric surgery

This patient has late dumping syndrome, also known as postprandial hyperinsulinemic hypoglycaemia. This occurs in approximately 0.2% of patients following bariatric surgery, in particular Roux en Y reconstruction. Hypoglycaemia occurs one to three hours following ingestion of carbohydrate heavy meals, and presentation is months to years after surgery. This would be managed generally with diet changes.

Early dumping syndrome is rapid emptying of food into the small bowel causing colicky abdominal pain, diarrhoea and nausea, and is far more common complication of bariatric surgery. Gliclazide abuse is very unlikely in the absence of any psychiatric elements of her history. Insulinoma can present with hypoglycaemia, but this usually occurs in the mornings, and is associated with weight gain.

Question 22 of 235

A 55-year-old woman is seen in the gastroenterology clinic with a history of itching and malaise, having been found to have a persistently elevated alkaline phosphatase for the last 12 months by her GP. She has a background in rheumatoid arthritis. Anti-mitochondrial antibodies and smooth muscle antibodies results are as below.

Anti-mitochondrial antibodies	positive
Smooth muscle antibodies	positive

What additional test is required before the diagnosis can be confirmed?

A. Faecal elastase

B. Gamma glutamyl-transferase
C. Liver biopsy
D. Rheumatoid factor
E. Ultrasound of abdomen

E. Ultrasound of abdomen

Imaging is required before making a diagnosis of PBC to exclude extrahepatic biliary obstruction

The diagnosis in this patient is primary biliary cholangitis (PBC) - as evidenced by her history of autoimmune conditions, her isolated elevated alkaline phosphatase, and her positive antimitochondrial antibodies. All patients who are suspected to have PBC should have imaging (either ultrasound or magnetic resonance cholangiopancreatography (MRCP) to exclude extra-hepatic biliary obstruction before making the diagnosis.

Faecal elastase is a test for pancreatic insufficiency and is not indicated here.

Gamma glutamyl-transferase is likely to be raised in this scenario but it would not add any diagnostic value to the investigations.

Liver biopsy is not required as the diagnosis can be made based on positive anti-mitochondrial antibodies combined with the cholestatic blood picture. A biopsy may occasionally be considered if there is uncertainty between the diagnoses of PBC and autoimmune hepatitis.

Rheumatoid factor testing would not be needed as the patient is already known to have established rheumatoid arthritis.

Question 23 of 235

A 53-year-old man presents to the emergency department with abdominal pain. He has not past medical history and does not take any regular medications. He works as a sheep farmer in rural Wales. He drinks 1-2 bottles of whiskey a week.

His observations are as follows:

Heart rate 88 beats per minute Blood pressure 120/65 mmHg Respiratory rate 18/minute Oxygen saturations 96% on room air Temperature 37.4C

On examination, he is tender in the right upper quadrant and there is evidence of hepatomegaly. Blood tests:

Hb	136 g/L	Male: (135-180)Female: (115 - 160)
Platelets	189 * 10 /L 9	(150 - 400)
WBC	12.2 * 10 /L 9	(4.0 - 11.0)
Na +	138 mmol/L	(135 - 145)
K +	4.2 mmol/L	(3.5 - 5.0)
Urea	5.2 mmol/L	(2.0 - 7.0)
Creatinine	89 µmol/L	(55 - 120)
Bilirubin	12 µmol/L	(3 - 17)
ALP	89 u/L	(30 - 100)
ALT	45 u/L	(3 - 40)
Î³GT	66 u/L	(8 - 60)
Albumin	36 g/L	(35 - 50)
CRP	22 mg/L	(<4)

CT imaging of the abdomen is arranged, which demonstrates a large cystic lesion with a calcified laminar wall along with floating inclusions and three peripheral daughter cysts.

Which investigation is most likely to confirm the diagnosis?

A. Genetic testing
B. Serological testing for Echinococcus granulosus
C. Serological testing for Entamoeba histolytica
D. Serum Alpha foetal protein (AFP)
E. Stool antigen testing for Entamoeba histolytica
F. Serological testing for Echinococcus granulosus

B. Serological testing for Echinococcus granulosus

Serology for *Echinococcus granulosus*can be useful in the investigation of suspected hydatid cysts

Serological testing for *Echinococcus granulosus* is the correct answer. Hydatid disease caused by *Echinococcus granulosus* has been noted to occur with increased frequency in rural Wales due to its association with sheep farming. If the cysts grow large enough, they can be associated with symptoms such as abdominal pain and signs such as hepatomegaly. The CT imaging findings are characteristic of this condition, with a large cyst characterised by a thick, calcified laminar wall, floating inclusions and smaller cysts near the main lesion called 'daughter cysts'.

Genetic testing is incorrect. If the diagnosis were polycystic liver disease, we would expect to see numerous liver cysts, rather than one large cyst surrounded by few peripheral daughter cysts. We may also expect to see kidney lesions.

Serological testing for *Entamoeba histolytica* is incorrect. The imaging findings suggestive of an amoebic liver abscess may demonstrate a 'double-target' sign, where there is central low attenuation fluid surrounded by an outer ring with high attenuation on the inside of the ring and lower attenuation on the outside. Additionally, amoebic liver disease is more common in tropical and sub-tropical areas, rather than the Welsh countryside.

Serum Alpha foetal protein (AFP) is incorrect. While alcohol is a risk factor for the development of liver cancer, the imaging findings, in this case, suggest a liver abscess rather than a malignancy.

Stool antigen testing for *Entamoeba histolytica* is incorrect. This is another means of diagnosing *Entamoeba histolytica* infection. However, the imaging and epidemiological history favour hydatid disease, as described above.

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A 35-year-old woman is referred to hepatology with deranged liver function tests and jaundice. She has no past medical history and is not on any regular medications. She does not smoke or drink alcohol.

On examination, there is scleral icterus and mild ascites. The BMI is 24 kg/m². Blood tests:

Hb	137 g/L	Male: (135-180)Female: (115 - 160)
Platelets	189 * 10 /L 9	(150 - 400)
WBC	8.2 * 10 /L 9	(4.0 - 11.0)

Na +	137 mmol/L	(135 - 145)
K +	4.2 mmol/L	(3.5 - 5.0)
Urea	5.2 mmol/L	(2.0 - 7.0)
Creatinine	77 µmol/L	(55 - 120)
CRP	4 mg/L	(< 5)
Bilirubin	62 µmol/L	(3 - 17)
ALP	248 u/L	(30 - 100)
ALT	320 u/L	(3 - 40)
Î³GT	88 u/L	(8 - 60)
Albumin	36 g/L	(35 - 50)

Further blood tests:

Alpha 1 anti-trypsin	negative	(negative)
Ferritin	420 ng/ml	(10-120)
Antinuclear antibody	positive	(negative)
Anti smooth muscle antibody	positive	(negative)
Caeruloplasmin	1.1 µmol/L	(0.93 to 2.65)
Anti mitochondrial antibody	negative	(negative)
Anti LKM1 antibody	negative	(negative)
Soluble liver-kidney antigen	negative	(negative)
Hepatitis B surface antigen	negative	(negative)
Hepatitis C antibody	negative	(negative)
HIV	negative	(negative)

What is the most likely diagnosis?

A. Haemochromatosis
B. Primary biliary cirrhosis
C. Type 1 autoimmune hepatitis
D. Type 2 autoimmune hepatitis
E. Type 3 autoimmune hepatitis

C. Type 1 autoimmune hepatitis

Deranged LFTs + anti-smooth muscle antibodies (SMA) → ?type 1 autoimmune hepatitis

Type 1 autoimmune hepatitis is the correct answer. This patient presents with signs of chronic liver disease (jaundice and ascites) and deranged liver function tests. The positive antinuclear antibody and anti-smooth muscle antibody suggests type 1 autoimmune hepatitis as the diagnosis.

Haemochromatosis is incorrect. A mildly raised ferritin is common in many forms of liver inflammation including autoimmune hepatitis. The positive anti-smooth muscle antibody is more suggestive of type 1 AIH.

Primary biliary cirrhosis is incorrect. This typically occurs in middle-aged females. The liver function test pattern tends to be cholestatic and anti-mitochondrial antibodies are usually positive.

Type 2 autoimmune hepatitis is incorrect. This disorder only occurs in children and is associated with Anti LKM1 antibody.

Type 3 autoimmune hepatitis is incorrect. This is associated with soluble liver-kidney antigen positivity and typically occurs in older patients.

Question 25 of 235

A 45-year-old man is referred to the gastroenterology outpatient department with worsening dyspepsia. He has a past medical history of gastroesophageal reflux disease. He takes low-dose omeprazole daily. He does not smoke or drink alcohol.

An endoscopy is arranged. A biopsy demonstrates low-grade dysplasia.

What is the optimum management of this patient?

A. High dose proton-pump inhibitor
B. Ivor Lewis oesophagectomy
C. Radiofrequency ablation
D. Submucosal resection
E. Surveillance

C. Radiofrequency ablation

Barrett's oesophagus: radiofrequency ablation is the preferred treatment for low-grade dysplasia

Radiofrequency ablation is correct. As this patient has a history of GORD, the most likely cause of the low-grade dysplasia on endoscopy is the progression of metaplasia from Barrett's oesophagus. She has no significant risk factors for squamous cell carcinoma e.g. smoking/alcohol. A high dose of PPI may help her symptoms but has not been proven to induce regression of dysplasia. The optimum treatment in this setting is radiofrequency ablation.

High dose proton-pump inhibitor is incorrect. This may help her symptoms of dyspepsia and reduce reflux, but it has not been shown to be effective in preventing the progression or inducing regression of dysplasia.

Ivor Lewis oesophagectomy is incorrect. This is a surgical procedure to treat oesophageal cancer. It is not necessary if merely low-grade dysplasia (rather than invasive cancer) is identified. As there is no invasive cancer in this case, a surgical procedure is not warranted.

Submucosal resection is incorrect. This is another option for treating dysplasia of any type identified. However, typically radiofrequency ablation is preferred for low-grade dysplasia rather than moderate-high grade.

Surveillance is incorrect. For patients with metaplasia (but not dysplasia) endoscopy is recommended every 3-5 years. This patient requires intervention at this point due to the existence of dysplasia.

Question 26 of 235

A 65-year-old man with his first exacerbation of COPD is admitted to the respiratory ward. He is started on doxycycline and prednisolone and starts to improve well. On day 3, he complains of feeling nauseous and begins to vomit. He develops central abdominal pain that is constant. He has two episodes of watery diarrhoea

On examination, his blood pressure is 90/65 mmHg, heart rate is 60 beats per minute, respiratory rate is 22/min and saturations are 92% maintained on 28% oxygen. There is guarding across the abdomen which is most pronounced over the umbilical area.

Hb	130 g/l
Platelets	500* 10 /l9
WBC	14* 10 /l9

Na +	135 mmol/l
K +	4.2 mmol/l
Urea	6.6 mmol/l
Creatinine	100 µmol/l
Bilirubin	22 µmol/l
ALP	120 u/l
ALT	35 u/l
Amylase	500 U/dl (normal range 28-100)
Albumin	33 g/l
Chest x-ray - small right sided effusion, bilateral perihilar infiltrates	
Abdominal x-ray - short segment of distended small bowel	
Ultrasound abdomen - two solitary stones seen in gallbladder, no wall thickening and a peripancreatic collection of fluid seen	

What is the underlying cause of his deterioration?

A. Prednisolone
B. Amoxicillin
C. Gallstones
D. Sepsis
E. Clostridium difficile Next question

A. Prednisolone

Steroids are a cause of acute pancreatitis

This is an important case at recognising the different causes of abdominal pain. The clearest features are the raised amylase and central abdominal pain which points towards pancreatitis. Gallstones are the main cause but this gentleman no obstructing stones and it is a common physiological finding to find stones in the bladder. The timing of the pancreatitis coincides with the onset of steroid use. Sepsis is unlikely since this gentleman was clinically improving but it can be a cause of acalculous cholecystitis.

Question 27 of 235

A 50-year-old woman attends her GP with general tiredness and aches throughout the body that are worst in the pelvic girdle. She has mild shoulder girdle pain but no visual change or jaw pain. There is no change to her bowels and her stools often are loose but brown in colour. She has not noticed any itching or jaundice. She reports difficulty sleeping at night following recent bereavement of her sister.

She has primary biliary cirrhosis and is managed on ursodeoxycholic acid. On examination, she is not clinically icteric and has red conjunctiva. She is alert with a soft abdomen and palpable liver 1cm below the costal margin. She has no asterixis or spider naevi. Her ankles are not swollen. She is has a full range of motion and no swelling in all joints but is tender over the gluteal and quadriceps muscles in the legs.

Hb	140 g/l	Na +	137 mmol/l
Platelets	295 * 10 /l 9	K +	4.3 mmol/l
WBC	10 * 10 /l 9	Urea	5.2 mmol/l
Neuts	9.4 * 10 /l 9	Creatinine	67 µmol/l
Calcium (adjusted)	2.1 mmol/l (range 2.2-2.6)	Phosphate	0.5 mmol/L (range 0.7-1.0)
Bilirubin	25 µmol/l	ALT	40 u/l
ALP	110 u/l	γGT	65 u/l
Albumin	32 g/l	Ferritin	15ng/ml (range 10-300)
B12	200 pg/ml (range 180-2000)	Folate	3.4 ng/ml (range >4.0)
ESR	17 (range 5-15)	Hip X-ray	ordered

What is the likely diagnosis?

A. Progression of primary biliary cirrhosis
B. Depression
C. Osteomalacia
D. Iron deficiency
E. Polymyalgia rheumatica

C. Osteomalacia

PBC -> high risk for osteomalacia Next question

This lady has fatigue and limb pains. She has possible stressors for depression but this should be a diagnosis of exclusion. She is iron deficient but not anaemic. Her LFTs are not deranged and she is clinically not in liver failure so her PBC does not seem to have progressed.

The limb girdle pain should prompt thoughts of polymyalgia but her ESR is only mildly raised. She has low phosphate and calcium which is seen in malabsorption and can occur following vitamin D deficiency. This can lead to bone resorption and osteomalacia with general fatigue and bony pains which is the likeliest answer. X-rays may show Looser's zones and confirm the diagnosis. She should have high dose vitamin D replacement.

Question 28 of 235

A 35-year-old male patient was admitted with acute severe ulcerative colitis. He had been having frequent episodes of bloody diarrhoea with abdominal pains. He had been on a reducing dose of steroids while at home, but failed to respond. After 5 days of treatment with intravenous hydrocortisone, he became tachycardic, hypotensive and worsening of his abdominal pain.

Investigations

Hb	136 g/l
WBC	10 * 10 /l9
Platelets	250 * 10 /l9
Serum albumin	31 g/l
Serum CRP	68mg/l

What is the next most appropriate investigation?

A. CMV serum PCR
B. Faecal calprotectin
C. CT abdomen
D. Stool for Clostridium difficile
E. Unprepared flexible sigmoidoscopy and biopsy

C. CT abdomen

CT abdomen is an important first line investigation to rule out complications in a patient with ulcerative colitis presenting with severe systemic symptoms

Acute tachycardia with or without abdominal pain (often masked by steroids) is a worrying feature in acute severe ulcerative colitis. It warrants urgent assessment and investigation for complications that may require urgent surgery. CT scanning has become the gold standard for identifying perforation, and where available is preferable to a plain erect chest X-ray and abdominal film. *Clostridium difficile* is an important consideration, but should already have been tested for by day 5 of the presentation.

Indications for surgery in the acute setting include the following:

- Failure of medical management
- Perforation
- Toxic megacolon
- Severe haemorrhage
- Malignancy

The other investigations CMV serum PCR, faecal calprotectin, stool for *Clostridium difficile* will be useful in subsequent evaluation of the patient but ruling out a surgical emergency which is likely in this scenario with a CT abdomen is most appropriate initial investigation for prompt evaluation and management.

Question 29 of 235

A 49 year-old woman is referred by her GP with a worsening 3-week history of chest pain and burning behind the sternum after eating. Other symptoms include tiredness and the patient has noticed that over the past four months her clothes have started to become much looser on her, although she has put this down to stress at work and skipping meals. She denies night sweats and her only past medical history is mild hypertension for which she takes ramipril. She is on no other regular medication and her father died of lung cancer at the age of 67. Her mother is still alive and well. She drinks 10-15 units per week and has a 25 pack year history.

On examination, heart sounds 1 and 2 are present with no added sounds, her pulse is regular and the character is good and she has clear lung fields. Abdominal exam reveals a soft, non-tender abdomen and bowel sounds are present.

She is referred to gastroenterology where an endoscopy reveals a 2cm diameter area that looks roughened and suspicious. Biopsy of this area is reported as having large number of lymphocytes that have irregular nuclear contours with abundant cytoplasm.

Which of the following is the most appropriate first-line treatment for this condition.

A. Endoscopic surgical excision
B. Chemotherapy
C. Open surgery
D. Amoxicillin, clarithromycin and omeprazole treatment
E. Radiotherapy

D. Amoxicillin, clarithromycin and omeprazole treatment

Gastric MALT lymphoma - eradicate *H. pylori*

The condition described here is a MALT lymphoma (or MALToma) which is a form of lymphoma invading the mucosa-associated lymphoid tissue. Because there is a causal relationship between Helicobacter Pylori infection and MALT lymphoma, it is vital to identify the presence of H. pylori and treat it. 50-90% of cases achieve complete response with H. pylori treatment and eradication.

While radiotherapy may be a valid option for MALT lymphoma, the irradiation fields can potentially be very large, as the whole stomach must be included. Chemotherapy is reserved for those patients with disseminated disease or a lack of response to local treatment.

Question 30 of 235

A 26-year-old woman presents to the emergency department with a three-day history of yellowing of the skin and feeling unwell. She is one-month postpartum and reports that she was diagnosed with pregnancy-induced hypertension in her third trimester for which she was prescribed labetalol. She reports no other medical history of note and takes no other regular medications. Her first pregnancy was uncomplicated and both her children are well. She reports no use of over the counter or herbal remedies and is not aware of any recent courses of antibiotics. She grew up in Pakistan and last visited her family in Lahore 6 months ago. She has no history of previous blood product transfusions or of any recreational drug use.

On examination she is haemodynamically stable, she is alert and orientated with no asterixis. An examination of the cardiovascular and respiratory systems and abdomen are unremarkable aside from jaundice and signs of excoriation. Her blood pressure in the department is 110/60mmHg. The decision is made to admit the patient for further testing.

Blood tests are performed and results are as follows:

Bilirubin	100µmol/L	(3 - 17)
ALP	346u/L	(30 - 100)
ALT	1051u/L	(3 - 40)
IgG	18.0 g/L	(6.0 - 16.0)

Hepatitis A IgM antibody	Negative
Hepatitis A IgG antibody	Positive
Hepatitis B surface antigen	Negative
Hepatitis B core IgM antibody	Negative
Hepatitis C antibody	Negative
HIV 1&2 antibodies	Negative
Antinuclear antibody	Negative
Anti-smooth muscle antibody	Negative

An abdominal ultrasound is unremarkable and hepatic dopplers show normal flow through the hepatic vessels.

What is the most likely explanation for this patient's acute hepatitis?

A. Autoimmune hepatitis
B. Hepatitis A acute infection
C. Hepatitis A chronic infection
D. Ischaemic hepatitis
E. Labetalol induced hepatitis

E. Labetalol induced hepatitis

Labetolol can cause acute hepatitis

Labetalol can cause acute hepatitis and is often used in the control of pregnancy induced hypertension.

Our differentials, in this case, would include use of other drugs (antibiotics are a common culprit), ischaemia, viral and autoimmune hepatitis.

Ischaemia is unlikely to have caused this patient's hepatitis as there is no clear history of acute hypoperfusion (we would expect patients to usually have an identifiable incident of symptomatic hypotension).

This patient has evidence of past infection with hepatitis A, but no evidence to suggest acute infection and hepatitis A does not cause chronic infection.

Pregnancy can unmask underlying autoimmune conditions such as autoimmune hepatitis, however, we would expect a much higher IgG level in patients with autoimmune hepatitis along with a positive

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A 48-year-old man has attended the gastroenterology clinic for the results of recent investigations. He was initially referred by his GP due to unexplained weight loss, dysphagia, and the regurgitation of food. His only regular medication includes omeprazole 40mg twice daily.

Blood results were unremarkable.

Oesophagoduodenoscopy (OGD) was performed and biopsies were sent for histology.

OGD	Endoscope passed successfully to D3 with retroflection in the stomach. The distal third of the oesophagus displayed 2x circumferential rings with evidence of furrowing. Small stricture present but no evidence of malignancy. 4x biopsies taken. The stomach and duodenum appeared normal.
Histopathology	Four specimens were examined. Microscopically, all specimens display increased eosinophil activity.

Given the likely diagnosis, what is the most appropriate initial management?

A. Dietary modification
B. Electrocautery via endoscopy
C. Oesophageal dilatation
D. Oesophageal stent
E. Systemic corticosteroids

A. Dietary modification

Dietary modification and topical steroids are used in the management of eosinophilic oesophagitis

The above symptoms of weight loss, dysphagia and food regurgitation are non-specific and are present in many oesophageal disorders. OGD findings, however, show circumferential oesophageal rings with furrowing. These findings in combination with increased eosinophil activity on histology should point towards the diagnosis of eosinophilic oesophagitis.

Eosinophilic oesophagitis is a diagnosis made through good symptom history and the ruling out of other differential diagnoses such as reflux. Macroscopically, the oesophagus may appear normal, however, OGD may show reduced vasculature, thick mucosa, mucosal furrows, strictures and laryngeal oedema. Diagnosis is confirmed through histology showing increased eosinophil activity and deposition.

Eosinophilic oesophagitis is treated with **dietary modifications**, in an attempt to eliminate the causative allergen, and topical corticosteroids, such as budesonide.

Electrocautery via endoscopy is an older endoscopic practice that was used for minor endoscopic surgeries, such as polypectomies. It is not used in the management of eosinophilic oesophagitis.

Oesophageal dilatation is a treatment used in patients with eosinophilic oesophagitis when there are symptoms of dysphagia and endoscopy shows oesophageal strictures. In the above patient, endoscopy showed circumferential rings but no evidence of stricturing and so oesophageal dilatation is not currently required.

Oesophageal stenting is not generally used in the management of eosinophilic oesophagitis as strictures can form in other areas of the oesophagus. Oesophageal stenting is more commonly used in oesophageal malignancy to aid in the symptoms of dysphagia.

The use of **systemic corticosteroids**, such as prednisolone, has been shown to be as effective in the management of eosinophilic oesophagitis when compared to topical steroids. However, systemic corticosteroids carry an increased side effect profile related to the systemic effect of the steroids and therefore are not often used. Topical steroids have a local effect in the oesophagus with only minor systemic effects.

Question 32 of 235

A 62 year old man presents to the medical assessment unit with intense pruritus and jaundice. He has recently been prescribed a new medication from his GP, but cannot remember the name.

His blood tests reveal the following:

Bilirubin	78 µmol/l
ALP	300 u/l
ALT	50 u/l
γGT	45 u/l

Which of the following medications would most likely be responsible?

A. Metformin
B. Flucloxacillin
C. Ibuprofen
D. Sodium valproate
E. Doxycycline

B. Flucloxacillin

Flucloxacillin is a well recognised cause of cholestasis

These liver function tests, are example of cholestatitic jaundice. The alkaline phosphatase and the bilirubin are raised out of proportion to the liver transaminases.

Common drugs causing cholestasis:

- Flucloxacillin
- Erythromycin
- Chlorpromazine
- Oral contraceptives
- Anabolic steroids

Question 33 of 235

The Medical Emergency Team was called to the interventional radiology department to review a 48 year-old farmer who was attending for liver biopsy. The procedure itself was uneventful but a few minutes afterwards he became acutely unwell and short of breath. His past medical history was unremarkable apart from on-going investigation for deranged liver function tests. He was on no medication and had no known allergies. He had no travel history of note, was a non-smoker and drank 14 units of alcohol per week.

On examination, he was laid flat on the trolley and appeared flushed. His temperature was 36.7ºC, heart rate was 110 beats per minute, blood pressure 82/40 mmHg, respiratory rate 28 breaths per minute and oxygen saturations 100% on non-rebreather mask. The chest was clear on auscultation and heart sounds were normal. His JVP was not visible. There was some mild right upper quadrant tenderness on palpation of the abdomen, but no guarding or rebound tenderness.

The investigations prior to the biopsy were as follows:

Haemoglobin	160 g/L (130-180)
White cell count	8.0 * 10 /l9
Neutrophil count	4.0 * 10 /l9
Lymphocyte count	3.0 * 10 /l9
Eosinophil count	0.8 * 10 /l9
Platelets	350 * 10 /l9
Sodium	140 mmol/L
Potassium	4.1 mmol/L
Urea	6.5 mmol/L
Creatinine	75 mol/L
Alkaline phosphatase	101 IU/L
Alanine aminotransferase	82 IU/L
Gamma-glutyl transferase	59 IU/L
Bilirubin	18 mol/L
Albumin	36 g/L
Prothrombin time	12 s (10-14)
Activated partial thromboplastin time	38 s (30-45)

Ultrasound abdomen: Hypoechogenic lesion in the right lobe of the liver

What is the most appropriate initial treatment?

A. Intravenous dobutamine
B. Intramuscular adrenaline
C. Intravenous antibiotics
D. O-negative blood transfusion
E. Emergency laparotomy

B. Intramuscular adrenaline

This patient is likely to have a hydatid cyst, which has ruptured during liver biopsy and caused anaphylactic shock. The disease is caused by larval encystment of the tapeworm *Echinococcus granulosus*, which can occur in the UK with dogs acting as reservoir host when they consume offel. Farmers therefore have an elevated risk of ingesting the eggs of the parasite through canine faecal contamination of food. The eggs then hatch into larvae and encyst in tissues such as the liver. Haemorrhage and sepsis are less likely in this case given the flushed appearance of the patient and lack of pyrexia respectively.

Question 34 of 235

A 72-year-old male under palliative care for metastatic antral gastric carcinoma presented to the Emergency Department on a Friday night. He was worried as the gastrostomy tube withdrew accidentally while he was taking a shower. This tube was used as a feeding route. On examination the patient was fully conscious, blood pressure 123/75 mmHg and pulse rate 94/min. His abdomen was soft and non-tender. Blood investigations showed:

Hb	114 g/l
Platelets	220 * 10 /l9
WBC	7.7 * 10 /l9

You are the core trainee doctor responsible for the patient care. What is the most appropriate action you should take?

A. Discharge with advice to come on Monday for senior review
B. Insertion of nasogastric tube as an alternative feeding pathway and discharge
C. Insertion of Foley's catheter in the previous gastrostomy opening
D. Admission and monitor
E. Trial to re insert the gastrostomy tube

C. Insertion of Foley's catheter in the previous gastrostomy opening

A gastrostomy tube is mainly used in cases of proximal gastrointestinal tract obstruction to facilitate feeding. If it withdrew accidentally, reinsertion of the tube as soon as possible would be the preferred action. However, it needs a good level of expertise to do this. Therefore, in this case, insertion of a Foley's catheter is the best practice as it is easy to do and this should preserve the opening of the skin and anterior abdominal wall muscles until a someone experience enough is available to re-insert the gastrostomy tube. Waiting until Monday would result in a higher chance of spontaneous closure of the opening.

A nasogastric tube is not the correct option because it would not bypass the antral tumour.

Question 35 of 235

A 72-year-old man attends gastroenterology clinic for follow-up after a recent upper gastrointestinal endoscopy. The patient had been referred by his GP for Barrett's oesophagus screening due to long-standing and severe gastro-oesophageal reflux symptoms, associated with obesity and current smoker status.The patient reported suffering from severe heartburn after eating over the previous 10 years. He had been a regular user of over the counter anti-acid preparations, but had only sought medical advice in recent weeks after a close friend was diagnosed with gastric cancer.

The patient's past medical history included obesity, hypertension, type 2 diabetes mellitus and gout. His regular medications were ramipril, allopurinol, metformin and gliclazide. The patient denied any drug allergies or intolerances. His father had undergone a partial gastrectomy for peptic ulcer disease, but there was no family history of Barrett's oesophagus or oesophageal adenocarcinoma.

The patient was a retired school teacher who lived with his wife. He had smoked 10 cigarettes per day throughout his adult life and was abstinent from alcohol.

Following initial clinic assessment, an upper gastro-intestinal endoscopy was arranged (results given below). The patient had also been initiated on high-dose proton-pump inhibitor therapy, which had been associated with significant improvement in reflux symptoms.

Report from upper gastro-intestinal endoscopy: no evidence of hiatus hernia; 8 cm segment of circumferential salmon-coloured epithelium extending above gastro-oesophageal junction (quadrant biopsies taken as per protocol); no mass lesion or ulceration throughout oesophagus, stomach or first-part duodenum; mild-moderate oesophagitis and mild gastritis.

Histology from endoscopic biopsies: flat intestinal metaplasia present in 15 of 16 samples; no evidence of dysplasia or carcinoma.

In addition to continued proton-pump inhibitor treatment, what is appropriate management of this patient's Barrett's oesophagus?

A. Endoscopic mucosal resection
B. Surveillance endoscopy every 3 years
C. Radiofrequency ablation
D. Surveillance endoscopy every 1 year
E. Risk factor modification advice only

B. Surveillance endoscopy every 3 years

Screening for Barrett's oesophagus is recommended in individuals with chronic or severe gastrooesophageal reflux disease symptoms, who also have three additional risk factors (out of: age > 50 years, male sex, white race, obesity, smoking), or who have a family history of Barrett's oesophagus or oesophageal carcinoma.

The patient in this question has Barrett's oesophagus with intestinal metaplasia but without dysplasia or intra-mucosal carcinoma. The risk of progression of non-dysplastic Barrett's oesophagus to oesophageal adenocarcinoma is 0.33 % annually, meaning that endoscopic surveillance is recommended over therapy. Recommended frequency for endoscopic surveillance is every 3-5 years.

In the presence of dysplasia, endoscopic therapy is required due to the much higher risk of progression to adenocarcinoma (10 % per annually in high-grade dysplasia). A tissue acquisitive technique such as endoscopic mucosal resection is required for nodular lesions, with ablative therapy such as radiofrequency ablation reserved for flat regions of dysplasia or intra-mucosal carcinoma.

Proton-pump inhibitor therapy is recommended in patients with Barrett's oesophagus due to several cohort studies suggesting that it reduces the risk of progression to adenocarcinoma.

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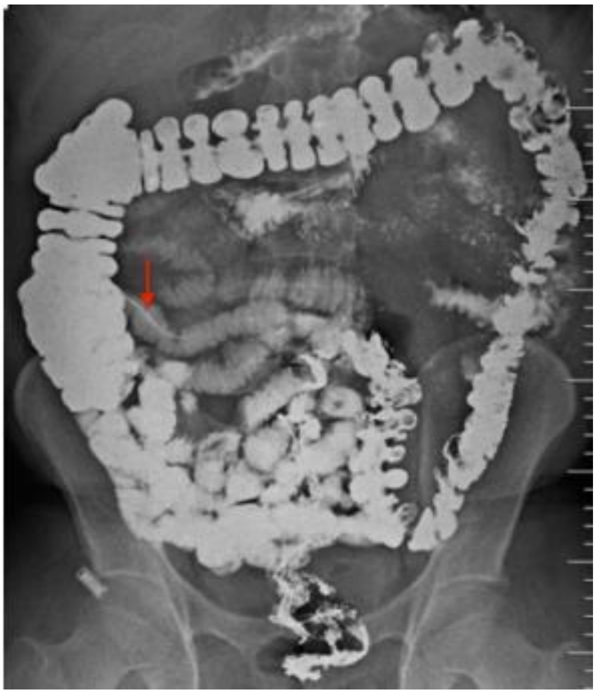
A 35-year-old female who has recently emigrated from Pakistan presents is investigated for abdominal pain and diarrhoea.

Around 4 months ago she was investigated in Pakistan for an episode of dyspnoea, wheezing, fever and malaise. No cause was found at the time and she improved after 3 weeks. A barium enema is performed:



What is the most likely diagnosis?

A. Ascariasis
B. Enterobiasis
C. Giardiasis
D. Carcinoid syndrome
E. Ulcerative colitis

A. Ascariasis

This patient has Ascariasis. The episode in Pakistan is likely to be Loffler's syndrome whilst the parasite was migrating through lungs.

Question 37 of 235

A 57-year-old gentleman with presents to gastroenterology clinic for review. He has been investigated for haemochromatosis and is being told that this diagnosis is confirmed. On referral, he had a past medical history of previously unexplained cirrhosis with transaminitis, arthritis causing arthralgia and hypogonadism. He is due to be started on regular venesection.

Which of his problems is most likely to improve with treatment?

A. Cirrhosis
B. Transaminitis
C. Arthritis
D. Arthralgia
E. Hypogonadism

B. Transaminitis

The correct answer is transaminitis. Venesection aims to rapidlly remove the excess iron within the body but does not generally reverse established damage. Removal of one unit per week or two is normally effective to deplete of iron, and then the frequency can be reduced. Chronic damage to liver, joints and heart is normally irreversible, but acute injuries can respond well.

Usually irreversible	Improvement in some patients	Usually reversible
Cirrhosis	Diabetes mellitus	Fatigue
Arthritis	Hypogonadism	Transaminase elevation
Arthralgia		

Question 38 of 235

A 55-year-old man attends outpatient clinic for review. He was diagnosed with ulcerative pancolitis 13 years ago, and is currently treated with Asacol (mesalazine). He had a colonoscopy last month that showed mild active inflammation throughout the colon, and no polyps. He has no relevant family history.

What should be the time interval until his next surveillance colonoscopy?

A. 2 years
B. 3 years
C. 5 years
D. 7 years
E. 1 year Next question

B. 3 years

Patients with inflammatory bowel disease are 6 times more likely to develop colorectal cancer compared to the general population. Because of this, it is recommended that regular colonoscopies are performed for surveillance. These are only initiated 10 years after diagnosis, and their frequency depends on previous endoscopic findings, distribution of inflammation, family history of colorectal cancer and other factors in the patient's past medical history (e.g. PSC).

This patient falls into the intermediate risk category as he has a pancolitis (inflammation throughout the colon), with mild inflammation on previous endoscopy. This means the time interval is 3 years between surveillance investigations.

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A 78-year-old woman is admitted to the acute medical unit due to shortness of breath. She had been living alone at home for the last 3 years since her husband died. In the last 6 months, she was struggling to manage due to fatigue, nausea and progressive breathlessness. She had noticed that her clothing was feeling tighter but could not understand why as she had reduced appetite and was eating less than normal. She is a non-smoker and drank 1-2 bottles of wine per week since the death of her husband.

She has a history of ischaemic heart disease following an acute myocardial infarction two years previously. An echocardiogram 6 months after the acute event showed a mildly dilated left ventricle with good overall function (ejection fraction 45-55%) and mild aortic stenosis. Past medical history also includes a left mastectomy 17 years ago for a small breast carcinoma. Lymph node sampling at the time showed clear nodes so axillary clearance was not carried out. She has been discharged from follow up at the breast clinic.

On examination, she is apyrexial with no evidence of clubbing or splinter haemorrhages nor any spider naevi. Her chest was clear to auscultation with dullness to percussion at both bases. The cardiac apex was non-displaced and heart sounds were normal with an additional ejection systolic murmur. Her abdomen appeared distended with large volume ascites but was soft and nontender. Bowel sounds were normal.

Bloods showed the following:

Haemoglobin	101 g/L	Sodium	134 mmol/L
Platelets	159 x10^9/L	Potassium	3.8 mmol/L
White cell count	9.0 x10^9/L	Urea	3.5 mmol/L
Neutrophils	3.5 x10^9/L	Creatinine	67 micromol/L
ESR	21 mm/h	Albumin	31 g/L
CRP	34 mg/L	Bilirubin	7 micromol/L
ALT	29 iu/L		
Alkaline Phosphatase	51 iu/L		

Ascitic fluid analysis:

Fluid albumin	22 g/L
Fluid glucose	4.2
Fluid gram stain	negative

Which of the following is the most likely cause of her ascites?

A. Alcoholic hepatitis
B. Heart failure
C. Peritoneal metastases from colon cancer
D. Portal vein thrombosis
E. Myxoedema due to hypothyroidism

C. Peritoneal metastases from colon cancer

The presenting symptoms in this scenario are fairly vague. The history does not help you narrow down your differentials for causes of ascites. The answer can be determined by working through the list of results. In particular, by calculating the serum-ascites albumin gradient (SAAG) you can work out the correct answer.

In this scenario, the SAAG is 9 (31-22). The only answer where this is possible is C as it is the only cause of ascites that has a SAAG < 11g/L.

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You are working on the gastroenterology ward in your local district general hospital, doing a ward round, when you come to examine one of your well known NASH (non-alcoholic steatohepatitis) cirrhosis patients. He's a 43-year-old man who had been admitted to the ward 3 days ago with a lower respiratory tract infection which has responded well to antibiotics. The patient appears clinically well from the end of the bed and has no complaints. He says that his ascites has been reasonably well managed over the past 4 years since diagnosis, needing only one ascitic drain during the admission that led to the diagnosis. On examination his chest appears to be clear today, his abdomen is soft and non-tender, and you can elicit a small amount of ascites on examination.

His blood results from this morning are as follows:

Hb	118 g/l
Platelets	102 * 10 /l9
WBC	4.5 * 10 /l9

Na +	128 mmol/l
K +	4.2 mmol/l
Urea	1.4 mmol/l
Creatinine	45 µmol/l
Bilirubin	20 µmol/l
ALP	134 u/l
ALT	24 u/l
Albumin	30 g/l

He tells you that his sodium has always given him a bit of trouble, and looking back at old results you noted that his usual sodium in clinic is consistently 133. His only medication is spironolactone 100mg twice daily

How should you manage this patient's hyponatraemia?

A. Carry on spironolactone and fluid restrict to 1L/day
B. Stop spironolactone and fluid restrict to 1L/day
C. Reduce spironolactone to 50mg twice daily and monitor
D. No change to medications - just monitor
E. Stop spironolactone and monitor

D. No change to medications - just monitor

The management of hyponatraemia in liver cirrhosis is a difficult area and a consensus hasn't been reached in all areas of management. However in patients with mild hyponatraemia (126-135 mmol/L) the recommendations are consistent. The British Society of Gastroenterology recommendations are as follows:

Sodium 126-135 mmol/L with normal creatinine - Continue normal diuretic regimen and observe, do not fluid restrict the patient.

Sodium 121-125 mmol/L with normal creatinine - International opinion is to continue diuretics, however, the British Society of Gastroenterology recommend a more cautious approach, and suggest either stopping diuretics or reducing the dose.

Sodium 121-125 mmol/L with raised creatinine (>150 mmol/L or >120 mmol/L and rising) - Stop diuretics and volume expand with human albumin solution 4.5%, gelofusine, or haemaccel

Sodium <121 mmol/L - Incredibly controversial, but the British society of gastroenterology suggest stopping diuretics and volume expanding with human albumin solution 4.5%, gelofusine, or haemaccel (which all contain sodium concentrations similar to that of normal saline).

They do, however, recognise that the level of evidence for this recommendation is 5 (or D) i.e. expert opinion, and further work needs to be done into the role of volume expansion and fluid restriction in cirrhosis.

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A young man known to the gastroenterology team with alcoholic cirrhosis and grade two varices on his last OGD 7 months ago is admitted as a medical emergency to the resuscitation part of Accident and Emergency with haematemesis and melaena. It started 45 minutes ago when he immediately called an ambulance. He is able to tell you that apart from the cirrhosis and varices he does not have any other problems, and he takes only thiamine, vitamin B co-strong and propranolol (but he is unsure of the dose). He says he has been well over the past week, with no symptoms of disease, and your screening questions reveal nothing important. He admits that he still occasionally drinks alcohol, but hasn't in the past 2 weeks, and that he used to have an opiate based drug addiction, for which he's been clean the past 3 years.

His initial observations are as follows:

Respiratory rate	22 breaths/minute
Heart rate	102 beats/minute
Temperature	36.2ºC
Blood pressure	85/33 mmHg
Saturations	100% on 4L nasal

Initial bloods

Hb	90 g/l
Platelets	72 * 10 /l9
WBC	6.4 * 10 /l9

His ECG shows RBBB and T wave inversion in leads V1 and V2 only. After 3L of fluid resuscitation:

Hb	78 g/l
Platelets	63 * 10 /l9
WBC	5.9 * 10 /l9
Na +	129 mmol/l
K +	4.2 mmol/l
Urea	14.2 mmol/l
Creatinine	75 µmol/l
Bilirubin	43 µmol/l
ALP	152 u/l
ALT	41 u/l
Albumin	29 g/l
PT	20.4 s
INR	1.7
APTT	38 s
Fibrinogen	0.8 g/L

His repeat observations are as follows:

Respiratory rate	18 breaths/minute
Heart rate	92 beats/minute
Temperature	36.4ºC
Blood pressure	105/65 mmHg
Saturations	99% on 2L nasal

What medication would you give him?

A. Fresh frozen plasma, Terlipressin, Ceftriaxone
B. Fresh frozen plasma, Terlipressin, Ceftriaxone, Platelets, Packed red cells
C. Fresh frozen plasma, Ceftriaxone, Platelets
D. Fresh frozen plasma, Terlipressin, Ceftriaxone, Packed red cells
E. Fresh frozen plasma, Terlipressin, Packed red cells, Platelets

A. Fresh frozen plasma, Terlipressin, Ceftriaxone

Guidelines issued by the British Society of Gastroenterology and NICE:

Packed Red Cells: Transfuse as per local massive bleeding protocol, recognising that over transfusion is as damaging as under transfusion and that a restrictive transfusion policy (aiming for 70-80 g/L is suggested in haemodynamically stable patients. As this patient is no longer vomiting blood and appears to be haemodynamically stable (as shown by the reducing pulse and rising blood pressure, transfusion is not required.

Platelets: Do not offer platelets to patients who are actively bleeding and are haemodynamically stable. To those still actively bleeding, offer platelets if count is <50 x 109/L. This patient does not need platelets as his platelet count is above the threshold at which it would be beneficial.

Fresh Frozen Plasma: Offer to those with a fibrinogen <1 g/L or a PT/APTT/INR over 1.5 times the normal value.

Antibiotics: All patients with acute variceal bleed should be offered antibiotics with Gram-negative cover.

Proton Pump Inhibitors: This is an issue that requires further research and thus has not been included in the question. The main concern is that in the small number of trials done on the role of PPIs in acute variceal bleed there has been no improvement in bleeding or survival. In addition to this it increases the risk of spontaneous bacterial peritonitis.

Terlipressin: Reduces failure to control bleeding and improves survival. The recommended dose is 2mg IV every 4 hours, but most centres reduce the dose to 6 hourly due to its vasoconstrictive effects causing painful hands and feet. Prolonged treatment with terlipressin has not been shown to improve survival further and thus treatment should be stopped shortly after satisfactory haemostasis has been achieved.

Question 42 of 235

A 45 year old man admitted to the gastroenterology ward to detox from excessive alcohol consumption. He is known to have alcohol dependence syndrome but has no other past medical history and no known diagnosis of alcoholic cirrhosis. On examination he is noted to have a distended abdomen with evidence of shifting dullness and a succussion splash. He is taken down for an ultrasound of his abdomen and an appropriate site for an ascitic tap is marked in the left iliac fossa by the radiologist. Before doing the ascitic tap you check his bloods from today, they are as follows:

Hb	83 g/l
Platelets	56 * 10 /l9
WBC	3.5 * 10 /l9
Na +	128 mmol/l
K +	3.2 mmol/l
Urea	2.2 mmol/l
Creatinine	45 µmol/l
Bilirubin	35 µmol/l
ALP	145 u/l
ALT	24 u/l
Albumin	32 g/l
INR	1.8
APTT	55 s
Fibrinogen	1.3 g/L

Before doing an ascitic tap, what needs to be done?

A. 1 unit of red blood cells
B. FFP till fibrinogen >1.5
C. IV vitamin K till INR <1.5
D. Nothing - the parameters are adequate
E. 1 pool of platelets

D. Nothing - the parameters are adequate question

Paracentesis is not contraindicated in patients with an abnormal coagulation profile. The majority of patients with ascites due to cirrhosis have prolongation of the prothrombin time and some degree of thrombocytopenia. There are no data to support the use of fresh frozen plasma before paracentesis although if thrombocytopenia is severe (<40 * 10 /l) most clinicians would give pooled platelets to reduce the risk of bleeding.

Question 43 of 235

A 35-year-old lady presents yellowing of her eyes and increased itchiness. This has increased gradually over the past three weeks. She is eating well and has no abdominal pain or fevers. She is known to have primary biliary cirrhosis and Sjogren's syndrome and she takes ursodeoxycholic acid and codeine. In addition, she is taking the maximum dose of chlorphenamine with no relief from her pruritus.

She works as a singer in a folk band and is on tour across the UK at present. She denies drinking any alcohol but admits to smoking occasional cannabis use. A member of her band is ill with a stomach bug.

On examination, she is icteric and has pallor. She has numerous erythematous linear scars across her arms and legs. She is alert and haemodynamically stable. Her abdomen has a palpable liver edge of 2cm below the subcostal margin with a nodule felt in the medial edge. Her abdomen is non tender and Murphy's sign negative with normal bowel sounds.

Hb	120 g/l	Na +	139 mmol/l	Bilirubin 75 µmol/l
Platelets	340 *10 /l 9	K +	4.8 mmol/l	ALP 180 u/l
WBC	10 * 10 /l 9	Urea	4.3 mmol/l	ALT 110 u/l
Neuts	8.4 * 10 /l 9	Creatinine	67 µmol/l	gamma#GT 80 u/l
Lymphs	0.5 * 10 /l 9	Eosin	0.1 * 10 /l 9	Albumin 27 g/l
Anti-mitochondrial antibody	positive	Anti-smooth muscle antibody	positive	AlphaFetoprotein30 mg/ml (normal0-10)
Hepatitis A IgM	negative	Hepatitis A IgG	positive	
HBsAg	negative	Hepatitis C PCR DNA	negative	

What is the likely cause of her deterioration?

A. Progression of primary biliary cirrhosis
B. Hepatic adenocarcinoma
C. Hepatitis A
D. Choledocholithiasis
E. Autoimmune hepatitis

B. Hepatic adenocarcinoma

Primary biliary cholangitis are at x20 risk of hepatocellular carcinoma

This lady with PBC has a deterioration of her liver function and a mass in the liver. She has no pain ruling out choledocholithiasis. Her serology suggests she has previous infection with Hepatitis A and no current infection. Her autoantibodies reflect her diagnosis of PBC and this would not explain the new palpable nodule. Her raised alpha fetoprotein and new nodule suggests likely hepatocellular carcinoma which is very common in people with autoimmune liver disease.

Question 44 of 235

A 55-year-old man presents with chronic diarrhoea. He describes a 10 month history of poorly formed stools, occurring 2-3 times a day which are difficult to flush. This is intermittently associated with generalised abdominal discomfort. He has not noticed any blood in his stools.

He also describes a 9kg unintentional weight loss within this 10 month period. He cannot understand this, as he still has a good appetite and he has not changed his diet.

He is still working full time as a joiner but has struggled on occasion when kneeling due to pains in his hips and knees and lethargy.

He has previously been fit and well, with a past medical history of childhood asthma. His two children are healthy. He has a family history ankylosing spondylitis, with his younger brother and dad suffering from the condition. He is not taking any regular medication.

On examination, he is pale and thin. Cardiopulmonary examination was unremarkable. He has generalised lymphadenopathy with small palpable lymph nodes in his cervical, axillary and inguinal region. There is no organomegaly on abdominal palpation but there is generalised tenderness. His observations are within normal limits, apart from his temperature which has been consistently between 37.6ºC and 37.8ºC.

Which investigation would most likely give the diagnosis?

A. Blood tests
B. Gastroscopy with biopsies
C. Colonoscopy
D. Stool culture
E. Joint xrays

B. Gastroscopy with biopsies

The likely diagnosis here is Whipple's disease. This is a notoriously difficult diagnosis in the clinical setting.

Whipple's disease is a rare multisystem disorder caused by the actinobacteria *Tropheryma whipplei*. It is more common in middle-aged, Caucasian men and in those with HLA-B27.

Common symptoms include diarrhoea, abdominal pain, arthralgia and weight loss.

Other symptoms can be divided by system:

- Cardiac: pericarditis, endocarditis, conduction defects
- Respiratory: pleurisy
- Neurological: seizures, myoclonus, cranial nerve lesions, dementia, confusion, insomnia and cerebellar signs.
- Other: lymphadenopathy, fever, skin hyperpigmentation

The diagnosis relies on biopsy of affected tissue, usually in the duodenum. This shows infiltration of the lamina propria with periodic acid-Schiff stain (PAS)-positive macrophages. This is achieved by biopsies taken during a gastroscopy.

Whilst blood tests may indicate malabsorption, this is not specific. Stool cultures would rule out infective causes of the diarrhoea and weight loss but again would not give a definitive diagnosis.

Whilst there are other important differentials to consider, including inflammatory bowel and coeliac disease, they cannot explain the diversity of symptoms.

Question 45 of 235

A 35-year-old man attends an outpatient gastroenterology clinic for review of his ulcerative colitis. At his last appointment, he reported that he felt his symptoms were well controlled as he was opening his bowels only once or twice per day, and there was no associated mucous or blood. However, he reported a new symptom of intense itch and felt his eyes had taken a yellow tinge. Today he reports noticing his urine being darker and stools paler.

He has no other medical problems apart from ulcerative colitis which is currently managed with mesalazine and azathioprine.

You arranged for some blood tests and a magnetic resonance cholangiopancreatography (MRCP) to be done after his last appointment. The results of which are shown below:

Hb	116 g/L	(115 - 160)
Platelets	352 * 10 /L 9	(150 - 400)
WBC	5.5 * 10 /L 9	(4.0 - 11.0)
CRP	10 mg/L	(< 5)
Bilirubin	75 µmol/L	(3 - 17)
ALP	250 u/L	(30 - 100)
ALT	45 u/L	(3 - 40)
Î³GT	79 u/L	(8 - 60)
Albumin	32 g/L	(35 - 50)

The MRCP showed 3 small gallstones in the gall bladder but no common bile duct dilation. It also did show multiple segmental strictures of the biliary tree.

From the information provided, what is the likely diagnosis?

A. Autoimmune hepatitis
B. Primary biliary cholangitis
C. Primary sclerosing cholangitis
D. Biliary colic
E. Mesalazine induced transaminitis

C. Primary sclerosing cholangitis

Ulcerative colitis + cholestatis (e.g. jaundice, raised ALP) → ? primary sclerosing cholangitis

Primary sclerosis cholangitis is associated with ulcerative colitis. It is a condition which causes inflammation and scarring of the intra- and extra-hepatic bile ducts. It classically presents with jaundice and pruritis and ALP/Î³GT are raised.

Autoimmune hepatitis commonly presents in young women. It would most likely present with a greater increase in ALT and AST compared to ALP.

Primary biliary cholangitis is a good differential as it also gives a cholestatic picture; however, is associated with other autoimmune diseases such as Sjogren's and does not result in progressive obstructive jaundice.

Biliary colic would also lead to a cholestatic picture on the blood tests. However, the MRCP showed no evidence of gallstones.

Mesalazine induced transaminitis is unlikely as the blood tests show a cholestatic picture. In addition, there is no mention that this is a new drug or the dose has been altered.

Question 46 of 235

A 58-year-old female presents to the clinic with a 6 month history of diarrhoea and abdominal pain. She has a past medical history of gastric cancer which was treated with a gastrectomy. She takes no regular medicines as she does not like taking drugs.

Blood results are as follows:

Hb	110 g/l	Na +	138 mmol/l	Bilirubin	24 µmol/l
Platelets	355 * 10 /l 9	K +	3.8 mmol/l	ALP	88 u/l
WBC	8.6 * 10 /l 9	Urea	6.8 mmol/l	ALT	44 u/l
Neuts	5.6 * 10 /l 9	Creatinine	48 µmol/l	γGT	105 u/l
Albumin	38g/l	Eosin	0.2 * 10 /l 9	CRP	4 mg/l
Anti TTG	negative	Faecal calprotectin	negative		

A blood film and haematinic screen was requested:

Blood film	Microcytes, macrocytes, hypersegmented neutrophils
MCV	85 fl
Red blood cell distribution width	Markedly elevated
B12	100 ng/l (low)
Folate	22.2 nmol/l (normal)
Ferritin	80 µg/L (low)

What is the most likely diagnosis?

A. Small bowel bacterial overgrowth
B. Coeliac disease
C. Dumping syndrome
D. Inflammatory bowel disease
E. Irritable bowel syndrome Next question

C. Dumping syndrome

Dumping syndrome features include osmotic diarrhoea, distension of the small bowel leading to abdominal pain, and intravascular depletion

The history of gastrectomy should alert you to the possibility of dumping syndrome. Rapid movement of food into the small bowel results in an intraluminal hyperosmolarity resulting in shift of fluid into the intestinal lumen, with plasma volume contraction and acute intestinal distension. The end result includes osmotic diarrhoea and distension of the small bowel leading to colicky abdominal pain. Large fluid shifts can also result in hypovolaemia.

The blood results are suggestive of a mixed microcytic and macrocytic anaemia. The MCV is normal due to a combination of microcytes and macrocytes. The elevated red blood cell distribution width confirms this. The patient is iron deficient (low ferritin) resulting in microcytic anaemia. They are also B12 deficient resulting in megaloblastic anaemia. Both have likely resulted due to the previous gastrectomy.

The stomach produces intrinsic factor which is essential for B12 absorption in the terminal ileum. The patient should therefore be on lifelong B12 injections.

The hydrochloric acid in the stomach is important for reducing Fe3+ (largely insoluble) to ferrous (Fe2+) iron which is more easily absorbed in the small bowel. Iron supplementation may also be required lifelong.

The normal anti TTG suggests that coeliac is not the diagnosis.

The normal inflammatory markers and negative faecal calprotectin suggests that inflammatory bowel disease is not the correct diagnosis.

Question 47 of 235

A 58-year-old man is reviewed on the ward. He has a past medical history of cirrhosis secondary to alcoholic liver disease. His current medications include furosemide, lactulose and spironolactone. He was admitted with decompensated liver disease one week ago. An ascitic drain was sited which has since drained 8 litres.

On examination he is clearly hypovolaemic with a blood pressure of 88/65 mmHg and heart rate 110 beats per minute. His abdomen is soft and non-tender. His chest sounds clear.

Blood results from 7 days ago are as follows:

Hb	115 g/l	Na +	138 mmol/l	Bilirubin	116 µmol/l
Platelets	285 * 10 /l 9	K +	3.5 mmol/l	ALP	128 u/l
WBC	12.8 * 10 /l 9	Urea	4.6 mmol/l	ALT	92 u/l
Neuts	8.2 * 10 /l 9	Creatinine	48 µmol/l	γGT	112 u/l
Lymphs	1.8 * 10 /l 9	Albumin	14 g/l		
Eosin	0.1 * 10 /l9				

Blood results from today are as follows:

Hb	112 g/l	Na +	128 mmol/l	Bilirubin	88 µmol/l
Platelets	295 * 10 /l 9	K +	3.3 mmol/l	ALP	118 u/l
WBC	10.8 * 10 /l 9	Urea	14.8 mmol/l	ALT	88 u/l
Neuts	6.2 * 10 /l 9	Creatinine	128 µmol/l	γGT	104 u/l

Lymphs	1.2 * 10 /l 9	Albumin	11 g/l
Eosin	0.1 * 10 /l9		

Ascitic tap results are as follows:

Neutrophil count	140 cells per mm^3

What is the most likely diagnosis?

A. Spontaneous bacterial peritonitis
B. Variceal bleed
C. Sepsis
D. Hypovolaemic shock
E. Paracentesis induced circulatory dysfunction

A. Paracentesis induced circulatory dysfunction

Paracentesis induced circulatory dysfunction can occur due to large volume paracentesis (> 5 litres). It is associated with a high rate of ascites recurrence, development of hepatorenal syndrome, dilutional hyponatraemia, and high mortality rat

The most notable laboratory features is the severe acute kidney injury. This followed shortly after large volume paracentesis (LVP). LVP is a major risk factor for paracentesis induced circulatory dysfunction, especially if the patient has not received a plasma expander.

Paracentesis induced circulatory dysfunction (PICD) is thought to occur secondary to fluid shifting after large volume (>5 litres) paracentesis, resulting in decreased circulating volume and renal dysfunction.

PICD is definitively diagnosed through laboratory results, with increases of more than 50% of baseline plasma rennin activity to > 4 ng/mL/h on the days 5-6 following paracentesis

One of the simplest ways to prevent PICD is to limit the volume of fluid removed to 5 to 6 L at a time. Current guidelines that recommend that if > 5 litres of ascitic fluid are removed then albumin should be administered as a plasma expander.

Spontaneous bacterial peritonitis is diagnosed with a neutrophil count > 250 cells / mm^3.

Although the patient is in shock, there is nothing to suggest that it is septic, or haemorrhagic in origin. The patient is in hypovolaemic shock however it is secondary to PICD which is the best answer in this case

Question 48 of 235

You are asked to review a 68-year-old male on the ward. The nursing staff are concerned that he has not opened his bowels in 4 days. He has a past medical history of COPD and is currently on the ward due to an infective exacerbation. On examination, his abdomen is distended. He has mild tenderness over the lower abdomen. There are no bowel sounds on auscultation. He states that he has been passing flatus regularly.

Blood results are as follows:

Hb	122 g/l	Na +	137 mmol/l
Platelets	285 * 10 /l 9	K +	3.0 mmol/l
WBC	12.5 * 10 /l 9	Urea	12.5 mmol/l
Neuts	10.5 * 10 /l 9	Creatinine	125 µmol/l
Lymphs	1.5 * 10 /l 9	CRP	128 mg/l

You order an abdominal x-ray:

Abdominal x-ray	Dilated small loops of bowel, no transition point visible. Visible gas in the rectum

What ant-emetic is most appropriate?

A. Cyclizine
B. Metoclopramide
C. Domperidone
D. Ondansetron
E. Hyoscine butylbromide

B. Metoclopramide

Prokinetic agents such as metoclopramide can be very effective in incomplete bowel obstruction. However they are contraindicated in complete bowel obstruction and in the presence of colic

The most likely diagnosis is a paralytic ileus. The presence of flatus and gas in the rectum suggests that there is not a complete mechanical obstruction. The absent bowel sounds are also indicativenof paralytic ileus.

Paralytic ileus has many causes. Some of the commoner causes include drugs, electrolyte abnormalities and post-surgery. The patient, in this case, has hypokalaemia which should be corrected.

Prokinetic agents such as metoclopramide can be very effective in incomplete bowel obstruction or paralytic ileus. However, they are contraindicated in complete bowel obstruction and in the presence of colic.

5HT3 antagonists (e.g ondansetron) and antimuscarinics (e.g. cyclizine and hyoscine butylbromide) can slow down the gut, cause constipation, and worsen an ileus.

Question 49 of 235

A 54-year-old lady presents with abdominal pain. The pain is over her central abdomen. She states that it came on earlier today and has progressively worsened. She is also nauseous and has experienced two episodes of non-bilious vomiting. She states that the abdominal pain is initially relieved after vomiting. however it returns shortly after. She has a past medical history of recurrent episodes of cholecystitis and is currently on the waiting list for a laparoscopic cholecystectomy. On examination her abdomen is not distended however it is very tender throughout. She is apyrexial.

Investigation results are as follows:

Hb	132 g/l	Na +	138 mmol/l	Bilirubin	42 µmol/l
Platelets	584 * 10 /l 9	K +	3.8 mmol/l	ALP	152 u/l
WBC	14.2 * 10 /l 9	Urea	6.2 mmol/l	ALT	52 u/l
Neuts	10.1 * 10 /l 9	Creatinine	48 µmol/l	γGT	124 u/l
Lymphs	2.2 * 10 /l 9	Albumin	38 g/l		
Amylase	198 U/L				

Abdominal ultrasound	Pneumobilia evident. Common bile duct diameter 8.2mm.
Chest x-ray	No evidence of pneumoperitoneum
Abdominal x-ray	Dilated loops of small bowel

What is the most likely diagnosis?

A. Cholecystitis
B. Pancreatitis
C. Paraduodenal abscess
D. Emphysematous cholangitis
E. Gall stone ileus

E. Gall stone ileus

Gallstone ileus is a mechanical small bowel obstruction which occurs due a gallstone obstruction most often in the ileum

The patient has presented with features of small bowel obstruction, namely the abdominal pain which is relieved by vomiting. It is important to remember that abdominal distension may be absent in small bowel obstruction (SBO). The most common cause of SBO is intra-abdominal adhesions, accounting for the majority of cases, followed by hernias and inflammatory structures (e.g. Crohn disease). Rarer causes include gallstone ileus.

The past medical history of recurrent cholecystitis, evidence of pneumobilia (air in the biliary tree), and the presence of SBO makes gallstone ileus the most likely unifying diagnosis. Gallstone ileus is an uncommon cause of a mechanical small bowel obstruction. It is a rare complication of chronic cholecystitis and occurs when a gallstone passes through a fistula between the gallbladder and small bowel before becoming impacted at the ileocaecal valve.

The amylase is only mildly raised and certainly not high enough to be diagnostic of pancreatitis. It is important to remember that amylase is non-specific and can be raised due to a variety of processes. Although pancreatitis can cause a sentinel loop, which is a short segment of adynamic ileus close to an intra-abdominal inflammatory process, it would not cause a small bowel obstruction.

A paraduodenal abscess and emphysematous cholangitis can also cause pneumobilia, however they would not result in small bowel obstruction, making gall stone ileus a better answer.

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A 78-year-old female is admitted with shortness of breath. She states that she is unable to perform basic activities of daily living without becoming breathless. The breathlessness is worse on lying flat and she needs to use three pillows to sleep. She also states that her legs have become much more swollen. Her drug history includes ramipril and bisoprolol. She drinks four units of whisky per week.

On examination she has crepitations to the midzones of her lung. Her JVP is raised at 8cm. She has pulsatile hepatomegaly. She has pitting oedema to the knees. Her blood pressure is 95/65mmHg and heart rate is 95 beats per minute.

You diagnose pulmonary oedema and treat her with IV furosemide. She responds well to treatment and her shortness of breath settles.

Blood results prior to discharge are as follows:

Hb	110 g/l	Na +	132 mmol/l	Bilirubin	24 µmol/l
Platelets	262 * 10 /l 9	K +	3.4 mmol/l	ALP	42 u/l
WBC	9.2 * 10 /l 9	Urea	10.2 mmol/l	ALT	1054 u/l
Neuts	6.4 * 10 /l 9	Creatinine	112 µmol/l	γGT	42 u/l
Lymphs	1.4 * 10 /l 9	Albumin	32 g/l		
Eosin	0.2 * 10 /l9				

What is the most likely cause of the deranged liver function tests?

A. Drug reaction to furosemide
B. Gall stones
C. Ischaemic hepatitis
D. Hepatitis A
E. Alcohol

C. Ischaemic hepatitis

In heart failure, both passive congestion of the liver secondary to right heart failure and decreased cardiac output, predisposes to ischaemic hepatitis

The patient has presented with acute heart failure with features of both right sided (e.g. peripheral oedema, raised JVP and pulsatile hepatomegaly) and left heart failure (e.g. pulmonary oedema). In heart failure, both passive congestion of the liver secondary to right heart failure and decreased cardiac output, predisposes to ischaemic hepatitis, which is the most likely explanation in this case. Ischaemic hepatitis tends to cause a transaminitis.

Her modest alcohol intake of 4 units per week makes alcoholic hepatitis unlikely.

Although hepatitis A is a possibility, there are no other features (e.g. fever, rash or arthralgia) or relevant travel history, making this a much less likely explanation.

Furosemide is a known cause of intrahepatic cholestasis which would typically present with raised ALP and bilirubin.

Gall stones are a well known cause of extrahepatic cholestasis which would typically present with raised ALP and bilirubin.

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A 25-year-old with recently diagnosed ulcerative colitis is started on mesalazine after a recent tapering of high dose steroids. Two weeks later, he develops severe pain in his epigastrium and right upper quadrant.

What is the most likely diagnosis?

A. Hepatitis
B. Acute pancreatitis
C. Duodenal ulceration
D. Flare in ulcerative colitis
E. Primary sclerosing cholangitis Next question

B. Acute pancreatitis

Mesalazine > sulfasalazine in terms of pancreatitis risk

Gastric side-effects are not uncommon with oral aminosalicylates, including diarrhoea, nausea, vomiting and exacerbation of colitis. In occasional cases, it can cause acute pancreatitis. Pancreatitis is significantly more common as a side-effect with mesalazine than sulfasalazine.

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A 44-year-old white male presents to his GP with a long history of joint pains in several joints, which has gradually affected his ability to work on his farm. He has seen another doctor before but no diagnosis was made. He has been taking ibuprofen with partial relief. He has now developed fever, diarrhoea and weight loss. He denies any genitourinary or eye symptoms. He does not use tobacco, alcohol or drugs.

On examination he has generalised lymphadenopathy and non-deforming arthritis. Small intestinal biopsy reveals macrophage infiltration into the lamina propria.

Which of the following is the most likely diagnosis?

A. Reactive arthritis
B. Sarcoidosis
C. Inflammatory bowel disease
D. Whipple's disease
E. Coeliac disease

D. Whipple's disease

Diarrhoea, weight, arthralgia, lymphadenopathy, ophthalmoplegia ?Whipple's disease

The patient described is most likely suffering from Whipple's disease. Diagnosis is made with small intestinal biopsy showing macrophages containing Periodic acid-Schiff (PAS) granules infiltrating into the lamina propria.

Choice 1: Reactive arthritis classically occurs following an infection, which this patient does not mention.

Choice 2: Sarcoidosis biopsy shows non-caseating granulomas, which is not mentioned.

Choice 3: On histopathology, ulcerative colitis shows acute and chronic inflammation of the mucosa leading to crypt abscess formation. Crohn's disease shows full-thickness inflammation with granuloma and lymphoid aggregate formation and skip regions. Neither are mentioned in the biopsy results.

Choice 5: A likely choice, however Coeliac disease biopsy results would mention lymphocyte infiltration (not macrophage infiltration), villous atrophy and crypt hyperplasia.

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An 86-year-old is brought to the emergency department by her husband after noticing her eyes have developed a yellow discolouration. On examination, she is disorientated to place and person but her husband reports this is not new.

She has a past medical history of Alzheimer's dementia and recurrent urinary tract infections with several antibiotic resistances. She recently complained of dysuria and increased urinary frequency. She has been started on antibiotics by her GP.

Initial blood tests show:

Bilirubin	45 µmol/L	(3 - 17)
ALP	178 u/L	(30 - 100)
ALT	33 u/L	(3 - 40)
Î³GT	103 u/L	(8 - 60)
Albumin	38 g/L	(35 - 50)

What is the most likely antibiotic she has been started on?

A. Amoxicillin
B. Ciprofloxacin
C. Co-amoxiclav
D. Nitrofurantoin
E. Trimethoprim

C. Co-amoxiclav

Co-amoxiclav is a well recognised cause of cholestasis

The examination and blood tests are consistent with jaundice with a cholestatic picture. Drugs are a common cause of cholestasis, especially certain antibiotics. As this patient has urinary tract infections with several antibiotic resistances, she may have been started on an antibiotic not conventionally used for a simple urinary tract infection.

Antibiotics that commonly cause cholestasis include flucloxacillin, co-amoxiclav, and erythromycin. The clavulanic acid component of co-amoxiclav makes it particularly prone to causing cholestasis, making it one of the most predominant causes of cholestasis. Therefore, **co-amoxiclav** is the correct answer.

Amoxicillin can cause hepatic disorders, including cholestasis, however, this risk is significantly lower than when it is combined with clavulanic acid to form co-amoxiclav. Therefore, this answer is incorrect.

Ciprofloxacin is not usually associated with cholestasis. It is associated with an increased risk of tendon injury and a prolongation of the QT interval. Other considerations for ciprofloxacin are that it can lower the seizure threshold and is associated with an increased risk of aortic aneurysms (especially in the elderly). It is also one of the antibiotics renowned for increasing the risk of *Clostridioides difficile*.

Nitrofurantoin tends to cause a hepatocellular picture of liver function test derangement rather than the cholestatic picture seen in this case.

Trimethoprim is not often associated with cholestasis. Adverse features include gastrointestinal upset and skin reactions. Also of note, there is a risk of teratogenicity in the first trimester.

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A 45-year-old man presents to the acute medical assessment unit with dysphagia. He reports a four-week history of relatively sudden-onset dysphagia to both liquids and solids and describes regurgitation and intermittent retrosternal pressure. The patient denies weight loss.

The patient examines normally and passes a bedside swallow assessment. What is the best investigation to confirm the diagnosis?

A. Barium swallow
B. CT chest/abdomen
C. Chest X-ray
D. Oesophageal manometry
E. Oesophagogastroduodenoscopy (OGD)

D. Oesophageal manometry

Dysphagia, regurgitation, no weight loss → ?achalasia

Dysphagia that is initially to both solids and liquids, regurgitation, no weight loss, and the relatively sudden-onset nature points towards achalasia.

Oesophageal manometry is the correct answer. It is the gold standard investigation to diagnose achalasia because it has the highest sensitivity. Manometry will show a high resting lower oesophageal pressure.

Barium swallow is the incorrect answer. A barium swallow can be used to diagnose achalasia, but it is not the gold-standard investigation - it has a lower sensitivity than oesophageal manometry. A barium swallow would show a dilated oesophagus that tapers smoothly to a beak-like narrowing at the gastro-oesophageal junction.

CT chest/abdomen is the incorrect answer. A CT cannot be used to diagnose achalasia because it is a functional not a structural problem. CT might be considered if there is a suspicion of extraluminal cancer that is compressing the oesophagus.

Chest x-ray is the incorrect answer. A chest X-ray would not confirm or exclude achalasia in the same way as a CT. However, this patient would likely receive one in their medical assessment to assess for thorax masses.

Oesophagogastroduodenoscopy (OGD) is the incorrect answer. An OGD would be useful to examine for intra-luminal masses or pathologies such as oesophageal cancer or Barrett's oesophagus but would not show achalasia as it cannot measure pressure.

Question 55 of 235

A 65-year-old man on the acute medical unit develops sudden onset, severe abdominal pain. He is an inpatient being managed for community-acquired pneumonia and has been in persistent atrial fibrillation during this admission; however, he is not normally on anticoagulants. His bowels have been opening regularly and are of a normal character and he has not vomited. He has a past medical history of type 2 diabetes and gastro-oesophageal reflux disease (GORD).

His oxygen saturations are 95% on 2L oxygen, respiratory rate is 20/min, heart rate is irregular at 103/min, blood pressure is 98/62mmHg, the temperature is 37.4ºC, and he remains alert. An abdominal examination reveals a widespread, exquisitely tender abdomen with some guarding and rebound tenderness.

Which of the following is the best next step in the management of this patient?

A. Anticoagulation
B. Emergency gastroscopy
C. Laxatives
D. Proton-pump inhibitors (PPIs)
E. Urgent laparotomy

E. Urgent laparotomy

Acute mesenteric ischaemia usually requires an immediate laparotomy, particularly if signs of advanced ischemia e.g. peritonitis or sepsis

This man is peritonitic as evidenced by the findings of guarding and rebound tenderness on examination. A diagnosis of acute mesenteric ischaemia must be considered in cases of sudden onset peritonitis on a background of atrial fibrillation (particularly when not on anticoagulation). As this man is particularly unwell, a referral to the surgical team and immediate laparotomy is indicated and is the correct answer.

As this man is in persistent atrial fibrillation, anticoagulation is likely to be indicated in the long term however this has no role in the acute management of acute mesenteric ischaemia. In fact, as this man is likely to require an urgent laparotomy, anticoagulation would only act to increase his risk of intraoperative bleeding.

Emergency gastroscopy would be of use in acute gastrointestinal bleeding. However, with pain being the primary complaint and bowels being normal in character, there is no evidence here that this is required.

Laxatives can be used to treat constipation which can cause acute abdominal pain, however, this man has been opening his bowels regularly and has signs of peritonism, both of which make constipation less likely.

PPIs may be useful in the treatment of gastritis which this man is at risk of due to his history of GORD however again this is not likely to present with signs of peritonitis and such deranged observations. The pain is also more likely to be epigastric rather than generalised.

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A 50-year-old male with a long history of cirrhosis secondary to chronic hepatitis C is brought to the emergency department by his partner with a 2-day history of increasing confusion.

He is drowsy but is rousable to voice. He is able to obey commands but is not oriented to where he is. Further examination revealed significant hepatic flap, multiple spider naevi on the torso, and mild abdominal distension with shifting dullness.

He is afebrile and his partner denies any history of recent infection.

His blood sugar level is 6.8 mmol/L

Which of the following should be started next?

A. Intravenous dextrose

B. Regular intravenous haloperidol
C. Oral rifaximin
D. Intravenous midazolam
E. Regular oral lactulose

E. Regular oral lactulose

Lactulose and rifaximin are used for the secondary prophylaxis of hepatic encephalopathy

Hepatic encephalopathy is a common complication of liver cirrhosis. It is a neuropsychiatric syndrome of varying severity, ranging from disturbances in cognitive function or sleep-wake cycle to coma. Potential precipitants include infection (including septicaemia and spontaneous bacterial peritonitis), gastrointestinal bleeding, and metabolic or electrolyte disturbances, among others.

Management of hepatic encephalopathy entails supportive care and treating potential precipitating factors.

Hepatic encephalopathy is largely believed to occur from accumulation of ammonia in the blood stream due to the livers decreased ability to detoxify ammonia that is produced and transported from the gastrointestinal tract. One of the aim of treatment of hepatic encephalopathy is therefore to reduce the production and absorption of ammonia from the gastrointestinal tract. Lactulose is often used as the first line therapy. It is usually administered orally, but can be given per rectum if the patient is too drowsy to tolerate oral intake. Mechanisms of action of lactulose include the reduction of intestinal ammonia load through its action as a cathartic and its ability to inhibit ammoniagenic coliform bacteria by acidifying the colonic lumen.

Despite its common use in the treatment of hepatic encephalopathy, evidence regarding the efficacy of lactulose is however conflicting.

Oral rifaximin is sometimes used in cases of hepatic encephalopathy that are refractory to treatment with lactulose. It is also used to prevent hepatic encephalopathy in patients who have recurrence despite lactulose.

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A 40-year-old man presents with jaundice.

His past medical history includes cellulitis 6 weeks ago which was treated with flucloxacillin, asthma and psoriasis. He drinks 30 units of alcohol per week and is a smoker of 10 pack years.

On examination he is icteric and obese, with a soft, non-tender abdomen and no palpable organs. Digital rectal examination is unremarkable.

Hb	145 g/l
Platelets	206 * 10 /l9
Amylase	30 u/l
Bilirubin	79 µmol/l
ALP	300 u/l
AST	30 u/l

What is the most likely cause of his jaundice?

A. Alcoholic cirrhosis
B. Gilbert's syndrome
C. Common bile duct gallstone
D. Drug induced cholestasis
E. Alcoholic hepatitis

D. Drug induced cholestasis

Flucloxacillin is a well recognised cause of cholestasis

This is a young gentleman who has presented with jaundice and obstructive LFTs. The key to this is in the question - flucloxacillin produced obstructive jaundice which can occur weeks after the initial treatment.

Gilbert's syndrome causes an unconjugated hyperbilirubinaemia.

A common bile duct stone would be expected to be associated with abdominal findings on examination.

Whilst he does have a high alcohol intake, there is no history of a recent alcoholic binge, and this would be associated with a transaminitis. Similarly, whilst he is at risk of alcoholic cirrhosis, this would be likely to be associated with findings of chronic liver disease, and elevation in the transaminases.

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You are asked to review a 63-year-old woman with new onset confusion. Her past medical history is significant for alcoholic cirrhosis of the liver. On examination, you note the patient is not orientated to time, place or person. She has asterixis and several telangiectasias over her trunk. Given the most probable diagnosis,

which of the following is the most appropriate medication to prescribe?

A. Lactulose
B. Senna
C. Macrogol
D. Rifaximin
E. Rifampicin Next question

A. Lactulose

Lactulose should be prescribed in all patients with suspected hepatic encephalopathy

Management of hepatic encephalopathy is designed to treat associated hyperammonemia. Of all the medications listed in this question, only lactulose is licensed for use in hepatic encephalopathy by the BNF. Lactulose works to inhibit production of ammonia in the intestine.

Rifaximin is a nonabsorbable derivative of rifampicin. Rifaximin is thought to affect the metabolic function of the intestinal flora. NICE guidance recommends the use of rifaximin to reduce the recurrence of episodes of overt hepatic encephalopathy.

Neomycin is another medication that is licensed for use in hepatic encephalopathy. It reduces ammonia levels by killing intestinal bacteria that produce ammonia.

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A 28-year-old woman is referred to hepatology with deranged liver function tests. She describes a twelve month history of fatigue. She has a past medical history of asthma and anxiety. Her regular medications include symbicort and sertraline. She smokes approximately 8 cigarettes per day and drinks 1-2 bottles of wine per week.

On examination, there is mild right upper quadrant tenderness and her sclerae are mildly icteric. There is no clinical evidence of ascites, asterixis or encephalopathy.

Blood tests:

Hb	133 g/L	Male: (135-180)Female: (115 - 160)
Platelets	174 * 10 /L 9	(150 - 400)
WBC	6.2 * 10 /L 9	(4.0 - 11.0)
Na +	137 mmol/L	(135 - 145)
K +	4.2 mmol/L	(3.5 - 5.0)
Urea	5.4 mmol/L	(2.0 - 7.0)
Creatinine	51 µmol/L	(55 - 120)
CRP	8 mg/L	(< 5)
Bilirubin	54 µmol/L	(3 - 17)
ALP	133 u/L	(30 - 100)
ALT	343 u/L	(3 - 40)
Î³GT	226 u/L	(8 - 60)
Albumin	31 g/L	(35 - 50)
Prothrombin time	14.2 seconds	(9.5 - 13.5 seconds)
Anti nuclear antibody	positive	
Anti smooth muscle antibody	positive	
Anti mitochondrial antibody	negative	
HIV	negative	
Hepatitis C antibody	negative	
Hepatitis B core antibody	negative	
Hepatitis surface antigen	negative	
Hepatitis B surface antibody	positive	
IgG	19.4 g/L	(6.0 - 16.0)
IgA	1.1 g/L	(0.8 - 2.8)
IgM	1.2 g/L	(0.5-1.9)
Epstein Barr virus	negative	
Cytomegalovirus	negative	
Serum ferritin	650 µg/L	(41-400)

An ultrasound abdomen demonstrates evidence of cirrhosis. What is the most likely diagnosis?

A. Alcoholic liver disease
B. Haemochromatosis
C. Hepatitis B
D. Type 1 autoimmune hepatitis
E. Type 2 autoimmune hepatitis

D. Type 1 autoimmune hepatitis

Antinuclear antibodies, anti-smooth muscle antibodies and raised IgG levels are characteristic of autoimmune hepatitis

Alcoholic liver disease is the wrong answer. We are told that she drinks approximately 1.5 bottles of wine per week. This is approximately 15 units of alcohol per week. While this is slightly above the recommended allowance of alcohol, it is not the most likely cause of cirrhosis given the clinical context.

Haemochromatosis is incorrect. Her ferritin is mildly elevated but this is a non-specific marker of inflammation and is not the most likely diagnosis based on the information provided.

Hepatitis B is incorrect. Her hepatitis B serology indicates previous vaccination rather than infection.

Type 1 autoimmune hepatitis is the correct answer. Antinuclear antibodies, anti-smooth muscle antibodies and raised IgG levels are characteristic of autoimmune hepatitis. Additionally, it is more common in young females.

Type 2 autoimmune hepatitis is incorrect. This is characterized by anti-liver/kidney microsomal type 1 antibodies (LKM1) and affects children only.

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A 64-year-old man presents to his general practitioner complaining of a change in the colour of his skin. He has a past medical history of hypertension. He drinks 13 units of alcohol weekly.

On examination, a 'slate-grey' appearance of his skin is noted. Blood tests:

Hb	154 g/L	Male: (135-180)Female: (115 - 160)
MCV	105 fl	(80-96)
Platelets	189 * 10 /L 9	(150 - 400)
WBC	4 * 10 /L 9	(4.0 - 11.0)
Na +	137 mmol/L	(135 - 145)
K +	4.2 mmol/L	(3.5 - 5.0)
Urea	5.2 mmol/L	(2.0 - 7.0)
Creatinine	89 µmol/L	(55 - 120)
CRP	4 mg/L	(< 5)
Bilirubin	12 µmol/L	(3 - 17)
ALP	70 u/L	(30 - 100)
ALT	32 u/L	(3 - 40)
Î³GT	44 u/L	(8 - 60)
Albumin	36 g/L	(35 - 50)
Ferritin	1100 ng/ml	(20-200)

Given the likely diagnosis, which complication is this patient most at risk of developing?

A. Chronic pancreatitis
B. Gastritis
C. Hepatocellular carcinoma
D. Macrophage activation syndrome
E. Pulmonary hypertension Next question

C. Hepatocellular carcinoma

Patients with haemochromatosis are at an increased risk of hepatocellular carcinoma

Hepatocellular carcinoma is correct. This patient has a 'slate-grey' appearance of his skin and a significantly raised ferritin and MCV without another obvious cause. The likely diagnosis is haemochromatosis. These patients are at risk of developing hepatocellular carcinoma.

Macrophage activation syndrome is incorrect. This is a complication of systemic autoimmune disease, most commonly, Adult-onset Still's disease (AOSD), which is characterized by hyperferritinemia. However, these patients tend to be systemically unwell with high fevers and cytopenias. The clinical history, in this case, does not point to this diagnosis.

Pulmonary hypertension is incorrect. This is a complication of thalassaemia, which can cause high ferritin levels. However, it also causes anaemia and a low MCV making the diagnosis unlikely in this case.

Chronic pancreatitis is incorrect. Alcohol misuse can cause high ferritin levels and this is a complication of excess alcohol use. However, this patient drinks within recommended guidelines, making this diagnosis unlikely.

Gastritis is incorrect. Alcohol misuse can cause high ferritin levels and this is a complication of excess alcohol use. However, this patient drinks within recommended guidelines, making this diagnosis unlikely.

Question 61 of 235

A 36-year-old male is referred to the acute medical unit by his general practitioner with reports of 5 bloody stools a day for the last 5 weeks. He has a past medical history of ulcerative proctitis but is not on any regular medications. Initial treatment with rectal mesalazine has proven ineffective in controlling the symptoms.

Observations:

- Heart rate 84 beats per minute
- Blood pressure 120/77 mmHg
- Respiratory rate 18/minute
- Oxygen saturations 98% on room air
- Temperature 37ºC

On examination, there is mild abdominal tenderness but no guarding and the abdomen is soft. Blood tests:

	106 g/L	Male: (135-180)Female: (115 - 160)
Platelets	444 * 10 /L 9	(150 - 400)
WBC	4.7 * 10 /L 9	(4.0 - 11.0)
Na +	136 mmol/L	(135 - 145)
K +	4.2 mmol/L	(3.5 - 5.0)
Urea	5.4 mmol/L	(2.0 - 7.0)
Creatinine	89 µmol/L	(55 - 120)
ESR	25 mm/h	(<20)

What is the most appropriate treatment at this point?

A. IV steroids
B. Intravenous ciclosporin
C. Oral aminosalicylate
D. Oral steroids
E. Topical steroids

C. Oral aminosalicylate

If a mild-moderate flare of distal ulcerative colitis doesn't respond to topical (rectal) aminosalicylates then oral aminosalicylates should be added

Oral aminosalicylates are the correct answer. The patient is experiencing a moderate flare of ulcerative colitis as defined by Truelove and Witts' classification of the severity of UC (4-6 bloody stools, ESR 20-30 and no features of severe disease). This is persisting despite initial treatment with rectal aminosalicylates. The next step in management is oral aminosalicylates.

IV steroids are not the correct answer. This is required for severe disease requiring inpatient admission.

Oral steroids are not the correct answer. This class of medication can be added to extensive ulcerative colitis that has not responded to trials of topical and oral aminosalicylates.

Intravenous ciclosporin is incorrect. This is an option for treating severe, refractory life-threatening ulcerative colitis in addition to IV steroids and can also be used to induce remission where steroids are contraindicated.

Topical steroids are not the right answer. If the patient had a persisting flare of moderate distal ulcerative colitis despite topical and oral aminosalicylates, then topical steroids could be added.

Question 62 of 235

A 74-year-old patient presents to the emergency department with three days of diffuse diarrhoea. He has a past medical history of hypertension and type 2 diabetes. He is on amlodipine and metformin. He has recently been treated with co-amoxiclav for a lower respiratory tract infection.

Observations:

- Heart rate 105 beats per minute
- Blood pressure 88/62 mmHg
- Respiratory rate 19/minute
- Oxygen saturations 97% on room air
- Temperature 38.2

On examination, there is mild generalized abdominal tenderness without peritonism. The patient is commenced on intravenous fluids.

Hb	138 g/L	Male: (135-180)Female: (115 - 160)
Platelets	412 * 10 /L 9	(150 - 400)
WBC	15.4 * 10 /L 9	(4.0 - 11.0)
Na +	131 mmol/L	(135 - 145)
K +	4.2 mmol/L	(3.5 - 5.0)
Urea	8.2 mmol/L	(2.0 - 7.0)
Creatinine	88 µmol/L	(55 - 120)
CRP	44 mg/L	(< 5)

A CT abdomen and pelvis is arranged, which demonstrates severe colitis. The diameter of the transverse colon is 4cm.

A stool sample demonstrates *Clostridium difficile* toxin.

What is the appropriate management?

A. Bezlotoxumab
B. Oral fidaxomicin
C. Oral vancomycin
D. Oral vancomycin and IV metronidazole
E. Surgery

D. Oral vancomycin and IV metronidazole

In life-threatening *C. difficile* infection treatment is with ORAL vancomycin and IV metronidazole

Oral vancomycin and IV metronidazole is the correct option. The patient has Clostridium difficile colitis. The combination of severe disease on CT imaging and hypotension suggests a lifethreatening infection. The correct initial management is with a combination of oral vancomycin and IV metronidazole.

Bezlotoxumab is incorrect. This is a human monoclonal antitoxin antibody, which binds to *Clostridium difficile* toxin B and neutralises its activity, preventing recurrence of infection. It is not well established in practice and would not be used to treat the acute infection.

Oral fidaxomicin is incorrect. This can be used for treating recurrent *Clostridium difficile* infection or as a second-line therapy in a first infection but would be inadequate to treat a life-threatening infection.

Oral vancomycin is incorrect. This is first line therapy for a first presentation of *Clostridium difficile* infection that is not life threatening.

Surgery is incorrect. While surgical options may be considered in this case if the patient failed to improve, it would be appropriate to try antibiotics and fluids in the first instance as the hypotension may respond to fluid resuscitation and antibiotics. There is no toxic megacolon (transverse colon diameter > 6cm) or ileus, which is reassuring.

Question 63 of 235

A 72-year-old man presents to the hospital with three days of profuse watery diarrhoea. He was treated successfully for an episode of *Clostridium difficile* diarrhoea eight weeks previously with oral vancomycin. He has a past medical history of hypertension and takes regular amlodipine.

Observations:

- Heart rate 88 beats per minute
- Blood pressure 120/77 mmHg
- Respiratory rate 18/minute
- Oxygen saturations 96% on room air
- Temperature 37.8°C

On examination, he has mild abdominal tenderness with no peritonism. Plain radiography of the abdomen is normal.

Blood tests:

Hb	136 g/L	Male: (135-180)Female: (115 - 160)
Platelets	189 * 10 /L 9	(150 - 400)
WBC	4.2 * 10 /L 9	(4.0 - 11.0)
Na +	137 mmol/L	(135 - 145)
K +	4.2 mmol/L	(3.5 - 5.0)
Urea	5.2 mmol/L	(2.0 - 7.0)
Creatinine	66 µmol/L	(55 - 120)
CRP	33 mg/L	(< 5)

A stool sample demonstrates toxin associated with C. difficile . What is the appropriate management?

A. IV vancomycin
B. Oral fidaxomicin
C. Oral vancomycin and IV metronidazole
D. Repeat oral vancomycin
E. Surgery

B. Oral fidaxomicin

A recurrent episode of *C. difficile* within 12 weeks of symptom resolution should be treated with oral fidaxomicin

Oral fidaxomicin is correct. This patient has a recurrence of diarrhoea with positive *C. difficile* toxin. This constitutes a recurrence of *C. difficile* infection. If this occurs less than 12 weeks after initial treatment then the patient should be treated with oral fidaxomicin.

IV vancomycin is incorrect. This is an unsuitable route of administration of vancomycin in *C. difficile* infection.

Oral vancomycin and IV metronidazole are incorrect. This would be a suitable option if the patient had a severe or life-threatening disease. However, the clinical, laboratory and radiology findings indicate mild-moderate disease.

Repeat oral vancomycin is incorrect. Oral fidaxomicin is the preferred option in recurrence of *C. difficile* infection at < 12 weeks.

Surgery is incorrect. The patient does not have an immediately life-threatening disease and therefore a colectomy is not indicated.

Question 64 of 235

A 68-year-old man is reviewed on an inpatient ward round by the palliative care team. He reports distressing diarrhoea. He has a past medical history of metastatic lung cancer. His medications include codeine, cyclizine, metoclopramide, morphine, and ondansetron.

On examination, there is no abdominal tenderness and the abdomen is soft. What is the likely cause of his symptoms?

A. Codeine
B. Cyclizine
C. Metoclopramide
D. Morphine
E. Ondansetron

C. Metoclopramide

Diarrhoea is a common side-effect of metoclopramide

Metoclopramide is correct. Diarrhoea is a common side effect of this medication, which has prokinetic properties in addition to its anti-nausea effect. Nausea is common in palliative care and may be disease or drug-related.

Codeine is incorrect. This medication commonly causes constipation rather than diarrhoea as a side effect. It is good practice to consider the co-prescription of a laxative when prescribing this medication, especially to the elderly.

Cyclizine is incorrect. This anti-emetic is more likely to cause constipation as a side effect. The mechanism by which it causes constipation is not definite but may be due to its partial anticholinergic mechanism of action. It can also cause tachycardia so should be prescribed with caution to patients who may tolerate this side effect poorly e.g. those with ischaemic heart disease or heart failure.

Morphine is incorrect. Again, constipation is a common side effect of opioid medications. They may also cause nausea.

Ondansetron is incorrect. This form of anti-emetic medication is preferred in those patients with chemotherapy-induced nausea. It causes constipation rather than diarrhoea as a side effect. It has been shown to delay colonic transit in research studies, which may explain its constipating effect.

Question 65 of 235

A 55-year-old woman is referred to the medical assessment unit with reports of flushing and weight gain. She has no past medical history, She smokes 10 cigarettes daily. She is from India.

On examination, she had a round face with centripetal obesity and a pronounced interscapular fat pad.

A chest x-ray revealed a rounded opacity in the right lung that is centrally located. What is the likely diagnosis?

- **A. Adenocarcinoma of the lung**
- **B. IgG4-related disease**
- **C. Lung carcinoid**
- **D. Squamous cell carcinoma of the lung**
- **E. Tuberculosis**

C. Lung carcinoid

Lung carcinoid may results in Cushing's syndrome secondary to ACTH release

Lung carcinoid is correct. The patient presents with weight gain and has features suggestive of Cushing's syndrome i.e. rounded facies, central obesity, and a 'buffalo hump'. In the context of flushing and a lung lesion, this suggests lung carcinoid is the cause of Cushing's syndrome. Lung carcinoid tumours are well-differentiated neoplasms, with neuroendocrine differentiation further divided into typical and atypical carcinoids. Histological classification is based on mitotic count per 2 mm² and the presence/absence of necrosis. Typical carcinoids are classified if they exhibit a mitotic count < 2 per 2 mm², absence of necrosis and a tumour size > 0.5 cm.

Adenocarcinoma of the lung is incorrect. These tumours tend to be peripherally located in the lungs of non-smokers. It is not commonly associated with paraneoplastic Cushing's syndrome.

IgG4-related disease is incorrect. This multisystem disease can affect almost any organ and can cause lung lesions. However, IgG4-related disease affecting the lung tends to be multifocal rather than solitary. It is not classically associated with the development of Cushing's syndrome. IgG4- related disease should always be considered in patients with mass lesions that present atypically.

Squamous cell carcinoma of the lung is incorrect. This may cause centrally located lung cancer in smokers. However, it is less commonly associated with Cushing's syndrome compared with lung carcinoid. It is more likely to cause hypercalcaemia. It would not explain the flushing the patient is experiencing.

Tuberculosis is incorrect. This may cause a lung lesion. However, it would be more likely to cause fevers, sweats, or weight loss rather than flushing. It may cause Addison's as a consequence of adrenal

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A 52-year-old woman attends the emergency department with a 2-month history of nausea and weight loss. There is no history of vomiting or diarrhoea. She has a past medical history of Sjogren's syndrome but has otherwise been well. She smokes 10-12 cigarettes/day and denies alcohol use.

On examination, her sclerae are jaundiced. She has normal vesicular breath sounds, and her heart sounds are normal. On palpation, her abdomen is soft and non-tender. There is no palpable lymphadenopathy.

Laboratory tests:

Hb	118 g/L	(115 - 160)
Platelets	307 * 10 /L 9	(150 - 400)
WBC	8.2 * 10 /L 9	(4.0 - 11.0)
Na +	136 mmol/L	(135 - 145)
K +	4.1 mmol/L	(3.5 - 5.0)
Urea	5.5 mmol/L	(2.0 - 7.0)
Creatinine	99 µmol/L	(55 - 120)
Bilirubin	36 µmol/L	(3 - 17)
ALP	156 u/L	(30 - 100)
ALT	33 u/L	(3 - 40)
Albumin	36 g/L	(35 - 50)
Glucose	17 mmol/L	(4 - 7)

CT abdomen and pelvis: there is evidence of global pancreatic enlargement.

What additional investigation will confirm the likely diagnosis?

- A. Faecal elastase
- B. Serum Ca 19-9 levels
- C. Serum IgG4
- D. Serum amylase
- E. Transferrin saturation

C. Serum IgG4

Autoimmune pancreatitis can be diagnosed with a combination of imaging and raised serum IgG4

This patient has a diagnosis of autoimmune pancreatitis. Clinical features suggestive of pancreatitis from this scenario include jaundice, nausea and pancreatic enlargement on the abdominal CT scan. Unlike acute pancreatitis that typically presents with abdominal pain and vomiting, autoimmune pancreatitis presents differently with symptoms that include nausea, weight loss and jaundice. In some patients, there may be extra-pancreatic manifestations or a new diagnosis of diabetes mellitus. It is associated with autoimmune conditions such as rheumatoid arthritis and Sjogren's syndrome. Antinuclear antibodies and **IgG4 levels** are raised in autoimmune pancreatitis and are commonly used in conjunction with radiographic imaging (such as a CT abdomen and pelvis) to confirm the diagnosis. Radiographic features of autoimmune pancreatitis include diffuse enlargement, giving the appearance of a 'sausage-shaped' pancreas.

Faecal elastase is a useful marker of pancreatic exocrine function. It is often raised in patients with chronic pancreatitis. Features of chronic pancreatitis include malaise, weight loss, abdominal pain and steatorrhoea. It has no role in the diagnosis of autoimmune pancreatitis.

Serum Ca 19-9 levels are used in the diagnosis of pancreatic malignancy. Patients with painless jaundice (as described in this question stem) and weight loss should be investigated for pancreatic malignancy. However, the presence of global pancreatic enlargement over a focal pancreatic mass on this patient's CT imaging combined with a past medical history of autoimmune disease makes pancreatic cancer less likely.

Unlike acute pancreatitis, **serum amylase** is not necessarily elevated in autoimmune pancreatitis, and therefore it is not the most sensitive diagnostic marker to request.

Transferrin saturation is used in the diagnosis of haemochromatosis, an autosomal recessive disorder of iron absorption. Iron can accumulate in the pancreas to give rise to diabetes mellitus and in the liver to cause chronic liver disease. Other common symptoms include arthralgia and bronze skin pigmentation that are not described here.

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A 28-year-old man with newly-diagnosed distal ulcerative colitis is seen in the gastroenterology clinic for a follow-up appointment. He was last seen 4 weeks earlier with abdominal pain and bloody diarrhoea and reported opening his bowels about 5 times a day without fever or vomiting. He was started on rectal mesalazine.

On return to the clinic this afternoon, he reports that his symptoms have remained the same and his bowels are still opening 4-5 times a day with cramping abdominal pain.

His observations are as follows:

- Temperature 37.2ºC
- Heart rate 88bpm
- Blood pressure 124/77mmHg
- Respiratory rate 14 breaths/min
- Oxygen saturations 99% on air

On examination, his abdomen is soft but mildly tender in the left iliac fossa. His cardiovascular examination is unremarkable.

What is the next best step in the management of this patient?

A. Intravenous hydrocortisone
B. Oral azathioprine
C. Oral mesalazine
D. Oral metronidazole
E. Topical corticosteroids

C. Oral mesalazine

If a mild-moderate flare of distal ulcerative colitis doesn't respond to topical (rectal) aminosalicylates then oral aminosalicylates should be added

The patient has been having a moderate ulcerative colitis flare with 4-6 episodes of bloody stools per day without any systemic symptoms. Having a diagnosis of distal ulcerative colitis means that rectal aminosalicylates can be used initially, such as rectal **mesalazine**. NICE guidelines recommend that if patients with distal ulcerative colitis do not respond to topical medication within 4 weeks an oral aminosalicylate should be tried, making oral mesalazine the correct answer.

Intravenous hydrocortisone is reserved for acute, severe flares of ulcerative colitis that are associated with systemic upset and warrant hospital admission.

Azathioprine is a drug used to maintain remission in Crohn's disease and ulcerative colitis. As this patient is not yet in remission and is still experiencing symptoms, it would be inappropriate to start treatment.

Oral metronidazole is used in the treatment of rectal and fistulating Crohn's disease. It is not used in the treatment of ulcerative colitis.

Topical corticosteroids can be tried in the treatment of distal ulcerative colitis. However, their indication is following unsuccessful remission with oral aminosalicylate use.

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A 51-year-old man presents to the emergency department with a 1-week history of increasing abdominal distention. He has recently been referred to the liver team by his GP following some derangement of his liver function tests performed in a routine Wellman health check. He reports some sporadic right upper quadrant pain but denies any fever or jaundice. He drinks no alcohol and takes no regular medications. There is no family history of note.

Clinical assessment demonstrates a distended abdomen with shifting dullness, caput medusae are visible around the umbilicus.

His blood tests are as follows:

Hb	130 g/L	Male: (135-180)Female: (115 - 160)
Platelets	100 * 10 /L 9	(150 - 400)
WBC	10.5 * 10 /L 9	(4.0 - 11.0)
Ferritin	350 ng/mL	(12-300)
Na +	137 mmol/L	(135 - 145)
K +	4.2 mmol/L	(3.5 - 5.0)
Urea	6.9 mmol/L	(2.0 - 7.0)
Creatinine	150 µmol/L	(55 - 120)
Bilirubin	25 µmol/L	(3 - 17)
ALP	150 u/L	(30 - 100)
ALT	60 u/L	(3 - 40)
Albumin	40 g/L	(35 - 50)

An ascitic tap is performed:

Albumin	25 g/L	(35 - 50)
WCC	3	(<50)

A liver ultrasound shows hypertrophy of the caudate lobe of the liver which is confirmed by a CT Triple Phase is performed to assess further. The CT report also mentions very poor visualization of the hepatic veins with hypoattenuation of the peripheral zones.

Given this patient's results, what is the most likely cause of his current clinical presentation?

A. Budd-Chiari syndrome
B. Haemochromatosis
C. Hepatocellular carcinoma
D. Spontaneous bacterial peritonitis
E. Undisclosed alcohol excess

A. Budd-Chiari syndrome

Budd-Chiari syndrome can cause decompensation of chronic liver disease

This patient has evidence of chronic liver disease and CT findings of poor visualization of the hepatic veins with hypoattenuation of the peripheral zones are consistent with a diagnosis of Budd Chiari syndrome. Increasing abdominal distention suggests that his liver disease has decompensated.

A ferritin level below 1000ng/mL is inconsistent with a diagnosis of haemochromatosis and is suggestive that the patient is being truthful about his alcohol intake.

The ascitic fluid analysis does not suggest a diagnosis of spontaneous bacterial peritonitis.

Hepatocellular carcinoma is a known complication of chronic liver disease but this patient does not show any ultrasound features consistent with such a diagnosis.

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A 36-year-old woman has presented to the hospital feeling generally unwell. She describes varying symptoms following eating which she states can vary day by day. On further questioning, her most common symptoms include early satiety, nausea, and bloating. However, she also describes diaphoresis, flushing, and palpitations.

Her past medical history includes poorly-controlled type 2 diabetes mellitus, obesity, anxiety, and a proximal Roux-en-Y bypass five months ago.

It is felt that she may be suffering from dumping syndrome.

What other feature would be in keeping with the diagnosis?

A. Bradycardia
B. Deranged liver function tests
C. Electrolyte deficiency
D. Hypoglycaemia
E. Jaundice

D. Hypoglycaemia

Dumping syndrome features include osmotic diarrhoea, distension of the small bowel leading to abdominal pain, and intravascular depletion

This question describes a young woman with recent gastric bypass surgery. Her symptoms include early satiety, nausea, and bloating which are often associated with the reduced gastric volume. However, autonomic symptoms of diaphoresis, flushing, and palpitations, following meals are indicative of early **dumping syndrome**.

Dumping syndrome is caused by a rapid passage of food contents from the stomach into the small intestine. Symptoms can be divided into early and late. Early symptoms occur minutes after eating and include the above symptoms plus diarrhoea and abdominal pain. Late symptoms occur 1-3 hours following a meal and are secondary to sudden insulin release in response to rapid carbohydrate movement. This commonly causes **hypoglycaemia** and symptoms related to it.

Bradycardia is not associated with dumping syndrome. Dumping syndrome symptoms can be separated into early and late symptoms. Palpitations are secondary to the early vasomotor component as well as the later adrenergic component of dumping syndrome, rather than bradycardia.

Dumping syndrome is not associated with hepatic dysfunction. Afferent loop syndrome, on the other hand, can be associated with it. Afferent loop syndrome is a complication of different gastric bypass surgeries including Roux-en-Y surgery. It is a partial or complete obstruction of the afferent limb of the gastrojejunostomy. Symptoms typically include epigastric pain and projectile vomiting. Significant obstruction can lead to obstruction of the common bile duct leading to **deranged liver function tests** and cholestasis.

Electrolyte deficiency may occur in dumping syndrome secondary to osmotic diarrhoea, however, this is not as common as findings of hypoglycaemia.

Jaundice may occur as a part of afferent loop syndrome, rather than dumping syndrome, secondary to common bile duct obstruction and cholestasis.

Gastrectomy: complications

- Dumping syndrome
    - early: food of high osmotic potential moves into small intestine causing fluid shift
    - late (rebound hypoglycaemia): surge of insulin following food of high glucose value in small intestine - 2-3 hours later the insulin 'overshoots' causing hypoglycaemia
- Weight loss, early satiety
- Iron-deficiency anaemia Osteoporosis/osteomalacia Vitamin B12 deficiency
- Other complications increased risk of gallstones
- increased risk of gastric cancer

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A 54-year-old male nurse suffers a needlestick injury whilst taking blood from a patient. The patient has a history of intravenous drug use however there is no known history of an infectious disease. The nurse himself has no past medical history. He has recently migrated from India and is unsure if he has been vaccinated for hepatitis B.

Results from his hepatitis B tests are as follows:

HBsAg	Negative
Anti-HBs	Positive
Anti-HBc	IgG positive

What is his hepatitis B status?

A. Acute infection
B. Chronic infection
C. Previous immunisation
D. Previous infection, carrier
E. Previous infection, not a carrier

E. Previous infection, not a carrier

HBsAg negative, anti-HBs positive, IgG anti-HBc positive - previous infection, not a carrier

This question examines the ability to interpret hepatitis screening serology.

HBsAg (hepatitis B surface antigen) detects whether a person is currently infected with hepatitis B.

Anti-HBs (hepatitis B surface antibody) status indicates immunity to the virus and is therefore present in people with either previous immunisation or infection.

Anti-HBc (hepatitis B core antibody) indicates previous and/or ongoing infection with hepatitis B. It can be further subdivided into IgG and IgM which indicates a timeframe for infection. IgM is present in infection of <6 months, and IgG in infections of >6 months.

This patient has positive anti-HBs and anti-HBc. This indicates antibodies to both the surface and core antigens which can only occur from a previous infection, rather than immunisation. As the surface antigen is negative, this means he is not a carrier.

An acute infection will have positive surface antigen testing and will be hepatitis B core antibody positive. As immunity has not been obtained in acute infection, anti-HBs will be negative.

As with acute infection, those with chronic infection will continue to have positive HBsAg and antiHBc but will be negative for anti-HBs as they have not gained immunity.

Vaccination provides immunity and therefore anti-HBs will be positive. HBsAg will be negative as the person has not come into contact with the virus itself, as will anti-HBc.

People with a previous infection will be positive for anti-HBs and anti-HBc but if they are a carrier, they will also be positive for HBsAg.

See the below table for a summary:

Acute infection	Chronic infection	Previous immunisation	Previous infection, carrier	Previous infection, not a carrier	
HBsAg	Positive	Positive	Negative	Positive	Negative
AntiHBs	Negative	Negative	Positive	Positive	Positive
AntiHBc	Positive	Positive	Negative	Positive	Positive

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A 25-year-old female undergraduate student presented to the gastroenterology outpatient clinic after referral from her GP. The referral letter stated that the patient had a three-year history of irritable bowel syndrome (IBS) and was complaining mainly of constipation. The GP had tried dietary advice and different laxatives, including senna and sodium docusate in full doses and a trial of macrogol with no benefit. The patient stated that the problem was mainly happening during exams. She underwent appendectomy 5 years ago with no postoperative complications. Blood investigations were as follows:

Hb	120 g/l
Platelets	310* 10 /l9
WBC	8* 10 /l9
Na +	140 mmol/l
K +	4.4 mmol/l
Urea	6.5 mmol/l
Creatinine	100 µmol/l

What is the next step in management?

A. Add tricyclic anti-depressant (TCA)
B. Add selective serotonin re-uptake inhibitor (SSRI)
C. Increase the dose current laxatives and advice to relax
D. Add lactulose
E. Add linaclotide

E. Add linaclotide

Recent changes in NICE guidelines suggested that when a patient with IBS has resistant constipation despite using multiple laxatives with proper doses, linaclotide (which is a guanylate cyclase-C receptor agonist) should be used as the next step in management. It works by increasing intestinal fluid secretion and transit and decreases visceral pain.

- Lactulose is not advised by NICE to be given in patients with IBS.
- Adding a TCA or SSRI is the next step after linaclotide if it is found to be of no benefit.

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A 25-year-old woman is reviewed in gastroenterology clinic six weeks after a recent hospital admission with abdominal symptoms. During the initial admission, the patient had presented with a 6 week history of very frequent bloody diarrhoea, a 10 kg weight loss and intermittent severe abdominal pain. Endoscopy had demonstrated a pan-colitis with histological features consistent with Crohn's disease. In addition, a CT scan of the abdomen had revealed a localised abscess associated with the proximal colon that had required percutaneous drainage and a course of IV antibiotics.

In addition to the above, the patient had been treated with IV hydrocortisone followed by a reducing course of prednisolone (initially 40 mg daily, tapered over 8 weeks). Steroid treatment had initially given a good response with the patients symptoms markedly improving during her hospital stay. Azathioprine treatment had been initiated prior to discharge in an attempt to maintain remission during wean of oral steroids.

In clinic, the patient reported being concordant with azathioprine therapy. However, she found that when she had reduced her prednisolone dose below 15 mg daily, she had recurrent abdominal pains and frequent bloody diarrhoea (although less severe than at presentation). She had also noted some pain to the left side of her anus when defecating.

Examination of the patients abdomen revealed some lower abdominal tenderness but without features of peritonitis. External examination showed an area of ulceration one centimetre lateral to the anus.

Haemoglobin	95 g / dL
Mean cell volume	98 fL
White cell count	16.9 x 10>3 / microlitre
Neutrophils	12.6 x 10>3 / microlitre
Platelets	451 x 10>3 / microlitre
Urea	8.0 mmol / L
Creatinine	97 micromol / L
Sodium	143 mmol / L
Potassium	3.9 mmol / L
CRP	125 mg / L

What is the appropriate next line treatment to induce remission in this patient?

A. Colectomy
B. Methotrexate
C. Metronidazole
D. Infliximab
E. Budesonide

D. Infliximab

This patient has multiple risk factors for a severe phenotype of Crohn's disease. In particular, her young age (< 40 years), female sex, the need for steroids to control her first exacerbation, the presence of an intra-abdominal abscess and likely perianal disease are all associated with an aggressive disease course. Another feature of concern not present in this patient is lesions of the upper GI tract.

While having given some improvement, the appropriate initial steroid treatment has failed to induce full remission. In cases with features of a severe phenotype, a rapid step-up to biological therapy is indicated. Infliximab is effective at inducing remission in moderate-to-severe Crohn's disease compared to placebo (remission rate at four weeks 81 % vs 17 %) and is also an effective treatment of peri-anal disease.

Budesonide is indicated to induce remission in mild-moderate disease confined to the small bowel or proximal colon and is associated with fewer side effects compared to other corticosteroids. Like azathioprine, methotrexate is effective in maintaining but not inducing remission from Crohn's disease.

Surgery in Crohn's disease is reserved to treat the consequences of failure of medical therapy only (for example, fistulas, abscesses or strictures). Ileocaecal resection can be first line treatment for discrete terminal ill disease, although anastomotic recurrence remains common. Metronidazole has a role in treating peri-anal Crohn's disease although would not be sufficient to induce remission in this patient.

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A 50-year-old woman presents with a 12-month history of fatigue and pruritus. She has a history of rheumatoid arthritis which was diagnosed 4 years ago and has been quiescent. Aside from a flare-up of her rheumatoid arthritis 2 years ago requiring a course of steroids, she has never been on any disease modifying drugs. Her present medications include naproxen 500mg BD and omeprazole 20mg OD.

On examination, there is evidence of scratch marks and icteric sclera. There is some fullness in the right upper quadrant of her abdomen on palpation.

Her blood tests are:

Hb	105 g/l	Na^+	137 mmol/l	Bilirubin	56 µmol/l
Platelets	190 * 10 /l 9	K^+	4.2 mmol/l	ALP	210 u/l
WBC	9 * 10 /l 9	Urea	2.6 mmol/l	ALT	60 u/l
Neuts	6.5 * 10 /l 9	Creatinine	77 µmol/l	γGT	80 u/l
Lymphs	1.4 * 10 /l 9	Albumin	32 g/l		
Eosin	0.2 * 10 /l9				

Her antibody screen reveals:

ANA	Positive at 1:160 titre
ANCA	Negative
AMA	Positive at 1:40 titre
ASMA	Negative

What drug can be used to provide symptomatic relief for the patient's pruritus?

A. Ursodeoxycholic acid
B. Desferrioxamine
C. Prednisolone
D. Cholestyramine
E. Pentoxifylline

D. Cholestyramine

Cholestyramine is used to provide symptomatic relief of pruritus in PBC

This patient has primary biliary cirrhosis (PBC). Patients with PBC is classically a middle aged woman and has a history of other autoimmune diseases, rheumatoid arthritis in this case. Antimitochondrial antibodies (AMA) are present in up to 95% of patients and a proportion of patients with PBC also have detectable antinuclear antibodies (ANA).

Symptoms of fatigue and pruritus are common in patients with PBC and in some cases can be extremely debilitating. Cholestyramine is used first line to sequestrate bile acid salts in the enteric lumen and to provide relief for the patient's pruritus. Other drugs such as colestipol or rifampicin can also be used. Ursodeoxycholic acid is used to slow the progression of the disease and patients on this medication have shown biochemical and histological improvement.

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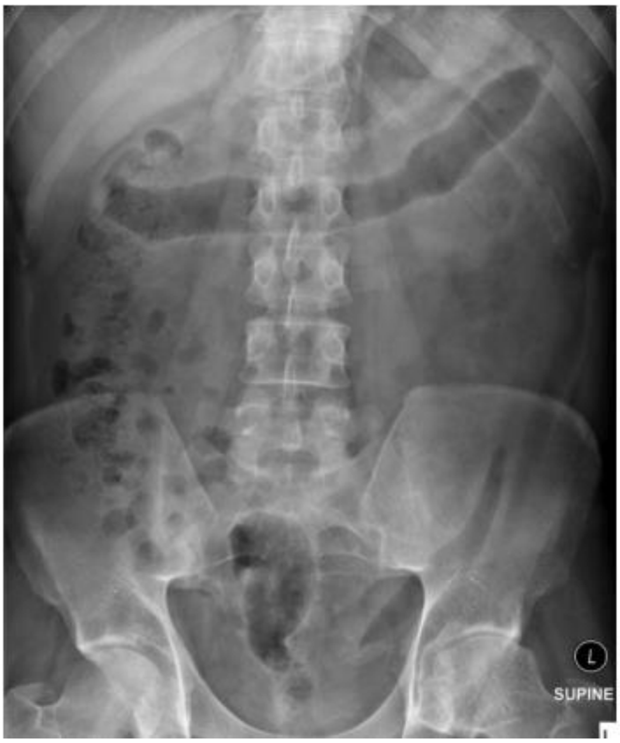
A 42-year-old man presents with a three week history of abdominal pain and diarrhoea to the Emergency Department. Over the past 48 hours the pain has become quite severe and is scored at 8/10 in terms of severity. The pain is worse in the left lower quadrant and has no obvious aggravating features. He is now passing around 8 loose stools per day. He has a history of irritable bowel syndrome and depression. He also completed a course of flucloxacillin for a skin abscess around 6 weeks ago.

On examination he is diffusely tender across the lower abdomen but there is no guarding. The heart rate is 102/min, blood pressure 104/66 mmHg and temperature 37.2ºC.

Bloods show the following:

Hb	13.4 g/dl	Na +	144 mmol/l
Platelets	421 * 10 /l 9	K +	4.3 mmol/l
WBC	9.6 * 10 /l 9	Urea	6.8 mmol/l
Creatinine	72 µmol/l		
CRP	62 mg/l		

An abdominal film is requested:



What is the most likely diagnosis?

A. Crohn's disease
B. Acute diverticulitis
C. Ulcerative colitis
D. Clostridium difficile colitis
E. Coeliac disease

C. Ulcerative colitis

The abdominal film shows a featureless transverse colon and distal descending colon and proximal sigmoid consistent with the lead pipe sign of chronic ulcerative colitis (red arrows). Additionally chronic sacroiliitis with ankylosis on the left and partial ankylosis on the right (yellow arrows). Asymmetric left hip joint arthropathy is also seen.

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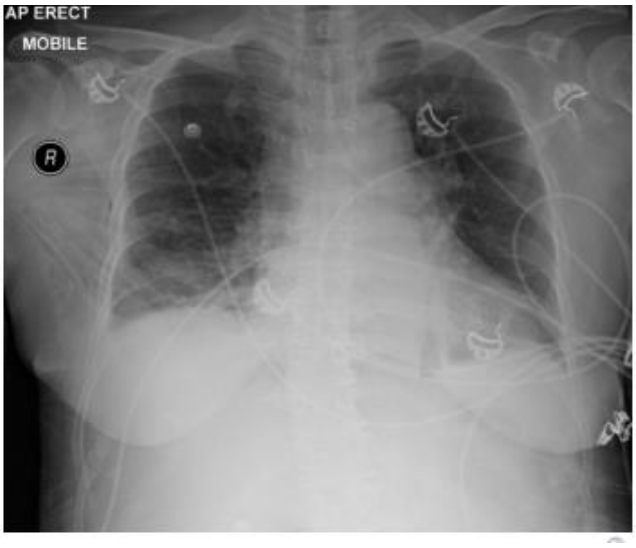
You are asked to review a 65-year-old woman who has become breathless on the surgical ward. Earlier in the day she had a laparoscopic cholecystectomy for gallstone disease. A chest x-ray has already been obtained:



What complication has developed?

A. Pneumothorax
B. Intestinal perforation resulting in pneumoperitneum
C. Subcutaneous emphysema
D. Pulmonary embolism
E. Acute respiratory distress syndrome

C. Subcutaneous emphysema

This radiograph demonstrates subcutaneous (surgical) emphysema which is a known complication of laparoscopic surgery. If the anterior chest wall is affected air can outline the pectoralis major muscle, giving rise to the 'ginkgo leaf' sign.

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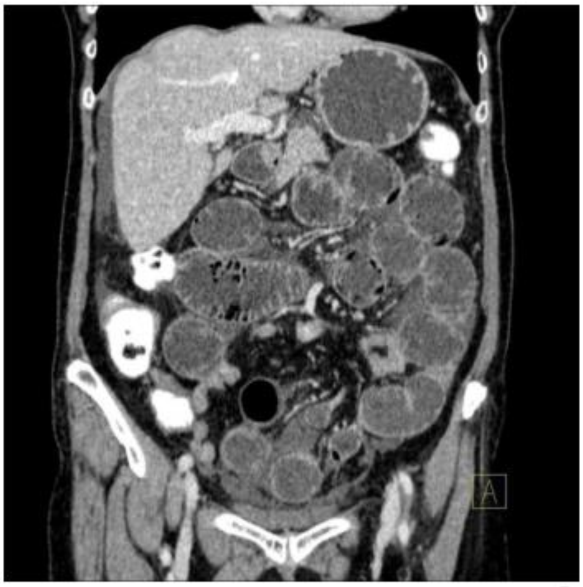
A 60-year-old woman presents to the Emergency Department with abdominal distension and pain. This has been getting progressively worse over the past 24 hours. Her past medical history includes a hysterectomy 5 years ago with ovarian preservation. A CT of her abdomen is requested:



What is the most likely diagnosis?

- A. Autosomal dominant polycystic kidney disease
- B. Pseudomyxoma peritonei
- C. Metastatic renal cell cancer
- D. Meigs' syndrome
- E. Small bowel obstruction

E. Small bowel obstruction

The CT shows small bowel obstruction with small bowel loops proximally (duodenum and jejunum) with abrupt transition to intestinal segment of normal caliber. Presence of small amount of free fluid intracavity. In this lady the cause was adhesions secondary

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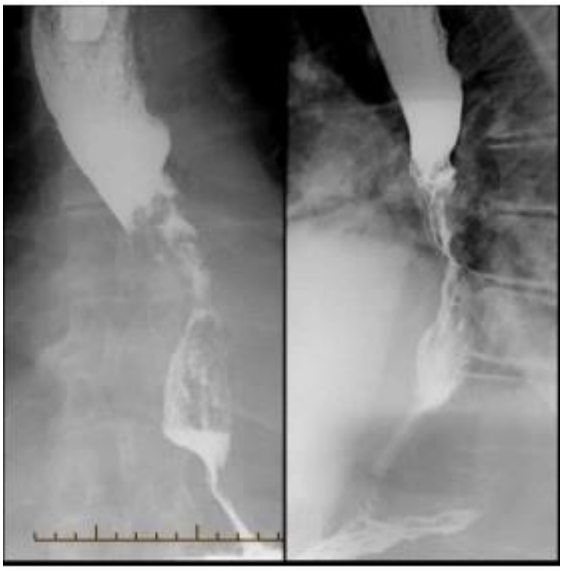
A 68-year-old man is investigated for dysphagia. This has been getting progressively worse for the past 3-4 months. It occurs mostly with foods such as bread and meat and is associated with some odynophagia. He has vomited once after eating. There is no history of weight loss or anorexia. His bowel motions have not changed in terms of consistency or colour. A barium swallow is performed:



What is the most likely diagnosis?

- A. Gastric cancer
- B. Achalasia
- C. Gastro-oesophageal reflux disease
- D. Barrett's oesophagus
- E. Oesophageal cancer

E. Oesophageal cancer

The barium swallow shows 5cm of irregular narrowing of the mid-thoracic oesophagus with proximal shouldering, consistent with oesophageal cancer.

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A 26-year-old primigravida lady is brought to the hospital at 17 weeks gestation with worsening confusion. Her husband states that she has been vomiting profusely for the past 3 weeks. She has been unable to eat but she has managed to keep down small amounts of fluid.

Her past medication history is unremarkable and she takes no regular medications.

On examination, she is afebrile. Her pulse is 108bpm and her blood pressure is 103/68mmHg. She is alert but disorientated to time and place. Cranial nerve examination is remarkable for bilateral VIth nerve palsy and multi-directional nystagmus. Peripheral neurological examination reveals MRC grade 4/5 power in both lower limbs. Her lower limb jerks are absent and there is bilateral loss of distal vibration sense. Her plantar responses are downgoing.

A set of blood tests are requested:

Hb	117 g/l	Na +	132 mmol/l
Platelets	362 * 10 /l 9	K +	2.8 mmol/l
WBC	12.3 * 10 /l 9	Urea	8.9 mmol/l
Neuts	10.1 * 10 /l 9	Creatinine	112 µmol/l
Lymphs	1.9 * 10 /l 9	CRP	17 mg/l
Eosin	0.01 * 10 /l 9	Glucose	3.1 mmol/l

What is the most appropriate treatment?

- A. Folic acid

B. Niacin
C. Vitamin B12
D. Intravenous dextrose
E. Thiamine

E. Thiamine

The patient has presented with symptoms and signs consistent with Wernicke's encephalopathy (confusion, nystagmus) and dry beriberi (peripheral neuropathy). There is a clinical association between Hyperemesis gravidarum and thiamine deficiency, particularly when it is prolonged and normal dietary intake is compromised. This patient should receive high-dose parenteral thiamine without delay.

Intravenous dextrose administered before correction of thiamine deficiency may exacerbate her condition and should be avoided.

Subacute combined degeneration of the cord would be one of the major differentials in this case. However, the downgoing plantar responses and presence of ocular signs make this less likely.

Niacin is used to treat pellagra. This patient is confused, but there is no mention of diarrhoea or a skin rash in the vignette.

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A 47-year-old lady is admitted to hospital with a 5-day history of abdominal pain, fever, anorexia, and malaise. Her past medical history is remarkable for depression and there is a long history of alcohol excess.

On examination, the patient is jaundiced and unwell. Her temperature is 37.9ºC, her pulse is 106bpm and her blood pressure is 97/61mmHg. Her chest is clear but her abdomen is distended. There is right upper quadrant tenderness on palpation of the abdomen without overt peritonism, and evidence of shifting dullness on percussion. A hepatic bruit is also present.

Her bloods are as follows:

Hb	99 g/l	Na +	128 mmol/l	Bilirubin	92 µmol/l
Platelets	113 * 10 /l 9	K +	4.8 mmol/l	ALP	138 u/l
WBC	11.2 * 10 /l 9	Urea	7.9 mmol/l	AST	168 u/l
Neuts	8.7 * 10 /l 9	Creatinine	103 µmol/l	ALT	82 u/l
Lymphs	2.3 * 10 /l 9	γGT	208 u/l		
INR	1.8	Albumin	28 g/l		

An ascitic tap is performed and reported as having a neutrophil count of 56 cells/mm³. She is started on vitamin supplementation, nutritional support, and oral prednisone.

After 7 days the patient is re-assessed. She remains significantly jaundiced and has become progressively more confused. Her repeat blood tests show:

Na +	131 mmol/l	Bilirubin	127 µmol/l
K +	4.2 mmol/l	ALP	172 u/l
Urea	11.2 mmol/l	ALT	76 u/l
Creatinine	134 µmol/l	γGT	168 u/l
INR	2.3	Albumin	21 g/l

Her Lille score is calculated as 0.52. How should her treatment be altered?

A. Discontinue prednisolone and add etanercept
B. Discontinue prednisolone and add pentoxifylline
C. Continue prednisolone and add pentoxifylline
D. Discontinue prednisolone and continue supportive care
E. Palliate

D. Discontinue prednisolone and continue supportive care

Prednisolone 40mg for 28 days has been shown to increase survival in patients with severe alcoholic hepatitis. However, response to steroid treatment should be re-evaluated at 7 days, as not all patients respond and continuing immunosuppressive treatment in patients who are unlikely to benefit may prove deleterious.

As a rule of thumb, an increase in bilirubin over the first 7-days indicates non-response. The Lille score is a more formal method of assessment that takes into account a range of demographic and laboratory values to give a more nuanced evaluation. A Lille score of > 0.45 signifies the failure of prednisolone and justifies its discontinuation. The 6-month survival rate for this group of patients is approximately 25%, so supportive treatment should be continued.

Switching non-responders to pentoxifylline has not been shown to be beneficial.

The inflammatory cytokine TNF-α is thought to be key to the pathophysiology of acute alcoholic hepatitis and several studies have investigated the efficacy of anti-TNF-α therapies in this setting. Unfortunately, a number of studies have shown that infliximab and etanercept are associated with higher mortality rates at 2 and 6 months, largely secondary to the development of acute infection. As a result, these agents are not recommended for use in alcoholic hepatitis.

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The Emergency Medical Team were summoned by the urology team. A 62-year-old gentleman was admitted to the care of the urology team a few hours ago complaining of haematuria and loin pain. A CT scan of his abdomen revealed the presence of renal calculi and he was being treated conservatively with intravenous saline hydration therapy, as well as intravenous paracetamol 1g QDS and IV morphine 10mg stat boluses when required. Approximately an hour ago he complained of distressing nausea and vomiting and was administered metoclopramide 10mg IV bolus with a good response. However, over the last few minutes he developed severe neck stiffness and was unable to open his eyelids. His jaw was locked and his tongue was protruding. He had a past medical history comprising panic attacks, type 2 diabetes mellitus, hypertension, hypercholesterolaemia, chronic kidney disease stage 3 and osteoarthritis. In addition to the above medication, his drug history included diazepam 2mg TDS PRN, metformin 500mg TDS, gliclazide 80mg OD, ramipril 5mg OD, atorvastatin 20mg ON and naproxen 500mg BD PRN.

Examination revealed the presence of an acutely compromised gentleman. His head was rotated to the left and fixed in position, with a protruding tongue and a fixed locked jaw. His eyelids were closed. His back was arched, his upper limbs were flexed and his lower limbs were extended. He was very distressed but was sustaining respiratory effort with a respiratory rate of 32/min and oxygen saturations of 96% on air. His chest was clear to auscultate and cardiovascular examination was unremarkable except for the presence of a tachycardia of 132 bpm. His blood pressure was 122/78 mmHg. His temperature was 37.4ºC. He was unable to speak but seemed to be fully alert. His capillary blood sugar was 8.2.

A 15 litre/min non-rebreathe oxygen mask was attached; he did not tolerate a Guedel airway insertion. Intravenous access was secured.

What is the next single best immediate management step?

A. Crash bleep the anaesthetics team to assist in supporting his airway
B. Administer IM lorazepam
C. Administer IV adrenaline
D. Administer IV dantrolene
E. Administer IM procyclidine

E. Administer IM procyclidine

may lead to complete airway collapse. First line treatment in the UK is procyclidine, a rapid acting anticholinergic, with benzodiazepines and anticholinergic antihistamines 2nd and 3rd line respectively. One may argue that option 1 is the most appropriate option, but in the above situation his airway was intact (just!) and he was sustaining respiratory effort. Anaesthetic input would assist with securing a compromised airway, but would not address the cause of the compromise. Prompt treatment may prevent further deterioration

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A 68-year-old man who is on aspirin following a myocardial infarction 2 years ago has been admitted and treated successfully endoscopically for a non-variceal upper gastrointestinal bleed with clips and adrenaline.

What should be done in the acute phase with regards to this patient's aspirin therapy?

A. Stop aspirin
B. Replace aspirin with clopidogrel
C. Continue aspirin
D. Replace aspirin with low molecular weight heparin
E. Replace aspirin with unfractionated heparin

C. Continue aspirin

NICE guidelines indicate that in patients following an upper gastrointestinal bleed in whom haemostasis has been achieved, aspirin should be continued when it is being used for secondary prevention of vascular events.

There is no need to replace aspirin with clopidogrel or either form of aspirin or heparin in this setting.

Question 82 of 235

A 62-year-old man presents with abdominal pain, jaundice and a distended abdomen. He has a background of liver cirrhosis secondary to chronic liver disease. He also has diverticular disease, ischaemic heart disease and hypertension. On admission, he was found to be jaundiced, confused and had ascites and was treated for spontaneous bacterial peritonitis with an ascitic sample sent to the microbiology lab. His abdomen is very tender as well. The lab calls and reports a mixed growth.

What is the most likely cause?

A. Sponatenous peritonitis
B. Urosepsis
C. Contamination
D. Sepsis secondary to respiratory infection
E. Perforation

A. Perforation

The correct answer is a perforation. Ascitic fluid is normally sterile and any growth of organisms is indicative of infective pathology. Mixed growth suggests a large communication of microorganisms into the abdominal cavity, which makes perforation the most likely cause. Spontaneous peritonitis occurs with bacterial translocation across the bowel and can usually identify a single species of normal gut flora as a pathogen, whilst urosepsis might be considered the source with a urinary pathogen such as Proteus mirabilis.

Question 83 of 235

A 48-year-old female who was diagnosed with type 2 diabetes mellitus six months ago presents with fatigue. She had a long standing history of obesity and a recent high cholesterol level of 6.5 mmol/l.

She doesnt smoke, and drinks alcohol on special occasions only. She has one sister who has Graves' disease.

The only abnormality noted on the examination is a soft yellow plaque on the eyelids bilaterally. The following investigations had been ordered:

Hb	13 g/dl
Platelets	175 * 10^9/l
WBC	5 * 10^9/l
MCV	80 fl
MCH	0.4 fmol/cell
MCHC	20 mmol/l
Na+	135 mmol/l
K+	4 mmol/l
Creatinine	80 µmol/l
Urea	3 mmol/l
ESR	40 mm/hr
Alkaline phosphatase	300 IU/l
Gamma glutamyl transpeptidase	100 IU/l
Alanine transaminase	60 IU/l
Aspartate transaminase	15 IU/l
Bilirubin	10 µmol/l
Serum albumin	40 g/l
Serum cholesterol	7 mmol/l

What is the most useful additional investigation to reach a diagnosis?

A. Immunoglobulin level
B. Liver biopsy
C. Antimitochondrial antibodies (AMA)
D. Viral screening
E. Abdominal ultrasound

C. Antimitochondrial antibodies (AMA)

Primary biliary cholangitis - the **M** rule

Ig**M**

anti-**M**itochondrial antibodies, **M**2 subtype

Middle aged females

This lady has primary biliary cirrhosis (PBC) as evidenced by the history of fatigue, elevated liver enzymes, xanthelasma, history of autoimmune disease in the family, raised ESR, and the elevated cholesterol level.

Synthetic functions of the liver wouldnt be deranged until late in the disease process and usually indicate a poor prognosis. The same applies to jaundice.

AMA is present in 95% of cases and is highly specific for PBC. When combined with a clinical history fitting the diagnosis no additional tests need to be made.

Liver biopsy is increasingly losing support as a diagnostic tool for the following reasons:

1. The high value and the specificity of AMA

2. The changes in the liver are focal

Serum IgM is elevated in pure PBC but its neither sensitive nor specific. It is useful in cases where doubt exists. There is nothing in the history or the clinical examination to suggest that this lady is at an increased risk of viral hepatitis. Abdominal ultrasound would help to exclude obstructive duct lesions. However it would not be as valuable in diagnosing PBC as AMA.

Question 84 of 235

A 50-year-old lady is seen in the Emergency Department with abdominal pain and vomiting. She has had generalised colicky abdominal pain, worst in the centre of her abdomen, for the last 2 days. She started vomiting six hours ago and has been unable to keep fluids down. She has not opened her bowels for 3 days and her abdomen has become distended. She does not recall passing flatus today.

She is known to have ovarian cancer with metastases to her bones, liver and peritoneum. She has had a previous abdominal hysterectomy and bilateral salpingoophorectomy and two previous courses of chemotherapy. She has no other past medical history.

On examination she has a diffusely tender tympanic abdomen with no rebound or guarding. She has palpable peritoneal nodules, a 2 cm liver edge and mild shifting dullness. Bowel sounds are hyperactive. She has reduced air entry at both lung bases and mild ankle oedema.

An abdominal x-ray demonstrates dilated loops of small bowel and evidence if ascites. A CT scan of the abdomen with contrast shows small bowel obstruction with an ileal transition point adjacent to a site of peritoneal metastasis. Her liver lesions appear stable and there is no evidence of ureteric obstruction.

An NG tube is inserted and she is started on intravenous fluids and analgesia.

Which is the best initial measure to help resolve the obstruction?

A. Bowel rest alone
B. Dexamethasone
C. Chemotherapy
D. Metoclopramide
E. Surgery

B. Dexamethasone

This lady has complete small bowel obstruction related to her metastatic cancer. Steroids may help to reduce the inflammation surrounding the tumour and resolve the obstruction.

Bowel rest is an essential component of the treatment of small bowel obstruction but unlike in cases of obstruction due adhesions, it is unlikely to resolve the obstruction when used in isolation.

Metoclopramide is a prokinetic anti-emetic used in pseudo-obstruction to help stimulate the bowel. It should not be used in small bowel obstruction due to the risk of perforation and an alternative anti-emetic should be used.

Chemotherapy and surgery are both important considerations to treat tumour-related bowel obstruction that is not resolving with steroids. This should be carefully planned with the input of consultant oncologists and surgeons, with the patient's fitness for these procedures assessed.

Reference: Tuca et al. Malignant bowel obstruction in advanced cancer patients: epidemiology, management, and factors influencing spontaneous resolution. Cancer Management and Research. 2012.

Question 85 of 235

An 18-year-old migrant from Syria presents to you after recent successful application for asylum. He has no family with him in the country. Since he can remember he gets severe burning pain and tingling in the hands and feet, particularly after exertion or when weather temperatures are extremely hot or cold. He remembers once having a transient episode of slurred speech and right facial droop that lasted less than 24 hours. He has never had the opportunity to consult about his problems. Physical examination reveals a rash of blue-black telangiectasia across his trunk and a mid-systolic murmur heard loudest in the apex and radiating into the axilla. The examination is otherwise unremarkable. He recalls that his younger brother, who died some years ago, had similar problems and his sister, who he is no longer in touch with, seems to have not had any problems

Which of the following findings is consistent with the underlying diagnosis?

A. Positive anticentromere antibodies
B. Deficiency in plasma levels of alpha-galactosidase
C. Aortic coarctation visualised by aortic angiography
D. Raised HBA1C
E. Excessive excretion of mucopolysaccharides in urine

B. Deficiency in plasma levels of alpha-galactosidase

The diagnosis is Fabry disease.

This is an X-linked recessive lipid storage disorder in which there a deficiency in the fat enzyme alpha-galactosidase. This results in the accumulation of alpha-galactosyl-lactosyl-ceramide in various tissues, including kidney, liver, blood vessels and nerve ganglion cells. It often presents with severe features in childhood/adolescence but can also less typically present in the 5th or 6th decade with milder features. The clinical features of Fabry disease include:

- acroparaesthesia: tingling, burning pain in the hands and feet triggered by stress such asemotion, extreme temperatures, or exercise
- Angiokeratoma corporis diffusum: lightly verrucous, deep-red to blue-black papules on the trunk (in the bathing trunk distribution)
- Cardiac: mitral valve prolapse or regurgitation usually, but any valvular heart defect can occur
- Strokes: including young strokes/TIAs
- Chronic Kidney Disease: proteinuria usually, can present late in fulminant renal failure

Classically death occurred in the 4th decade but this has improved since the availability of dialysis. It would be important to organise urgent renal investigations in this young man amongst your workup.

Another lipid storage disorders that may come up:

- Gaucher's disease: autosomal recessive disorder characterised by a deficiency of glucocerebrosidase, resulting in glucocerebroside accumulating in the phagocytic cells of the body and hence you get massive (painless) splenomegaly (can be a typical otherwise well, young PACES patient and the condition is found principally in Jews of European stock). You expect the pancytopenia picture on blood tests; typical of hypersplenism. Life span is shortened but not markedly.

Question 86 of 235

A 29 year old man with ulcerative colitis presents to clinic. His colitis has been relatively well controlled since his diagnosis at 18 years old, with two flare-ups requiring steroids in the last 3 years. He has never needed hospital admission. He is currently in full time employment as an engineer.

He has been taking Pentasa (mesalazine) for two years, which is very well tolerated. On examination, his height is 180cm, and weight 58kg (body mass index 18 kg/m²). His abdomen is soft and non-tender. His last colonoscopy 8 months ago showed no active inflammation and no suspicious areas.

He is anxious as his father died from colorectal cancer in his 60's. There is no other family history of gastrointestinal disease.

How regularly should he have a surveillance colonoscopy?

A. Yearly
B. 3 years
C. 2 years
D. 4 years
E. 5 years

B. 3 years

Since this patient has a first degree relative with a history of bowel cancer over 50 years old, he would fall into the 'intermediate category', and thus require a 3 yearly colonoscopy.

Guidelines: BSG Guidelines for the management of inflammatory bowel disease

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A 26-year-old woman presents with a flare-up of her ulcerative colitis. Since admission, she has been treated with intravenous steroids and ciclosporin. You are asked to review her for discharge on day 4 of her admission. On examination she has a temperature of 37.7 °C, pulse is 91/min, She has opened her bowels 9 times today (>10 times a day on admission), has been eating and drinking and says she feels ready to go home. Her abdomen is mildly tender in the left flank, but is otherwise soft with active bowel sounds present. Current blood tests are as follows:

Hb	101 g/l
Platelets	200 * 10 /l9
WBC	9.5 * 10 /l9
ESR	35 mm/h
CRP	98 mg/l

What is the next step in this patient's management?

A. Start infliximab 5mg/kg
B. Commence daily steroid enemas
C. Discharge home with reducing course of oral steroids
D. Request an urgent abdominal radiograph and surgical review
E. Consider oral loperamide for her diarrhoea

D. Request an urgent abdominal radiograph and surgical review

The Truelove and Witts criteria helps to guide physicians in the management of ulcerative collitis:

- Mild: Bowel movements <4/day, temp <37.8 °C, pulse <90/min, ESR<30mm/hour, No anaemia, no or small amounts of blood in stool,
- Moderate: Bowel movements 4-6, the remainder of the parameters are the same for mild.
- Severe; Bowel movements 6 or more plus one or more of the systemic features such as: temp >37.8°C, Pulse > 90/min, ESR >30mm/hour, visible blood in stool and anaemia.

In this scenario, the patient has a severe exacerbation of her disease despite medical treatment. As a result, early involvement of the surgeons is paramount as it is likely that she will need surgical intervention.

> Nice state that:
>
> *'Assess and document on admission, and then daily, the likelihood of needing surgery for people admitted to hospital with acute severe ulcerative colitis.'*
>
> *'Be aware that there may be an increased likelihood of needing surgery for people with any of the following:'*
>
> - *stool frequency more than 8 per day*

- *pyrexia*
- *tachycardia*
- *an abdominal X-ray showing colonic dilatation*
- *low albumin, low haemoglobin, high platelet count or C-reactive protein (CRP) above 45 mg/litre (bear in mind that normal values may be different in pregnant women).*

Question 88 of 235

A 36-year-old man was diagnosed with ulcerative colitis 10 years previously. At his initial presentation a course of intravenous hydrocortisone was required to achieve remission. Subsequently, the patients disease has been fairly well controlled with azathioprine, although occasion courses of oral steroids had been required to treat minor flares. The patient had experienced no extra-gastrointestinal manifestations of his disease and had no other past-medical history. Family history included the patients father being diagnosed with sigmoid adenocarcinoma at the age of 67 years.

The patient was referred for routine screening colonoscopy. This demonstrated extensive colitis extending to the hepatic flexure but no active endoscopic inflammation or post-inflammatory polyps. Mapping biopsies were taken with no evidence of histological inflammation.

Prior to the procedure the patient stated his concern regarding his future risk of colorectal cancer due to his ulcerative colitis and expressed his willingness to undergo surveillance colonoscopy at the recommended time interval.

In what time interval should the patient undergo his next surveillance colonoscopy?

A. 1 year
B. 2 years
C. 3 years
D. 5 years
E. 10 years

C. 3 years

A meta-analysis of population-based studies found that patients with ulcerative colitis have approximately double the incidence of colorectal cancer than individuals without the disease. In the UK, surveillance colonoscopy is recommended for all patients (excluding those with isolated proctitis alone) starting 10 years after diagnosis.

The interval of subsequent surveillance colonoscopy depends on risk stratification of the patient. Factors to be considered include the extent of colitis, the presence of endoscopic or histological inflammation, the presence of pseudo-polyps, a history of primary sclerosing cholangitis and family history of colorectal cancer. Patient preference is also a factor to be included in decisions around surveillance.

In this case, the patient has a family history of colorectal cancer in a first-degree relative diagnosed at an age greater than 50 years placing him in the intermediate risk category. A further colonoscopy in three years is therefore appropriate. Those in lower risk and higher risk groups are recommended to have colonoscopies every 5 years and 1 year respectively.

Question 89 of 235

A 45-year-old man with long-standing Crohns disease is reviewed in gastroenterology clinic. Initial diagnosis was 12 years previously when he presented as an emergency due to a perianal fistula and associated abscess. This had necessitated emergency surgery and treatment with intravenous corticosteroids. Once remission had been obtained through use of corticosteroids, azathioprine had been used as an immunomodulating agent to maintain remission. Subsequently, the patients disease had been fairly well controlled with disease flares on average less than every 2 years. When flares occurred they tended to cause the patient severe bouts of bloody diarrhoea. Large bowel endoscopies performed following disease exacerbation tended to show colonic inflammation with multiple biopsies consistent with the original histological diagnosis of Crohns disease.

Two months previously, the patients routine monitoring bloods had demonstrated derangement of liver function tests prompting further investigations (details given below). Following conclusion of these investigations, the patient had been initiated on cholestyramine and vitamin supplementation.

Alanine aminotransferase	45 U / L
Alkaline phosphatase	356 U / L
Bilirubin	105 micromol / L
Albumin	30 g / L
ANCA (PR3)	Negative
ANCA (MPO)	Positive
Anti-smooth muscle antibody	Positive

Endoscopic retrograde cholangio-pancreatogram: multiple intrahepatic bile duct strictures and beading

Following discussion with the patient about his new diagnosis he raised his concerns about his future risk of bowel cancer and whether he would require regular endoscopic surveillance.

What is the appropriate frequency of surveillance colonoscopy for this patient?

A. Not indicated
B. Every 1 year
C. Every 3 years
D. Every 5 years
E. Every 10 years

B. Every 1 year

The patients investigation results point to a new diagnosis of primary sclerosing cholangitis, known to be associated with Crohns disease and ulcerative colitis.

Patients with Crohns disease have an increased risk of small bowel and colorectal malignancy (standard incident ratios 40.6 and 1.9 respectively). Surveillance usually begins 10 years after the diagnosis of inflammatory bowel disease with risk stratification to determine frequency of surveillance. The concurrence of Crohns disease and primary sclerosing cholangitis confers greatly increased risk of malignancy with recommendations calling for annual surveillance after diagnosis.

Question 90 of 235

A 62-year-old man presented to the emergency department with fever and confusion. His wife reports that he had a cough productive of green sputum over the last few days. He had seen his GP who prescribed oral co-amoxiclav and doxycycline.

He has no other past medical history. In addition to the recently prescribed antibiotics, he has been taking paracetamol for his fever. He has no known allergies.

On arrival, he had a temperature of 38.8ºC, heart rate was 125bpm, blood pressure was 60/40mmHg and he was started on oxygen due to peripheral saturations reading 88% on air. On examination, he was peripherally cool, had bilateral coarse crackles on auscultation of his chest and was responsive to voice. He was immediately given a bolus of fluids and commenced on broad-spectrum antibiotics for sepsis.

A portable chest x-ray shows bilateral patchy opacifications. His blood results are shown below:

Hb	130 g/L	(135-180)
Platelets	451 * 10 /L	(150 - 400)
WBC	15.7 * 10 /L	(4.0 - 11.0)
Na	135 mmol/L	(135 - 145)
K	5.2 mmol/L	(3.5 - 5.0)
Urea	11.2 mmol/L	(2.0 - 7.0)
Creatinine	320 µmol/L	(55 - 120)
CRP	222 mg/L	(< 5)
Bilirubin	45 µmol/L	(3 - 17)
ALP	248 u/L	(30 - 100)
ALT	2015 u/L	(3 - 40)
Albumin	34 g/L	(35 - 50)

His systolic blood pressure is 65mmHg despite several fluid boluses. He is admitted to ITU for inotropes.

What is the most likely cause of his deranged LFTs?

A. Co-amoxiclav induced cholestasis
B. Ischaemic hepatitis
C. Biliary sepsis
D. Alcoholic hepatitis
E. Paracetamol overdose

B. Ischaemic hepatitis

Acute hypoperfusion (e.g. low BP secondary to blood loss) may result in ischaemic hepatitis

This man is suffering from septic shock and end-organ damage secondary to the hypoperfusion. This is evidenced by the deranged LFTs and AKI. Therefore ischaemic hepatitis is the most likely diagnosis.

Co-amoxiclav is known to cause cholestatic liver injury. If this was the cause, the ALP would be proportionally higher compared to the ALT.

There is no question that the patient is septic with the presentation of fever, tachycardia, hypotension and raised inflammatory markers. However, there is no evidence of abdominal pain; and the symptoms and x-ray finding are in keeping with community-acquired pneumonia therefore biliary sepsis is not the most likely cause.

Alcoholic hepatitis would be associated with a greater increase in bilirubin. There is also no evidence provided to support alcohol misuse.

Whilst we know that patient had been taking paracetamol, there are no details indicating overdose. In these situations, it would be good practice to check paracetamol levels.

Question 91 of 235

A 55-year-old male presents with severe epigastric pain. The pain is constant, sharp in character and radiates to his back. He has a past medical history of 5 episodes of pancreatitis secondary to chronic alcoholism. On systematic enquiry he states that he has been suffering from diarrhoea over the past year. The diarrhoea is foul smelling and difficult to flush. He also has excessive thirst and passes large quantities of urine each day.

Blood results are as follows:

Hb	102 g/l	Na +	136 mmol/l
Platelets	288 * 10 /l 9	K +	3.8 mmol/l
WBC	18.2 * 10 /l 9	Urea	12.1 mmol/l
Neuts	14.8 * 10 /l 9	Creatinine	98 µmol/l
Lymphs	2.2 * 10 /l 9	CRP	66 mg/l
Random glucose	13.8 *mmol/l	Amylase	120 U/l (normal < 140)

What is the most likely diagnosis?

A. Acute on chronic pancreatitis
B. Duodenal ulcer
C. Gastric ulcer
D. Cholecystitis
E. Ruptured abdominal aortic aneurysm

A. Acute on chronic pancreatitis

In patients with acute on chronic pancreatitis, the amylase may not be raised if there is significantly poor residual pancreatic function

The presence of new diabetes and steatorrhoea, in the context of multiple episodes of pancreatitis, suggests that the patient has developed chronic pancreatitis. This can be confirmed by measurement of faecal elastase and imaging.

This acute presentation is a classic presentation of pancreatitis. In patients with acute on chronic pancreatitis, the amylase may not be raised if there is significantly poor residual pancreatic function. Indeed this patient likely has poor residual pancreatic function as discussed above.

Question 92 of 235

A 21-year-old female presents for the first time with crampy right iliac fossa pain and diarrhoea. Colonoscopy shows patchy erythema in what appears to be a cobblestone appearance in her caecum and terminal ileum. She refuses to take prednisolone as her mother had taken it previously and had some particularly 'nasty' side-effects.

What is the next best agent to offer?

A. Mercaptopurine
B. Budesonide

C. Azathioprine
D. Methotrexate
E. Infliximab

B. Budesonide

Patient choice and involvement are particularly important in inflammatory bowel disease. Although you as a professional may feel prednisolone is the best option for her, you have to respect her decision to decline.

In patients who present for the first time and a corticosteroid is declined, contraindicated or cannot be tolerated, consider budesonide. Budesonide is not as effective in inducing remission but does have fewer side-effects.

Question 93 of 235

A 36-year-old woman sees her general practitioner with a 6-month history of fatigue. She is unable to do most of her chores and complains of excessive daytime sleepiness. She also reports generalised itchiness that started approximately 4 months ago. There is no history of abdominal pain, fever or weight loss.

Her vital signs are within normal limits. Physical examination shows yellowing of the conjunctivae and skin. There are numerous scratch marks over her trunk. The remainder of her examination is unremarkable.

Her laboratory studies show:

Reference range		
Haemoglobin	125 g/L	Men: 135-180 g/L Women: 115-160 g/L
White blood cells	8.6 * 10 /L 9	4.0-11.0 * 10 /L9
Platelets	280 * 10 /L 9	150-400 * 10 /L9
ESR	29 mm/h	Men: < (age / 2) mm/hr Women: < ((age + 10) / 2) mm/hr
Creatinine	65 umol/L	55-120 umol/L
TSH	3.2 mu/L	0.5-5.5 mu/L
Bilirubin	34 umol/L	3-17 umol/L
ALP	480 u/L	30-100 umol/L
ALT	44 iu/L	3-40 iu/L
AST	65 iu/L	3-30 iu/L
Antimitochondrial antibody	positive	
Antinuclear antibody	positive	

Urinalysis and ultrasound of the abdomen show no abnormalities.

Which of the following is the most appropriate next step in this patient's management?

A. Budesonide
B. Cholestyramine
C. Liver biopsy
D. Liver transplantation
E. Ursodeoxycholic acid

E. Ursodeoxycholic acid

Ursodeoxycholic acid is the first-line medication for primary biliary cholangitis

This patient has fatigue, generalised itching, elevated bilirubin, alkaline phosphatase, and slightly elevated aminotransferases, suggesting a diagnosis of primary biliary cholangitis (PBC). PBC is the slow, progressive autoimmune destruction of the bile ducts, leading to biliary stasis, hyperbilirubinemia, and pruritus. Antimitochondrial antibodies are highly specific for this condition. Antinuclear antibodies are often positive in autoimmune conditions and are not specific to PBC. Ursodeoxycholic acid is the first-line medication for primary biliary cholangitis. It slows down the progression of PBC and provides relief for symptoms like itching.

Budesonide is given to patients with some immunological disorders, such as autoimmune hepatitis. Patients with autoimmune hepatitis will have typical features like weight loss, abdominal pain, anti-smooth muscle antibodies, and hypergammaglobulinaemia. Autoimmune hepatitis is also not usually associated with a positive antimitochondrial antibody.

Cholestyramine is used in patients with hypercholesterolemia. It can also provide symptomatic relief in itching secondary to cholestatic disease like PBC. Although cholestyramine could help relieve itching in this patient, it would not slow disease progression. It is not the first-line management for this patient's condition.

A liver biopsy may be needed for the diagnosis and management of several hepatic and biliary conditions. It is indicated only if PBC is suspected but ambiguous or if there is no response to treatment. This patient has a clear diagnosis of PBC and should start therapy.

A liver transplant is indicated in the cases of PBC only when bilirubin levels rise above 100 µmol/l.

Question 94 of 235

A 58-year-old man with long-standing Crohn's disease presents with a 1-year history of fatigue. In the last month, he has developed weakness and muscle pain. He also reports a number of trips and falls.

On examination, he is thin and pale. Heart rate is 85 beats per minute and blood pressure is 130/80 mmHg. His chest is clear and heart sounds normal, with no added sounds. His abdomen is soft and has a laparotomy scar. Neurological examination reveals bilateral foot drop, with reduced ankle jerks. He has widespread muscle wasting and calf fasciculations. He has reduced pin-prick sensation to mid shin.

Blood tests show:

Hb	144 g/l
Platelets	195 * 10 /l9
WBC	6.3* 10 /l9
Na +	137 mmol/l
K +	4.5 mmol/l
Urea	7 mmol/l
Creatinine	91 µmol/l
Corrected calcium	2.5 mmol/l
HbA1c	45 mmol/mol

What is the diagnosis?

A. Diabetes mellitus
B. Motor neurone disease

C. Beriberi
D. Pellagra
E. Friedreich's ataxia

C. Beriberi

Vitamin B1 deficiency may cause peripheral neuropathyext question

This patient is malnourished due to Crohn's disease. Chronic thiamine deficiency has led to his fatigue and neurological symptoms. Beriberi typically presents with fatigue, nausea and abdominal pain. If left untreated it can progress, leading to peripheral neuropathy, muscle pain and congestive cardiac failure. Diagnosis is usually made after a response to parenteral thiamine and symptoms tend to improve rapidly with treatment.

Diabetes mellitus and pellagra can cause a peripheral neuropathy, however, this man has a normal HbA1c. Pellagra presents with confusion, rash and diarrhoea. The sensory findings rule out motor neurone disease. Friedrich's ataxia is an autosomal recessive disorder that typically presents in much younger patients.

Alcohol dependency, renal dialysis and a diet high in milled rice are also risk factors for Beriberi.

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A 54-year-old man is referred to gastroenterology clinic by his GP for review following an incidental finding of hepatic steatosis on a recent abdominal ultrasound. The patient had previously presented to the emergency department complaining of recurrent abdominal pain and had been diagnosed with biliary colic following the demonstration of gallstones on ultrasound scan. An elective laparoscopic cholecystectomy was planned in the coming months.

At the clinic, the patient stated that he had researched the details of his ultrasound scan report online and then became concerned about the risk of developing serious liver disease secondary to hepatic steatosis. Between occasional attacks of biliary colic, the patient reported feeling well in himself. Aside from a lifelong tendency to overweight, the patient had no other significant medical history and took no regular medications. In particular, there was no personal or family history of liver disease. The patient consumed only small amounts of alcohol on special occasions and denied having any risk factors for blood-borne viruses.

Physical examination indicated a moderately overweight middle-aged man who appeared generally well. No signs of chronic liver disease were evident on abdominal examination and the patient's liver was not palpable. Please see below for the results of basic blood tests arranged by the patient's GP prior to attendance at the clinic.

Haemoglobin	158 g / dL
Mean cell volume	95.0 fl
White cell count	6.5 x 10>3 / microlitre
Platelets	167 x 10>3 / microlitre
Urea	5.4 mmol / L
Creatinine	98 micromol / L
Albumin	48 g / L (reference 35-50)
Alkaline phosphatase	93 U / L (reference 35-100)
ALT	30 U / L (reference 3-36)
Bilirubin	19 micromol / L (reference < 26)
Ferritin	185 microgram / L (30-300)
International normalised ratio	1.1 (reference 0.8-1.2)

What is the appropriate next investigation to assess the patient's risk of developing a serious liver disease as a consequence of hepatic steatosis?

A. MRI liver
B. Liver biopsy
C. Enhanced liver fibrosis blood test
D. Hepatitis virus antibodies
E. Gamma-GT blood test

C. Enhanced liver fibrosis blood test

In patients with non-alcoholic fatty liver disease, enhanced liver fibrosis (ELF) testing is recommended to aid diagnosis of liver fibrosis

The patient has an incidental finding of non-alcoholic fatty liver disease (NAFLD). In a minority of cases, NAFLD progresses to serious liver diseases such as non-alcoholic steatohepatitis, fibrosis or cirrhosis.

NAFLD is known to have a poor prognosis when associated with severe liver fibrosis. Therefore, recent guidelines suggest that individuals with an incidental finding of NAFLD should be offered an enhanced liver fibrosis (ELF) blood test. If the ELF result indicates advanced liver fibrosis (10.51) then the individual should receive specialist monitoring and intervention. If the ELF result is negative (< 10.51) then the individual is likely to have a benign prognosis from their NAFLD and can be monitored in primary care. For these individuals, a repeat ELF blood test is recommended every 3 years.

NICE recommends that individuals with NAFLD are encouraged to undertake lifestyle modifications in order to lose weight and to remain within the recommended limits for alcohol consumption. Individuals with NAFLD who are taking statins should be encouraged to continue to do so.

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A 45-year-old man with alcoholic liver cirrhosis is admitted with a one-week history of increasing abdominal distension and pain. His medications include thiamine 100mg BD, vitamin B co-strong one OD, spironolactone 100mg OD and omeprazole 20mg OD. He has no known drug allergies.

On examination, his temperature is 38.2ºC, pulse rate is 120 beats per minute and blood pressure is 100/60 mmHg. His sclera is icteric and there are multiple bruise marks all over his body. Examination of his abdomen reveals a distended abdomen which is generally tender all over on palpation. There are reduced breath sounds at his lung bases on auscultation.

Investigations reveal:

Hb	90 g/l	Na +	129 mmol/l	Bilirubin	60 µmol/l
Platelets	78 * 10 /l 9	K +	3.6 mmol/l	ALP	110 u/l
WBC	13.5 * 10 /l 9	Urea	1.2 mmol/l	ALT	40 u/l

Neuts	10.5 * 10 /l 9	Creatinine	35 μmol/l	γGT	150 u/l
Lymphs	1.0 * 10 /l 9	Albumin	24 g/l		
Eosin	0.1 * 10 /l9				

An ascitic tap was performed and shows:

Neutrophil count	600 neutrophils/mm3
Fluid protein	<10 g/L
Serum albumin-ascites gradient	>11

He was treated with piperacillin-tazobactam for spontaneous bacterial peritonitis (SBP).

Which antibiotic should be considered for long-term prophylaxis to prevent recurrence of SBP?

A. Ciprofloxacin
B. Doxycycline
C. Amoxicillin
D. Trimethoprim
E. Co-amoxiclav

NOT ANSWERED IN FILE

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A 60-year-old man who is known to have Barrett's oesophagus is reviewed with the results of his surveillance biopsies. These show high-grade dysplasia but no evidence of carcinoma. He is asymptomatic apart from his gastro-oesophageal reflux disease symptoms which are well controlled on high dose proton pump inhibitor therapy.

What treatment is he most likely to be offered?

A. Oesophagectomy
B. Continue high-dose proton pump inhibitor therapy
C. Endoscopic mucosal therapy
D. Increase frequency of surveillance endoscopies to every 12 months
E. Helicobacter pylori eradication therapy, regardless of previous status

C. Endoscopic mucosal therapy

Dysplasia on biopsy in Barrett's oesophagus requires an endoscopic intervention

The correct answer is **Endoscopic mucosal therapy**. In a patient with Barrett's oesophagus and high-grade dysplasia, endoscopic mucosal therapy is the recommended treatment option according to UK guidelines. This is because high-grade dysplasia is considered a precursor to oesophageal adenocarcinoma, and endoscopic mucosal therapy can effectively eradicate the dysplastic tissue, reducing the risk of progression to cancer. There are several types of endoscopic mucosal therapy, including radiofrequency ablation (RFA) and endoscopic resection (ER), which can be used alone or in combination depending on the extent and location of the dysplasia.

The option **Oesophagectomy** is not the first-line treatment for high-grade dysplasia in Barrett's oesophagus. Although it may be considered in selected cases where there is a high risk of progression to carcinoma or when endoscopic therapies have failed or are contraindicated, it carries significant morbidity and mortality risks compared to less invasive endoscopic treatments.

Continue high-dose proton pump inhibitor therapy alone would not be sufficient for managing high-grade dysplasia in Barrett's oesophagus. While PPIs help control symptoms of gastrooesophageal reflux disease and may reduce inflammation in the oesophagus, they do not directly address the presence of high-grade dysplasia or its potential progression to carcinoma.

Increase frequency of surveillance endoscopies to every 12 months would also not be an appropriate management strategy for this patient. While regular surveillance is important for monitoring patients with Barrett's oesophagus, increasing the frequency of endoscopies without addressing the underlying high-grade dysplasia does not provide any therapeutic benefit.

Lastly, ***Helicobacter pylori*** eradication therapy, regardless of previous status, would not be relevant to this patient's management. While *H. pylori* infection is a known risk factor for gastric cancer and peptic ulcer disease, it has not been directly linked to the development or progression of Barrett's oesophagus or oesophageal adenocarcinoma. Therefore, eradicating *H. pylori* would not address

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A 36-year-old man attends the gastroenterology clinic. He has a past medical history of Crohn's disease having presented 3 months earlier with refractory small bowel inflammation. This led to extensive small bowel resection at that time. He had weaned off corticosteroids and remains on maintenance azathioprine. Unfortunately, he tells you he is suffering from ongoing diarrhoea. He has roughly 5 episodes daily of watery diarrhoea. There is no blood or mucus. Blood tests are as follows.

Hb	129 g/L	(135-180)
Platelets	162 * 10 /L 9	(150 - 400)
WBC	4.5 * 10 /L 9	(4.0 - 11.0)
Na +	138 mmol/L	(135 - 145)
K +	3.6 mmol/L	(3.5 - 5.0)
Urea	3.1 mmol/L	(2.0 - 7.0)
Creatinine	66 μmol/L	(55 - 120)
Bilirubin	14 μmol/L	(3 - 17)
ALP	77 u/L	(30 - 100)
ALT	34 u/L	(3 - 40)
Albumin	31 g/L	(35 - 50)
CRP	1.6 mg/L	(< 5)

What is the most appropriate management?

A. Cholestyramine
B. Corticosteroids
C. Infliximab
D. Metronidazole
E. Referral for further surgery

A. Cholestyramine

Ongoing diarrhoea in Crohn's patient post-resection with normal CRP → cholestyramine

The correct answer is **cholestyramine**. This patient has had removal of the main area of inflammation but has ongoing symptoms. There is no evidence of ongoing inflammation biochemically. Clinically, diarrhoea without blood or mucus is also less suggestive of inflammation. In patients who have had the ileum removed, bile acid malabsorption is a major cause of diarrhoea as most bile acids are reabsorbed in the ileum. This condition can be treated with cholestyramine.

Corticosteroids is incorrect. This would be appropriate for a patient with a recurrence of Crohn's colitis. However, this patient has no evidence of inflammation biochemically or clinically so this would not be indicated at this time.

Infliximab is incorrect. This is generally used for Crohn's colitis refractory to first-line agents. As discussed, this patient does not appear to have ongoing inflammation and bile acid malabsorption is a more likely cause of the symptoms.

Metronidazole is incorrect. This antibiotic is used for some intestinal infections such as giardiasis. In the context of Crohn's, it may be used for complications such as abscess or peritonitis, usually in combination with a gram-negative antibiotic. There is no evidence of infection or such complications in the case described.

Referral for further surgery is incorrect. Surgery should be reserved for patients with severe disease refractory to medical management. This patient does not have evidence of inflammation. Even in the presence of ongoing inflammation, further medical management would be indicated before any further surgical intervention.

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A 55-year-old man presents to the Emergency Department with four episodes of severe haematemesis. This is his first experience of such symptoms. He has a history of heavy alcohol consumption but does not smoke. On examination, he shows pallor, scleral icterus, and diaphoresis. His blood pressure is 80/50 mmHg, and his pulse is 121 bpm. The abdomen is distended with visible engorged superficial veins radiating from the umbilicus, shifting dullness on percussion, and a palpable enlarged spleen. Bilateral lower limb pitting oedema is also noted.

Hb	65 g/L	Male: (135-180)
Platelets	85 * 10 /L 9	(150 - 400)
Bilirubin	75 µmol/L	(3 - 17)
ALT	86 u/L	(3 - 40)
Albumin	25 g/L	(35 - 50)
Urea	10 mmol/L	(2.0 - 7.0)
Creatinine	115 µmol/L	(55 - 120)
INR	2.6	

After initiating intravenous fluid resuscitation and blood transfusion:

What is the most appropriate next step in management for this patient?

A. Ciprofloxacin
B. Octreotide
C. Rifaximin
D. Terlipressin
E. Upper gastrointestinal endoscopy

D. Terlipressin

Terlipressin is used in the management of variceal haemorrhage

Terlipressin is the correct answer. This scenario indicates severe haematemesis, characterised by anaemia, hypotension, and tachycardia. The most likely underlying cause is portal hypertension secondary to decompensated liver cirrhosis. Indicators of portal hypertension include caputmedusae (distended engorged superficial veins radiating from the umbilicus), ascites (noted by shifting dullness on percussion), and oedema of the lower limbs. Additionally, the patient's low albumin level, elevated INR, high ALT, and jaundice are consistent with decompensated liver cirrhosis, possibly due to alcohol abuse. Terlipressin is currently the only licensed vasoactive agent for this condition and is endorsed by NICE guidelines. It acts as a synthetic vasopressin analogue that induces splanchnic vasoconstriction, aiding initial haemostasis and reducing the risk of rebleeding in patients with varices.

Ciprofloxacin plays a role in the initial management of variceal bleeding. During such an event, there is increased activity in the portosystemic shunt, which facilitates the direct translocation of intestinal bacteria into the bloodstream while circumventing hepatic clearance; this can lead to bacterial seeding with potentially negative consequences for these patients. Thus, prophylactic administration of IV ciprofloxacin is advised. Nevertheless, it should not take precedence over vasoactive agents like terlipressin, which is essential for achieving haemostasis.

Octreotide, a somatostatin analogue that results in splanchnic vasoconstriction, can benefit haemostasis during variceal bleeding episodes. However, terlipressin is more effective than octreotide and remains the sole licensed vasoactive agent for this indication; therefore, opting for octreotide would be less appropriate.

Upper gastrointestinal endoscopy is the definitive management of variceal bleeding. However, terlipressin should be given as a part of hemostasis before endoscopy to decrease portal pressure rapidly via splanchnic vasoconstriction. This helps control bleeding and increases the likelihood of successful hemostasis during endoscopy. It starts within minutes of administration. Endoscopy is ideally performed within 24 hours of admission except if there is haemodynamic instability when endoscopy is offered E. Omeprazole-induced Clostridium difficile infection immediately after resuscitation (as in this case), as per NICE guidelines.

Rifaximin, a non-absorbable antibiotic favoured for its safety profile in treating hepatic encephalopathy, might be anticipated as part of treatment considering complications associated with variceal bleeding. Despite this expectation, rifaximin does not address acute variceal bleeding nor fulfils the immediate requirement for a vasoactive agent such as terlipressin to secure haemostasis.

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A 60-year-old man presented to the Emergency Department with profuse, foul smelling diarrhoea, abdominal pain and fever.

His past medical history included hypertension, gout and osteoarthritis. His bowel habits are usually regular and there has been no recent change. Two weeks ago he underwent an endoscopy for dyspepsia and was diagnosed with gastritis. He was currently taking amlodipine 5mg, omeprazole 20mg, simvastatin 20mg, salbutamol inhaler one puff as required. He had no known drug allergies. He recently returned from a business trip to Paris.

On examination he was unwell with a heart rate of 110 beats/min and regular, a blood pressure of 100/60 mmHg, oxygen saturations of 96% on air and a temperature of 38ºC. He was peripherally shut down with a capillary refill time of 3 seconds. Abdominal examination revealed and distended and diffusely tender abdomen with guarding.

Initial bloods showed:

Na+	140 mmol/L
K+	5.0 mmol/L
Urea	10 mmol/L
Creatinine	130 mmol/L
Hb	13.0 g/dL
WBC	20.0 x10^9/L
Neutrophils	89%
LFTs	Normal

Abdominal X-ray showed a loss of bowel wall architecture and thumb-printing consistent. Erect chest x-ray showed clear lung fields with no air under the diaphragm.

What is the most likely cause of his symptoms?

A. Salmonellosis
B. Inflammatory bowel disease
C. Ischaemic colitis
D. Diverticulitis
E. Omeprazole-induced Clostridium difficile infection

E. Omeprazole-induced Clostridium difficile infection

PPIs are a risk factor for *C. difficile* infection

Proton pump inhibitors have been associated with *Clostridium difficile* infection and subsequent development of pseudomembranous colitis. The clues to this diagnosis included the recent omeprazole use, foul smelling diarrhoea, raised white cell count and abdominal film findings.

The patient is clearly unwell and will require adequate resuscitation before further investigation to confirm the diagnosis.

Diverticulitis can present with abdominal pain and diarrhoea but the clue here is the profuse foul smelling diarrhoea characteristic of *Clostridium difficile* infection.

Salmonellosis is unlikely as there is nothing from the history to suggest exposure to the bacterium. Ischaemic colitis typically presents in the elderly with abdominal pain and often bloody diarrhoea.

Inflammatory bowel disease can present similarly but one would expect to have seen a change in bowel habit prior to this acute episode.

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A 52-year-old woman presents to her general practitioner with fatigue. She has no significant past medical history and does not take any regular medications. She has a family history of haemochromatosis. She does not drink alcohol.

Cardiovascular, respiratory and abdominal examinations are unremarkable. Her body mass index is 36 kg/m².

Blood tests:

Hb	137 g/L	Male: (135-180)Female: (115 - 160)
Platelets	189 * 10 /L 9	(150 - 400)
WBC	5.2 * 10 /L 9	(4.0 - 11.0)
Na +	137 mmol/L	(135 - 145)
K +	4.2 mmol/L	(3.5 - 5.0)
Urea	5.2 mmol/L	(2.0 - 7.0)
Creatinine	88 µmol/L	(55 - 120)
CRP	4 mg/L	(< 5)
Bilirubin	12 µmol/L	(3 - 17)
ALP	99 u/L	(30 - 100)
ALT	74 u/L	(3 - 40)
Î³GT	84 u/L	(8 - 60)
Albumin	36 g/L	(35 - 50)
Ferritin	244 ng/mL	(18-160)
Transferring saturation	38%	(20-50%)

Further tests:

Antinuclear antibody	positive (1:80)
Anti-mitochondrial antibody	negative
Anti-smooth muscle antibody	negative
Anti-LKM1 antibody	negative
Anti soluble liver antigen antibody	negative

What is the most likely diagnosis?

A. Autoimmune hepatitis
B. Haemochromatosis
C. Non-alcoholic fatty liver disease (NAFLD)
D. Primary biliary cirrhosis
E. Primary sclerosing cholangitis

C. Non-alcoholic fatty liver disease (NAFLD)

Obesity with abnormal LFTs - ? non-alcoholic fatty liver disease

Non-alcoholic fatty liver disease (NAFLD) is the correct answer. Fatigue is a common symptom and may have a variety of including obesity. The patient presents with fatigue and mildly deranged liver function tests (ALT in particular). A mildly raised ferritin is common in NAFLD in addition. By far and away the most common cause of this presentation in someone with a BMI of 36 kg/m² is non-alcoholic fatty liver disease.

Haemochromatosis is incorrect. While she has a family history of this disorder, a normal transferrin saturation essentially excludes the diagnosis.

Primary biliary cirrhosis is incorrect. This can cause fatigue and deranged LFTs in a middle-aged woman. However, the LFTs will demonstrate an obstructive pattern and we would expect antimitochondrial antibodies to be positive in most cases.

Autoimmune hepatitis is incorrect. The ANA is only weakly positive (1:80) which is non-specific and commonly encountered in patients with NAFLD. The other antibodies that are more specific for autoimmune hepatitis (smooth muscle, LKM1 and soluble liver antigen) are all negative, making this diagnosis less likely.

Primary sclerosing cholangitis is incorrect. We would expect an obstructive pattern of LFT derangement in this case.

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A 74-year-old patient presents to the emergency department with three days of profuse diarrhoea. He has undergone treatment for cellulitis recently with clindamycin. He has no past medical history and is not on any other medications.

Observations:

- Heart rate 88 beats per minute
- Blood pressure 120/80 mmHg
- Respiratory rate 18/minute
- Oxygen saturations 97% on room air
- Temperature 37.8

Examination reveals mild abdominal tenderness with no peritonism.

Hb	138 g/L	Male: (135-180)Female: (115 - 160)
Platelets	189 * 10 /L 9	(150 - 400)
WBC	6.5 * 10 /L 9	(4.0 - 11.0)
Na +	138 mmol/L	(135 - 145)
K +	4.2 mmol/L	(3.5 - 5.0)
Urea	5.2 mmol/L	(2.0 - 7.0)
Creatinine	88 µmol/L	(55 - 120)
CRP	52 mg/L	(< 5)

An abdominal x-ray is unremarkable.

A stool sample is positive for *Clostridium difficile* toxin.

He has full treatment courses of oral vancomycin followed by oral fidaxomicin but has ongoing diarrhoea with type 7 stools eight days after the 2nd course of antibiotic treatment and remains *Clostridium difficile* toxin positive.

What is the appropriate management at this point?

A. Colectomy
B. Faecal transplant
C. IV vancomycin
D. Oral vancomycin and IV metronidazole
E. Repeat oral fidaxomicin

D. Oral vancomycin and IV metronidazole

> If a first episode of *C. difficile* doesn't respond to either vancomycin or fidaxomicin then oral vancomycin +/- IV metronidazole should be tried

Oral vancomycin and IV metronidazole is the correct option. The patient has experienced treatment failure of *Clostridium difficile* infection as demonstrated by ongoing diarrhoea and persisting detectable toxin in the stool. The 3rd line option to treat the infection in this circumstance is oral vancomycin +/- IV metronidazole.

Colectomy is incorrect. This option would only be used in the setting of life-threatening disease. There is no documentation to suggest that is the case here.

Faecal transplant is incorrect. While this is an option for treating recurrent *Clostridium difficile* infection, it is only used after more conservative measures have been exhausted.

IV vancomycin is incorrect. This method of administering vancomycin is ineffective in *Clostridium difficile* infection and should not be used.

Repeat oral fidaxomicin is incorrect. Repeating a course of oral vancomycin rather than fidaxomicin is a recognized option.

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A 53-year-old man presents to the emergency department with right upper quadrant pain and fever. He has a past medical history of type 2 diabetes and takes regular metformin.

Observations:

- Heart rate 101 beats per minute
- Blood pressure 101/54 mmHg
- Respiratory rate 20/minute
- Oxygen saturations 95% on room air
- Temperature 38.4C

On examination, there is tenderness without guarding in the right upper quadrant.

A CT abdomen with contrast is arranged, which demonstrates a central low attenuation lesion surrounded by a high attenuation inner rim and a low attenuation outer ring.

Given the likely diagnosis, what is the correct management?

A. Percutaneous drainage
B. Percutaneous drainage + amoxicillin + ciprofloxacin
C. Percutaneous drainage + amoxicillin + ciprofloxacin + metronidazole
D. Percutaneous drainage + benzylpenicillin + metronidazole
E. Percutaneous drainage + flucloxacillin + ciprofloxacin

C. Percutaneous drainage + amoxicillin + ciprofloxacin + metronidazole

> Liver abscesses are generally managed with a combination of antibiotics & drainage

Percutaneous drainage + amoxicillin + ciprofloxacin + metronidazole is the correct answer. The patient presents with fever and right upper quadrant pain and undergoes a CT scan, which demonstrates the 'double target sign', which consists of a central low attenuation lesion surrounded by a high attenuation inner rim and a low attenuation outer ring. This sign suggests a hepatic abscess. The management of a liver abscess is with a combination of drainage and antibiotics. Type 2 diabetes is a risk factor for liver abscess development. Most infections are polymicrobial, consisting of aerobic and anaerobic gram-negative and positive organisms and therefore the antibiotic choice should be broad enough to cover these types of organisms.

Percutaneous drainage is incorrect. Drainage alone is not optimal management, the patient should also be administered antibiotics.

Percutaneous drainage + amoxicillin + ciprofloxacin is incorrect. The antibiotic choice, in this case, would provide reasonable cover for gram-positive and negative aerobic organisms but does not provide adequate coverage against anaerobes.

Percutaneous drainage + benzylpenicillin + metronidazole is incorrect. This antibiotic choice would provide coverage against gram-positive organisms and anaerobes. However, it would not be sufficient to cover all gram-negative organisms.

Percutaneous drainage + flucloxacillin + ciprofloxacin is incorrect. Flucloxacillin is typically used for skin and soft tissue infections. The absence of metronidazole means anaerobic coverage is insufficient.

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A 40-year-old lady presents with irritation of her eyes. This has been ongoing for the past few weeks and she links this in time to the recent sunny weather and high pollen levels. She has primary biliary cirrhosis and is being managed conservatively with ursodeoxycholic acid. There is no exudate from the eyes or photophobia. She wears contact lens for myopia and reports changing the washing solution regularly.

On examination, both eyes are red with conjunctival injection. There is no watering or exudate. She has 6/6 visual acuity bilaterally and slit lamp exam is normal. Fluorescein staining shows no abrasion. She has a fine tremor and there are no lumps in the neck.

Bilirubin	34 µmol/l
ALP	89 u/l
ALT	76 u/l
γGT	50 u/l
Albumin	35 g/l
Rheumatoid factor	positive
TSH	3.0mU/l

What is the likely diagnosis?

A. Sjogren's syndrome
B. Allergic conjunctivitis
C. Grave's disease
D. Anterior uveitis
E. Amoebic keratitis

A. Sjogren's syndrome

Sjogren's syndrome is common in patients with PBC

This lady has red irritated eyes bilaterally. Anterior uveitis usually has a drop in acuity, circumlimbal redness and cells are seen on slit lamp. Amoebic keratitis is usually linked to one eye and has reduced visual acuity from corneal damage. Grave's disease has gritty eyes but there are signs of hyperthyroidism or proptosis. Allergic conjunctivitis is possible but this is a new onset of these symptoms and importantly there is no watery exudate which you would expect. Sjogren's syndrome which involves dry eyes is very common in patients with PBC and the lack of watery eyes suggests they are dry and this is the likely diagnosis.

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A 35-year-old man is admitted to the hospital with dehydration secondary to diarrhoea. He reports opening his bowels 4 times a day with loose brown stool. He denies any previous gastrointestinal infections.

On examination, his abdomen is mildly tender with no guarding or rebound tenderness. His observations are within the normal range.

His blood tests show the following:

Hb	140 g/L	(135-180)
Platelets	450 * 10 /L 9	(150 - 400)
WBC	16.3 * 10 /L 9	(4.0 - 11.0)
Na^+	139 mmol/L	(135 - 145)
K^+	3.7 mmol/L	(3.5 - 5.0)
Bicarbonate	22 mmol/L	(22 - 29)
Urea	6.5 mmol/L	(2.0 - 7.0)
Creatinine	79 µmol/L	(55 - 120)

His stool test is positive for* Clostridium difficile *toxin. What should be the first-line treatment for this patient?

A. Fluid rehydration and monitoring only
B. Intravenous fidaxomycin
C. Intravenous metronidazole
D. Oral metronidazole
E. Oral vancomycin

E. Oral vancomycin

Oral vancomycin is the first line antibiotic for use in patients with *C. difficile* infection

The patient has moderate *Clostridium difficile* infection, making the correct answer a 10 day course of **oral vancomycin** which is the first-line therapy for non-life-threatening *Clostridium difficile* infection.

Fluid rehydration and monitoring only would be inappropriate and risk the infection worsening.

Oral fidaxomycin, rather than **intravenous fidaxomycin** is second-line treatment for non-lifethreatening *Clostridium difficile* infection. It is also the recommended treatment for recurrent *Clostridium difficile*.

Intravenous metronidazole is used in conjunction with oral vancomycin to treat life-threatening *Clostridium difficile* infection, or as third line therapy for non-life-threatening *Clostridium difficile* infection. This patient does not have features of life-threatening *Clostridium difficile* infection which include hypotension, ileus, toxic megacolon or CT evidence of severe disease.

Oral metronidazole is no longer recommended for treating *Clostridium difficile* infection in the latest 2021 NICE *Clostridium difficile* antimicrobial prescribing guidance. This is due to lower

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A 62-year-old patient is referred to the acute medical service with jaundice. This has come on suddenly in the last few days. The patient denies any excessive alcohol consumption drinking only the occasional glass of wine every other week. There only previous history of note is recurrent urinary tract infections. This has been managed in the community with once daily prophylactic antibiotics.

Liver function tests reveal:

Total bilirubin	74 µmol/l
Unconjugated bilirubin	35 µmol/l
Conjugated bilirubin	39 µmol/l
ALP	110 u/l
AST	219 u/l

What is the most likely causative agent for these results?

A. Cephalexin
B. Co-amoxiclav
C. Amoxicillin
D. Trimethoprim
E. Nitrofurantoin

E. Nitrofurantoin

The results above represent a hepatocellular cause of jaundice. This is shown from the elevated unconjugated bilirubin levels and differentiates it from a post-hepatic (obstructive) jaundice. All of the agents above can cause a cholestatic (obstructive) picture but the most likely cause of a hepatocellular jaundice is nitrofurantoin. This occurs with chronic use of the medication. It is associated with hepatic necrosis and should be stopped immediately when there is any evidence of hepatic necrosis. The side effects of nitrofurantoin can be fatal and as such attention must be paid to its appearance on a drug history. Toxic effects can manifest as hepatic disease as shown here but also as both an acute and chronic respiratory condition, agranulocytosis and the acute hypersensitivity reaction of Stevens-Johnson Syndrome.

There is no clear guidance in the use of antibiotic prophylaxis in adult patients. However, recent Cochrane review questioned the use in children. The other common advice given to patients is to drink cranberry juice to prevent further infections, but this again has not been backed up by the study data. Therefore there is currently no consensus, but recurrent UTI should warrant further investigations by either a gynaecologist or urologist in adults and most certainly a

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A 44-year-old male is referred to gastroenterology clinic by his GP. He had attended his GP surgery due to persistent episodes of loose stool. On further questioning, his only other symptom was mild fatigue. He is a non-smoker and his alcohol intake is within the recommended weekly allowance.

On examination his GP noted mild jaundice but no other stigmata of chronic liver disease. Palpation of his abdomen revealed mild hepatomegaly. All other systems were normal. Prior to being referred to clinic, the patient underwent a non-invasive liver screen (NILS). The results are as follows:

Hb	11.0 g/dl
Platelets	175 * 10 /l9
WBC	9.5 * 10 /l9
Na +	139 mmol/l
K +	4.9 mmol/l
Urea	6.7 mmol/l
Creatinine	87 µmol/l
Bilirubin	52 µmol/l
ALP	389 u/l
ALT	68 u/l
Albumin	40 g/l
Hep B sAg	Negative
Anti-Hep C Ab	Negative
Anti-nuclear Ab	Positive
Anti-mitochondrial Ab	Negative
Auto-smooth muscle Ab	Negative
Serum caeruloplasmin	28mg/dL (20-40mg/dL)
Serum ferritin	490mg/L (15-250mg/L)
Total iron binding capacity	62mmol/L (40-80mmol/L)
Abdominal ultrasound	Mild hepatomegaly. Otherwise normal

What is the most likely diagnosis?

A. Primary biliary cirrhosis
B. Autoimmune hepatitis
C. Wilson's disease
D. Primary sclerosing cholangitis
E. Haemochromatosis

D. Primary sclerosing cholangitis

The mild fatigue, jaundice, abnormal LFTs and hepatomegaly are fairly non-specific and on their own are not enough to answer this question. The positive anti nuclear antibody result points towards an autoimmune pathology but the key is the history of persistent episodes of loose stool. When viewed in combination with the other abnormalities, the suggestion is that this patient is suffering with ulcerative colitis and primary sclerosing cholangitis. These two conditions which are known to be associated, and in combination tie all of the findings together.

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A 40-year-old man presented for the first time in a GP surgery. He had recurrent abdominal pain which improved with defecation, and mushy stools up to three times a day for the last 4 months. He denied weight loss, rectal bleeding, or a family history of colorectal cancer. Stool microscopy, culture and sensitivity were negative for ova, cysts and parasites.

Which of the following is the next most appropriate investigation to be done?

A. Abdominal ultrasound
B. Colonoscopy
C. Flexible sigmoidoscopy
D. Abdominal X-ray
E. Tissue transglutaminase

NOT ANSWERE

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A 29-year-old female is admitted to the gastroenterology ward with a flare up of her Crohn's disease. She has a one week history of passing loose stools, up to 8 times per day, alongside severe generalised abdominal pain and fevers. Her observations are as follows: heart rate 100/min blood pressure 114/78mmHg, respiratory rate 16/min, SpO 98%, Temperature 37.9ºC.

Blood results reveal the following:

CRP	98 mg/L

She is currently being treated with IV hydrocortisone, which she has taken for 5 days with no improvement in symptoms.

What is the next step in management to consider for this patient?

A. Infliximab
B. Oral prednisolone
C. Ileocaecal resection
D. Enteral nutrition
E. Rectal mesalazine

A. Infliximab

Biologic therapy is considered in treatment of an acute flare of Crohn's disease when symptoms don't improve after 5 days of IV hydrocortisone

This lady has severe active Crohn's disease, and she is on day 5 of treatment with IV hydrocortisone with no improvement of symptoms. The next step in management should be considered, which would be anti-TNFα agents e.g. infliximab or adalimumab.

Oral prednisolone is used in mild flare ups of Crohn's disease.

An ileocaecal resection may be considered in an unremitting attack, but this would require discussion with the surgical team and correct identification of the areas of the GI tract that are affected by Crohn's disease.

Enteral nutrition (a liquid diet) is a treatment option considered in children or young people where there are concerns about growth or side effects of treatment.

Rectal mesalazine is more often used in the management of ulcerative colitis, rather than Crohn's disease.

Question 110 of 235

A 58 year-old woman presents to hospital after 2 episodes of vomiting up blood. It began 2 hours prior to presenting at hospital and the patient was at work as a saleswoman at the time. She has a past history of a duodenal ulcer and also suffers from irritable bowel syndrome, hypertension and hypercholesterolaemia. Her regular medication includes simvastatin and ramipril. She is admitted under the gastroenterology team and undergoes fluid resuscitation and successful sclerotherapy. The next morning, the patient vomits up a moderate amount of blood.

What is the most appropriate management?

A. Intravenous omeprazole
B. Conservative management
C. Intravenous somatostatin
D. Repeat sclerotherapy endoscopically
E. Refer to a general surgeon for open surgery

D. Repeat sclerotherapy endoscopically

Re-bleeding can occur in approximately 15% of patients who had previously been treated and this rate of re-bleeding increases in parallel with the severity of the original bleed. Operative intervention is much more effective than conservative management in the case of re-bleeding and although omeprazole and somatostatin can be used in conjunction with operative treatment, the risks associated with re-bleeding mean operative intervention is an absolute indication. There are similar mortality rates for endoscopic treatment and open surgery in patients who have had rebleeding following an earlier successful endoscopic treatment and so endoscopic treatment is preferable in this case.

Question 111 of 235

A 26-year-old woman presents to the gastroenterology clinic with a flare of ulcerative colitis. She reports that for the last week he has been experiencing bloody streaked diarrhoea, with bowels opening up to 6 times per day. She reports no fevers. She has been taking her mesalazine enemas most nights but feels that these have not helped her. She reports full compliance with her oral contraceptives and her last menstrual period was 2 weeks ago. She has no other medical conditions, smokes occasionally and drinks alcohol once or twice weekly.

On clinical assessment, she is afebrile, basic observations are within normal limits aside from mild hypotension, felt to be the patient's baseline. Her abdomen is soft with mild left iliac fossa tenderness.

Blood tests show:

Hb	120 g/L	Male: (135-180)Female: (115 - 160)
Platelets	250 * 10 /L 9	(150 - 400)
WBC	11.7 * 10 /L 9	(4.0 - 11.0)
ESR	25 mm/hr	(<10)
bHCG	negative	

An abdominal X-ray is unremarkable.

A flexible sigmoidoscopy is arranged for the following day and shows mild inflammation. Of note, a previous colonoscopy demonstrated colitis up to the ileocaecal valve.

Given the clinical history, which of the following interventions should be considered for this patient?

A. Prednisolone
B. Hydrocortisone
C. Oral mesalazine
D. Azathioprine
E. Infliximab

C. Oral mesalazine

In a mild-moderate flare of ulcerative colitis extending past the left-sided colon, oral aminosalicylates should be added to rectal aminosalicylates, as enemas only reach so far

This patient has a mild flare of ulcerative colitis and has evidence of disease past the left-sided colon, thus oral mesalazine should be added.

If this were a moderate flare, oral prednisolone may be indicated and if moderate-severe, then hydrocortisone may be preferable.

Azathioprine enables maintenance of remission but is not appropriate in the treatment of an acute flare.

Infliximab would only be indicated in a moderate to severe flare which had failed steroid therapy.

Question 112 of 235

A 65-year-old woman was referred by her General Practitioner for routine upper gastrointestinal endoscopy. The patient had been suffering from persistent epigastric discomfort induced by eating. Testing for Helicobactor pylori had been negative. The patient had not experienced any dysphagia, vomiting or weight loss. Past medical history included osteoarthritis of the left knee for which she takes ibuprofen as required. The patient manages her own cleaning business and drinks 20-25 units of alcohol per week. Clinical examination was unremarkable.

At endoscopy a generalised mild gastritis was found. In addition, biopsies were taken from a 1.5 cm diameter non-ulcerated lesion noted at the fundus. The results of histology and further investigations subsequently requested are listed below.

Histology: sample of gastric mucosa exhibiting moderately differentiated adenocarcinoma without involvement of sub-serosal connective tissue

CT abdomen: no gastric mass or ulceration identified; no evidence of abdominal lymph node enlargement; liver, gallbladder, spleen and kidneys unremarkable.

PET: no evidence of distant metastasis or nodal involvement Staging laparoscopy: no evidence of peritoneal or metastatic disease

What is the appropriate treatment for this patient's gastric carcinoma?

A. Neoadjuvant chemotherapy
B. Endoscopic mucosal resection
C. Total gastrectomy
D. Partial gastrectomy
E. Radical radiotherapy

B. Endoscopic mucosal resection. Endoscopic

gastrectomy feasible for some distal tumours. Radiotherapy is not used as a first-line treatment of gastric carcinoma.

Question 113 of 235

A 65-year-old man attends the clinic with progressive shortness of breath and recurrent chest infections. He has also been experiencing episodes of flushing associated with dizziness. On examination, you note proximal myopathy and abdominal striae. Observations are as follows: heart rate 95 beats per minute, blood pressure 165/95 mmHg, respiratory rate 18 breaths per minute, SpO2 95% (on air), and temperature 37.5ºC.

Blood results are as follows:

Hb	145 g/L	Male: (135-180)Female: (115 - 160)
Platelets	660 * 10 /L $_9$	(150 - 400)
WBC	14.2 * 10 /L $_9$	(4.0 - 11.0)
Na $^+$	148 mmol/L	(135 - 145)
K $^+$	2.8 mmol/L	(3.5 - 5.0)
Urea	6.2 mmol/L	(2.0 - 7.0)
Creatinine	96 µmol/L	(55 - 120)
CRP	5 mg/L	(< 5)
Bicarbonate	34 mmol/L	(23 - 29)
Glucose	14.7 mmol/L	(4 - 7)

A CT scan is performed:

CT chest	Right-sided perihilar 3cm x 2cm well-defined ovoid mass

What is the most likely diagnosis?

A. Adenocarcinoma of the lung
B. Carcinoid of the lung
C. Large cell carcinoma of the lung
D. Small cell lung cancer
E. Squamous cell carcinoma of the lung

B. Carcinoid of the lung tion

Lung carcinoid may results in Cushing's syndrome secondary to ACTH release

The patient has clear evidence of lung cancer. The clinical features of flushing and dizziness raise suspicion of carcinoid syndrome. Furthermore, the radiological appearances are also consistent with a carcinoid tumour. The presence of proximal myopathy, abdominal striae, hypertension, hyperglycaemia, hypernatraemia, hypokalaemia and alkalosis are very suspicious of Cushing's syndrome.

Neuroendocrine tumours such as small cell lung cancer and bronchial carcinoid tumours are common causes of paraneoplastic Cushing syndrome secondary to ectopic ACTH secretion.

Carcinoid of the lung is correct. Carcinoid of the lung is the most likely diagnosis given the radiological appearances and likely ectopic ACTH production.

Adenocarcinoma of the lung is incorrect. This is not a neuroendocrine tumour and is thus not usually associated with ectopic ACTH production making this a less likely diagnosis.

Large cell carcinoma of the lung is incorrect. This is not a neuroendocrine tumour and is thus not usually associated with ectopic ACTH production making this a less likely diagnosis.

Small cell lung cancer is incorrect. Although small cell lung cancer is of neuroendocrine origin and frequently implicated with ectopic ACTH secretion, the radiological appearances, in this case, are more consistent with a carcinoid tumour. The CT appearances of small cell lung cancer frequently show mediastinal involvement which may appear similar to lymphoma, with numerous enlarged nodes. Direct infiltration of adjacent structures is also common. Only rarely do they present as a solitary pulmonary nodule.

Squamous cell carcinoma of the lung is incorrect. This is not a neuroendocrine tumour and is thus not usually associated with ectopic ACTH production making this a less likely diagnosis.

Question 114 of 235

A 45-year-old Caucasian man presents with an acutely painful left knee. He is unable to mobilise due to the pain and swelling. Prior to this episode, the patient states that he is generally active, often enjoying a round of golf every Sunday. Last week he had a minor fall onto both knees whilst clearing out the garage. He puts his current symptoms down to general aches and pain of 'old age'.

The patient has also noticed that he is waking up more often in the night to pass urine. His wife has noticed that he is looking more 'tanned' despite them not going abroad on holiday this year. On further questioning, he admits to drinking 5-6 pints of ale per week. He is a non-smoker.

On examination on the Medical Assessment Unit you note significant left sided knee swelling. The joint is hot and tender to touch. There is no evidence of any surrounding skin changes.

His random glucose is measured at 18mmol/L. Further blood tests are shown below:

Hb	138 g/l
Platelets	120 * 10 /l9
WBC	7.0 * 10 /l9
Na +	138 mmol/l
K +	4.0 mmol/l
Urea	6.2 mmol/l
Creatinine	88 µmol/l
CRP	72 mg/L

What is the most likely cause of his acutely swollen knee?

A. Pseudogout
B. Ruptured Baker's cyst
C. Septic arthritis
D. Gout
E. Haemarthrosis

A Pseudogout

This gentleman presents with arthralgia, polyuria and skin colour changes. These symptoms in a gentleman of this age and ethnicity suggest hereditary haemochromatosis. Polyuria and significantly elevated random blood glucose suggest diabetes. Pseudogout secondary to calcium pyrophosphate crystal deposition in large joints is a well-known complication of haemochromatosis.

Radiological manifestations of pseudogout show a classical chondrocalcinosis picture. See link below for images of this:

Question 115 of 235

A 32-year-old female presents with a significant upper gastrointestinal bleed. She has been vomiting frank blood over the past 36 hours. She has a history of chronic back pain and admits to taking naproxen over the recommended daily dosage, especially over the past two weeks due to a flare up of her pain.

On examination, she appears pale and her abdomen is soft with localised tenderness to the epigastric region. Blood pressure 86/50 mmHg and pulse rate is 110 bpm.

Hb	58 g/l
Platelets	60 *10 /l9
WBC	5 *10 /l9

With regards to blood transfusion in this patient's case what pre-endoscopy target haemoglobin range would give the best overall outcome?

A. Target haemoglobin 50-70 g/l
B. Target haemoglobin 70-80 g/l
C. Target haemoglobin 100-110 g/l
D. Target haemoglobin 120-130 g/l
E. No evidence of any difference of pre-endoscopic haemoglobin and outcome

B. Target haemoglobin 70-80 g/l

Recent evidence has shown that conservative blood transfusion in the setting of acute upper gastrointestinal bleeding produces better outcomes compared to liberal blood transfusion.

An important observation was that there was an improvement in survival rates with the restrictive transfusion strategy. There was a reduction in the risk of further bleeding, the need for rescue therapy, and the rate of complications.

In Villanueva et al patients who were transfused when the haemoglobin was below 70g/dL had better outcomes compared to those who received blood transfusion below 10g/dL.

Question 116 of 235

A 45-year-old female presents to the acute medical take with jaundice. She reports being tee-total and does not report any abdominal pain or recent travel. On examination, the patient is apyrexial. There is palpable tender hepatomegaly. The sclerae are icteric with no peripheral stigmata of chronic liver disease. An initial liver screen shows:

Bilirubin	141 µmol/l
ALP	208 u/l
ALT	635 u/l
Albumin	36 g/l
INR	1.2

On further questioning, the patient reports taking regular thyroxine post-thyroidectomy but takes no other prescription or over-the-counter medications and is generally fit and well. An ultrasound and viral hepatitis screen comes back as follows:

HBsAg	Negative
HBc Ab	Negative
HepC Ab	Negative
Ultrasound	Smooth hepatomegaly

Given the most likely diagnosis, which of the following is correct?

A. A full blood count will characteristically show lymphocytosis
B. Serum AMA titres will often be raised
C. Serum IgG levels will often be low
D. Treated mortality is 80% at 10 years
E. Liver biopsy will show an interface hepatitis

E. Liver biopsy will show an interface hepatitis

The clue in the history comes from the previous thyroidectomy which suggests the possibility of autoimmune disease in this patient. Autoimmune hepatitis (AIH) is a rare cause of acute hepatitis. However, in the history many of the more common causes (alcohol, medications, viral infection) have been excluded.

The diagnosis is usually confirmed by a liver biopsy which shows an interface hepatitis (inflammation with lymphocytic infiltrate at the junction between hepatocytes and portal tracts). Note that chronic viral hepatitis may also show a interface hepatitis however the viral screen was negative in this patient.

Autoimmune hepatitis is associated with hypergammaglobulinaemia i.e. serum IgG levels will typically be high. Anti-mitochondrial antibodies (AMA) are present in primary biliary cirrhosis (PBC) so point away from a diagnosis of AIH. Treated mortality of AIH is 10% at 10 years. EBV hepatitis usually shows a lymphocytosis, however there are no features of infectious

Question 117 of 235

A 75-year-old man is reviewed in gastroenterology clinic as part of on-going follow-up for Barrett's oesophagus. The patient had been referred by his GP 2 months previously due to longstanding symptoms of gastro-oesophageal reflux: specifically, heartburn following eating and acid reflux upon lying flat in bed. No red-flag features of upper gastrointestinal tract malignancy had been identified.

An initial endoscopy was performed with results as documented below. Following discussion of the histology results, the patient elected to undergo endoscopic radiofrequency ablation. The procedure was uncomplicated aside from some mild chest pain in the first few days of recovery.

The patient was otherwise in good health, with other past medical history including only hypertension and osteoarthritis of both knees. Regular medications included paracetamol, omeprazole, ramipril and bendroflumethiazide. The patient reported a previous adverse reaction to penicillin but could not recall the details of this event.

There was no family history of Barrett's oesophagus or oesophageal adenocarcinoma. The patient had never smoked and reported consuming 20 units of alcohol per week.

Report from initial endoscopy: minor hiatus hernia; 5 cm length of circumferential salmoncoloured epithelium extending above gastro-oesophageal junction; mild oesophagitis elsewhere; no mass lesion or ulceration in oesophagus or stomach; quadrant biopsies taken as per protocol.

Histology from endoscopic biopsies: all samples demonstrate flat intestinal metaplasia; mild dysplasia present in 4 of 8 samples; no high-grade dysplasia or adenocarcinoma.

Following successful endoscopic therapy, what is the appropriate schedule for follow-up surveillance endoscopy?

A. Every 6 months for 2 years, then every 1 year thereafter
B. Every 3 months for 1 year, then every 6 months for 1 year, then every 1 year thereafter
C. Every 1 year
D. Every 6 months for 1 year, then every 1 year thereafter
E. Every 3 years

D. Every 6 months for 1 year, then every 1 year thereafter

The patient has Barrett's oesophagus associated with low-grade dysplasia, most likely secondary to his chronic gastro-oesophageal reflux and hiatus hernia. Endoscopic eradication therapy is indicated if any grade of dysplasia is present. Radiofrequency ablation is the preferred ablative therapy in patients with flat dysplasia or intra-mucosal carcinoma. This procedure is generally well tolerated aside from some short-lived chest pain. In the long-term, oesophageal strictures may occur. Radiofrequency ablation is effective, with 91 % of patients achieving complete eradication of dysplasia.

Following successful eradication of low-grade dysplasia, surveillance endoscopy should be performed every 6 months for the first year and then annually thereafter. After eradication of highgrade dysplasia or intra-mucosal carcinoma, surveillance should be more frequent: every 3 months for the first year, every 6 months for the second year and then annually thereafter.

Question 118 of 235

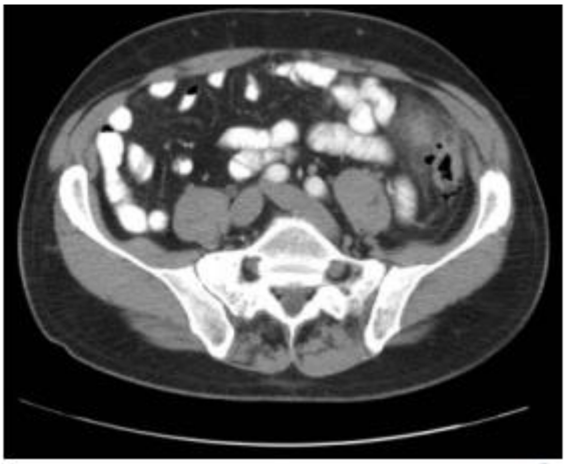
A 51-year-old man is admitted with a three-day history of abdominal pain, fever, and diarrhoea. The pain is localised to the left side of the abdomen. He has been opening bowels approximately five times per day. These have been loose and he has noticed some bright red blood mixed within the stools. There is a history of nausea but nil vomiting. There is no history of previous episodes nor any relevant past medical history. He is an active smoker with a 30 pack-year history.

His respiratory rate is 18/min, and oxygen saturations are 97% on air. The blood pressure is measured as 119/81mmHg and heart rate as 91/min. He is tender in the left lower quadrant and there are some loose, bloody stools on examination of the rectum. He is alert and has a temperature of 38.1ºC.

Admission blood results are shown below:

Hb	136 g/L	(135-180)
Platelets	201 * 10^9 /L	(150 - 400)
WBC	17.1 * 10^9 /L	(4.0 - 11.0)
Na^+	133 mmol/L	(135 - 145)
K^+	4.6 mmol/L	(3.5 - 5.0)
Urea	7.4 mmol/L	(2.0 - 7.0)
Creatinine	91 µmol/L	(55 - 120)
CRP	74 mg/L	(< 5)

A CT is performed and displayed below:



What is the most likely diagnosis?

A. Crohn's disease
B. Diverticulitis
C. Infective colitis
D. Large bowel obstruction
E. Ulcerative colitis

B. Diverticulitis

This man's clinical presentation and CT are both indicative of a diagnosis of diverticulitis. This commonly presents with abdominal pain (most commonly but not exclusively in the left lower quadrant) with bloody diarrhoea. The CT shows diverticulosis, represented by thickened, shortened bowel with outpouching of the bowel wall. This is typically located in the descending and sigmoid colon. The enhancement of the bowel wall and fat stranding are signs of active inflammation (and therefore diverticulitis).

Crohn's disease can present similarly to diverticulitis however the imaging here showing inflamed diverticula distinguishes between the two. Like other causes of colitis, Crohn's will present as thickened, inflamed bowel wall but importantly in the absence of diverticular.

Infective colitis may also present similarly to Crohn's, but as mentioned above, the presence of inflamed diverticular on the CT findings is specific for diverticulitis.

A large bowel obstruction would show a transition point with dilated bowel proximally however this is not shown on the CT in this question. This CT shows thickened bowel and fat stranding consistent with an inflammatory process so this answer is incorrect.

Ulcerative colitis is another reasonable differential diagnosis given the presentation however the CT showing the features listed above is specific for diverticulitis rather than colitis. Ulcerative colitis will appear in a very similar fashion to Crohn's however certain features such as the location and thickness of inflammation can help to distinguish between the two.

Question 119 of 235

A 74-year-old woman is being treated in a geriatric medical ward. She initially presented with community-acquired pneumonia with no relevant past medical history. After receiving a week of amoxicillin, she developed profuse diarrhoea and an acute kidney injury. Cultures from a stool sample grew the following:

Stool MC&S	*Clostridium difficile*

She has not previously had a *Clostridium difficile* infection. She was started on antibiotics and received rehydration. Her acute kidney injury is improving and she is clinically stable however she is still having profuse diarrhoea despite receiving 10 days of oral vancomycin.

What is the most appropriate ongoing antibiotic treatment?

A. Continue oral vancomycin
B. Intravenous metronidazole
C. Oral fidaxomicin
D. Oral vancomycin and intravenous metronidazole
E. Stop antibiotics

C. Oral fidaxomicin

If *C. difficile* does not respond to first-line vancomycin , oral fidaxomicin should be used next, except in life-threatening infections

NICE guidance for antibiotic use in *Clostridium difficile* infection advises oral vancomycin first-line for non-life-threatening cases, followed by oral fidaxomicin second-line. Oral vancomycin and intravenous metronidazole can be considered if first- and second-line therapies fail or in lifethreatening cases. Relapse and recurrence also have their own guidance.

This question asks for the second-line treatment for the first presentation of non-life-threatening *Clostridium difficile* infection. After not responding to 10 days of oral vancomycin, the correct next step would be to switch to **oral fidaxomicin** as per NICE guidance.

Continuing oral vancomycin is not the most appropriate option here. The patient has failed to respond to a 10-day course of oral vancomycin treatment and hence NICE states that escalation to fidaxomicin is indicated.

Intravenous metronidazole can be used together with oral vancomycin as a third-line therapy or in life-threatening *Clostridium difficile* infection however has no role in the guidance as a monotherapy.

As mentioned, **oral vancomycin and intravenous metronidazole** can be used as a third-line therapy or in life-threatening *Clostridium difficile* infection. However as the patient is clinically stable, oral fidaxomicin is a more appropriate second-line option.

Stopping antibiotics is not the most appropriate action here. The patient has not responded to therapy as demonstrated by their ongoing diarrhoea and hence escalation to a second-line antibiotic is indicated.

Question 120 of 235

A 24-year-old female is admitted to the gastroenterology ward with a one-week history of profuse bloody diarrhoea and abdominal cramps. On the second day of her admission, she undergoes a flexible sigmoidoscopy, at which a diagnosis of ulcerative colitis is made. She is commenced on an appropriate regime of intravenous steroids and mesalazine enemas. She is given intravenous fluid and electrolyte replacement and is started on low molecular weight heparin and venous thromboembolism prophylaxis. A pregnancy test is performed on admission which is negative. She is reviewed daily by the gastroenterology team.

After three days of therapy, the patient is still opening her bowels at least 10 times a day. Examination on day three reveals a soft but diffusely tender abdomen. Bloods show:

Hb	92 g/l
Platelets	220 * 10 /l9
WBC	15.2 * 10 /l9
Na +	145 mmol/l
K +	3.4 mmol/l
Urea	9 mmol/l
Creatinine	150 µmol/l
CRP	121 mg/L

What is the next most appropriate management step?

A. Urgent surgical review, further two days of intravenous steroids then re-evaluate
B. Urgent surgical review to request colectomy
C. Urgent surgical review, intravenous ciclosporin 2mg/kg/day
D. Urgent surgical review, intravenous tacrolimus 200mcg/kg/day
E. Application for urgent anti-TNF (tumour necrosis factor) therapy

C. Urgent surgical review, intravenous ciclosporin 2mg/kg/day

This patient is suffering a severe acute flare of ulcerative colitis. It would appear that so far he has been managed appropriately. The British Society of Gastroenterology recommend the following basic measures during an acute flare:

1. Intravenous fluid and electrolyte replacement
2. Blood transfusion if required to maintain a haemoglobin >10g/dL
3. Intravenous antibiotics ONLY if infection is considered or prior to surgery
4. Subcutaneous heparin to reduce the risk of thromboembolism
5. Intravenous corticosteroids (either hydrocortisone 100mg four times a day or methylprednisolone 60mg/day)
6. Flexible sigmoidoscopy and biopsy within 72 hours to confirm diagnosis and exclude cytomegalovirus infection
7. Daily monitoring with physical examination, recording of vital signs, stool chart, bloods (full blood count, c-reactive protein, serum electrolytes, serum albumin, liver function tests and glucose) and consider the need for daily abdominal X-ray.

Intravenous steroids are given for five days with no additional benefit seen beyond seven days.

At day three a c-reactive protein >45 mg/l or a stool frequency of >8/day predicts the need for surgery in 85% of cases.

The National Institute for Clinical Excellence (NICE) recommends that if no improvement is seen after 72 hours of intravenous steroid treatment or if the patient deteriorates at any time despite corticosteroid treatment then an urgent surgical review is required ciclosporin (2mg/kg/day) should be added. NICE recommends that in this acute setting infliximab reserved for patients in whom ciclosporin is contraindicated or clinically inappropriate.

Question 121 of 235

A 48-year-old known alcoholic liver disease patient is admitted via Accident and Emergency with profuse haematemesis. He has been drinking 6 litres of cider per day for the last week. He denies abdominal pain or melena. On examination, he has peripheral stigmata of chronic liver disease and is very pale. His abdomen is soft and he has no tenderness or hepatosplenomegaly. His blood pressure is 90/56 mmHg and he is tachycardic at 120/min. His last OGD 6 months ago showed 3 columns of small varices.

His blood results are as follows:

Hb	58 g/l
Platelets	109 * 10 /l9
WBC	8.4 * 10 /l9
INR	1.6
PT	19 seconds

Na +	144 mmol/l
K +	4.9 mmol/l
Urea	18.1 mmol/l
Creatinine	97 µmol/l
CRP	5 mg/l

Bilirubin	87 µmol/l
ALP	189 u/l
ALT	71 u/l
Albumin	28 g/l

He is transfused 2 units by A&E and given 2 units of Fresh Frozen plasma to correct his coagulopathy. He is also given Tazocin 4.5g TDS and Terlipressin 1mg QDS after discussion with the on call Gastroenterologist. He is taken for OGD which shows bleeding oesophageal varices. He has 5 bands applied to the varices but the endoscopist is unable to stop the bleeding. He is returned to the ward were he continues to have haematemesis with low blood pressure and ongoing tachycardia.

What is the next step in his management?

A. Repeat OGD
B. TIPSS
C. Sengstaken Blakemore tube
D. Increase Terlipressin to 2mg
E. Further FFP

C. Sengstaken Blakemore tube Next question

This gentleman has had a large variceal bleed. This is reflected in his low Hb and high urea. His initial management is appropriate and if haemostasis cannot be achieved then the next step is determined by the patient. If he is stable then a repeat OGD can be performed to try and stop the bleeding but if he is unstable then tamponade of the varices with a Sengstaken Blakemore tube is needed whilst the patient is resuscitated. TIPSS may be a potential treatment option in this patient but would usually follow Sengstaken tube insertion.

Question 122 of 235

A 49-year-old man is referred to the gastroenterology outpatient clinic with a 6 month history of abdominal pain, diarrhoea and weight loss. These symptoms have been getting progressively worse over the past few weeks and are now associated with some arthrlagia affecting his hands and feet. The abdominal pain does not follow a particular pattern. It is associated with bloating and not relieved by defecation. He thinks in total he has lost around 10kg in the past 6 months.

The patient works in the IT industry and regularly travels to South East Asia. He is married and does not smoke.

On examination his pulse, blood pressure and temperature are normal. Generalised lymphadenopathy is noted in the cervical and inguinal regions. All the nodes felt were soft and non-tender. His wedding ring is loose and loss of the nail angle is noted. Examination of the abdomen is unremarkable other than some mild tenderness around the epigastric region. No organomegaly is detected. There was no evidence of swelling, erythema or synovitis in the hands or feet.

Bloods show the following:

Hb	10.4 g/dl
Platelets	222 * 10 /l9
WBC	6.96 * 10 /l9
Na +	139 mmol/l
K +	3.6 mmol/l
Urea	5.1 mmol/l
Creatinine	78 µmol/l
Bilirubin	14 µmol/l
ALP	120 u/l
ALT	34 u/l
γGT	55 u/l
Albumin	26 g/l

What is the most appropriate treatment?

A. IV penicillin then co-trimoxazole
B. Prednisolone
C. Rifampicin, isoniazid, pyrazinamide and ethambutol
D. Gluten-free diet
E. Highly active antiretroviral therapy

A. IV penicillin then co-trimoxazole

This patient has a number of typical features suggestive of the rare condition Whipple's disease:

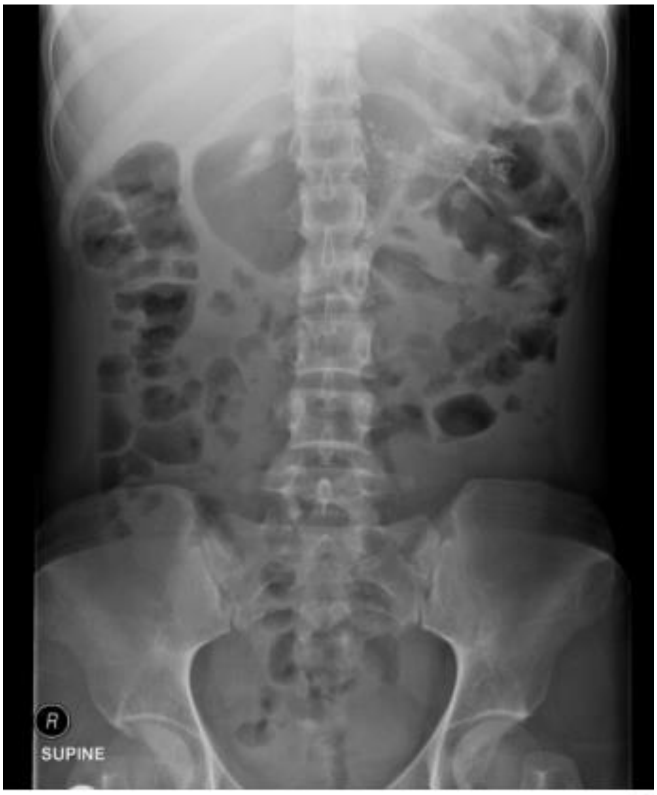
- middle-aged male
- abdominal pain
- weight loss
- diarrhoea
- arthralgia
- lymphadenopathy
- clubbing (loss of the nail angle)

Question 123 of 235

A 30-year-old woman presents to the Emergency Department with abdominal pain. She is currently being investigated by her GP for recurrent episodes of abdominal and back pain that have been ongoing for the past year or so. There is no pattern to when the pain comes on but it is often severe and usually lasts several hours. Her past medical history includes psoriasis for which she uses topical calcipotriol on a daily basis. She is a non-smoker and used to drink excess amounts of alcohol but stopped 6 months ago as it was exacerbating her psoriasis. Her bowels have been looser than normal for the past few months. She has lost around 3kg in the past year but thinks this is due to careful eating.

On examination her abdomen is soft and not distended. There is mild tenderness in the epigastrium but no guarding. Blood pressure, pulse and temperature are normal.

An abdominal x-ray is requested:



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What is the most likely underlying diagnosis?

A. Pancreatic cancer
B. Bony metastates
C. Chronic pancreatitis
D. Crohn's disease
E. Colorectal cancer

C. Chronic pancreatitis

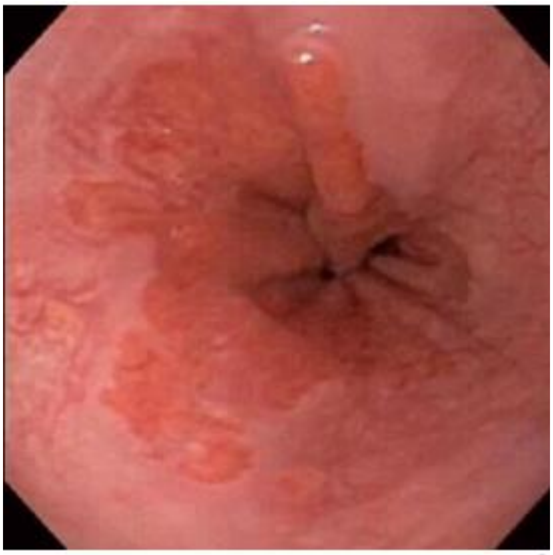
Multiple small calcific foci can be seen in the pancreas consistent with a background of chronic pancreatitis. The history of alcohol excess with recurrent abdominal pain is also in keeping with this diagnosis.

Question 124 of 235

A 53-year-old businessman is referred to for an endoscopy by his GP after complaining of persistent dyspepsia which was not fully controlled with lansoprazole. His symptoms have been getting gradually worse for the past six months. He smokes around 20 cigarettes/day and drinks about 30 units of alcohol per week. There is no history of dysphagia or vomiting although he does describe some pain when swallowing foods such as meat. Clinical examination is unremarkable.

A photograph is taken of his lower oesophagus during endoscopy:

Biopsies are taken and are reported as follows:



Intestinal metaplasia with a villiform pattern noted. Multiple intermediate mucous cells and goblet cells noted. No dyplasia noted.

What is the most appropriate management?

A. Oral fluconazole
B. Oesophagogastrectomy
C. High dose proton pump inhibitor therapy
D. Endoscopic ablation
E. High dose proton pump inhibitor therapy + endoscopic surveillance

E. High dose proton pump inhibitor therapy + endoscopic surveillance

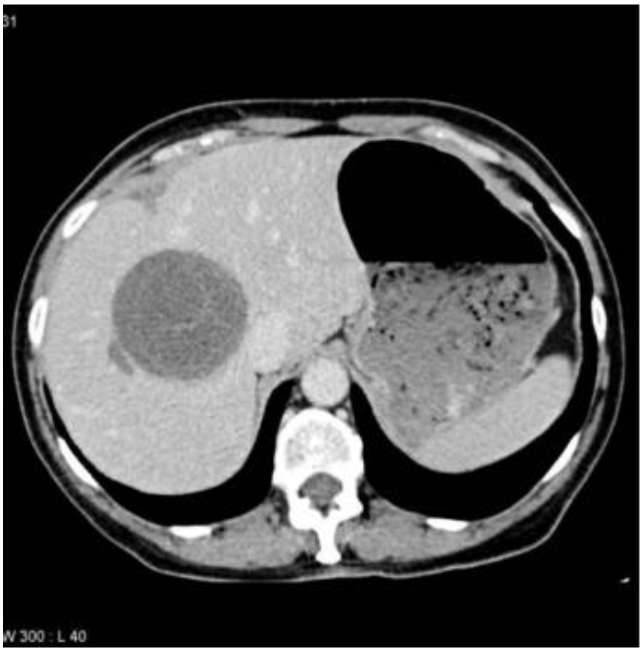
This is Barrett's oesophagus. The metaplastic mucosa needs to be monitored on a regular basis to check for any dysplastic or malignant changes. High dose proton pump inhibitor therapy is also

Question 125 of 235

A 62-year-old man who is an immigrant from Turkey is investigated for abnormal liver function tests. His most liver function tests show the following:

Bilirubin	42 µmol/l
ALP	221 u/l
ALT	98 u/l
γGT	131 u/l
Albumin	36 g/l

He does complain of an occasional dull ache in the right upper quadrant. A CT scan is requested:



Over the past two weeks he has also had recurrent episodes of an intensely itchy urticarial rash developing. These episodes typically last around 2 hours before settling and are helped by oral antihistamines.

What is the most likely diagnosis?

A. Budd-Chiari Syndrome
B. Amoebic liver abscess
C. Cysticercosis
D. Hepatocellular carcinoma
E. Hydatid cyst

E. Hydatid cyst question

The CT scan shows a hydatid cyst. These are more common in Mediterranean countries, the Middle East and southern parts of South America.

The recurrent episodes of urticaria are likely to be secondary to the contents of the cyst leaking. Much of the morbidity related to hydatid cysts is caused by anaphylactic reactions.

Question 126 of 235

A 84-year-old lady is admitted to the Emergency Department from her nursing home with diarrhoea, abdominal pain and fever. Her symptoms started around 2 days ago and have been getting progressively worse. She is now opening her bowels every other hour and is complaining of severe 'cramp' in her lower abdomen. Her past medical history includes hypertension, ischaemic heart disease, hypothyroidism and recent treatment for suspected pyelonephritis.

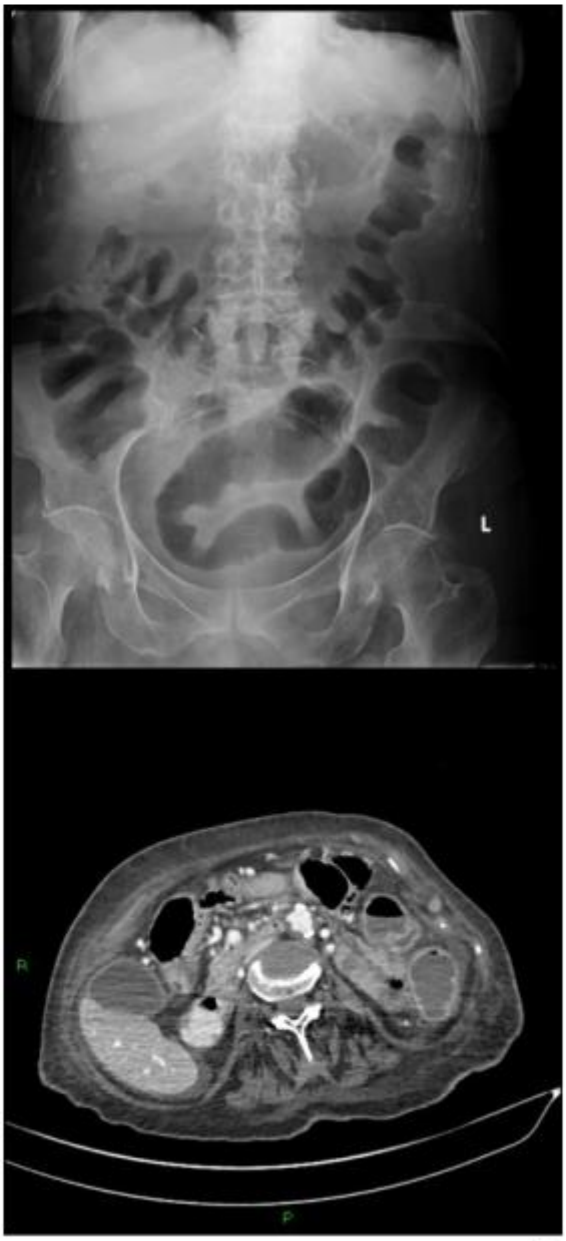
On examination she is diffusely tender across the lower abdomen. Her pulse is 90/min, blood pressure 100/60 mmHg and temperature 37.4ºC.

Bloods show the following:

Hb	11.1 g/dl	Na^+	144 mmol/l
Platelets	365 * 10^9/l	K^+	4.6 mmol/l

WBC	25.2 * 10 /l 9	Urea	10.6 mmol/l
Creatinine	99 µmol/l		
CRP	86 mg/l		

The surgeons request an abdominal film and later a CT abdomen:



What is the most likely diagnosis?

A. Perinephric abscess
B. Ischaemic colitis
C. Acute diverticulitis
D. Metastatic colorectal cancer
E. Clostridium difficile colitis

E. Clostridium difficile colitis

The clues to *Clostridium difficile* colitis in this question include

- age of patient
- high white cell count
- profuse nature of diarrhoea
- recent antibiotic use for pyelonephritis (likely to have been co-amoxiclav or ciprofloxacin)
- imaging findings (see below)

On the abdominal film note the loss of bowel wall architecture and thumb-printing consistent with colitis. The CT from the same patient is enhanced by oral contrast. There is moderate free fluid in pelvis and peritoneum. The colon is oedematous throughout with enhancing walls, but of normal calibre. The sigmoid colon is smooth and featureless. Small bowel, liver, spleen, kidneys, adrenals and pancreas are normal.

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A 33-year-old lady has a diagnosis of irritable bowel syndrome (IBS). She has previously been seen in the gastroenterology clinic and all investigations including colonoscopy were normal. She is mainly troubled by abdominal pain, bloating and constipation. She continues to have symptoms despite the use of antispasmodics, regular Movicol (macrogol laxative) and input from a dietician. She has tried other laxatives in the past with limited benefit.

What would be the next most appropriate option?

A. Lactulose
B. Linaclotide
C. Loperamide
D. Acupuncture
E. Reflexology

B. Linaclotide

NICE have written guidance on the diagnosis and management of IBS.

- First line pharmacological treatments include antispasmodics e.g. hyoscine or mebeverine; loperamide for diarrhoea and laxatives for constipation. Lactulose should be avoided.
- 2nd line options include tricyclic antidepressants e.g. up to 30mg amitriptyline if above treatments have not helped
- 3rd line: serotonin selective reuptake inhibitors
- Linaclotide can be considered if there has been no benefit from different laxatives and the patient has had constipation for at least 12 months.

Other management options include dietary advice and psychological treatments.

Acupuncture and reflexology are not recommended for managing IBS.

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A 38-year-old female presents to the emergency department with a 6-hour history of severe upper abdominal pain with radiation to the back. She reports some nausea and vomiting but denies any diarrhoea or fever.

She has no significant past medical history apart from a laparoscopic appendicectomy for appendicitis when she was 20 years old, and a

normal vaginal delivery 5 years ago. She does not smoke but has 1 glass of wine approximately 3 times per week.

She looks unwell and examination of her abdomen reveals epigastric tenderness.

Hb	135 g/l
Platelets	402 * 10 /l9
WBC	14 * 10 /l9
Neutrophils	13.5 * 10 /l9
Lipase	1200 U/l
Total cholesterol	5.4 mmol/L
HDL	1.2 mmol/L
LDL	4 mmol/L
Triglyceride	2 mmol/L

What is the most likely underlying cause of her symptoms?

A. Coxsackie virus
B. Hypertriglyceridaemia
C. Hepatitis B virus
D. Alcohol abuse
E. Gallstones

A. Gallstones

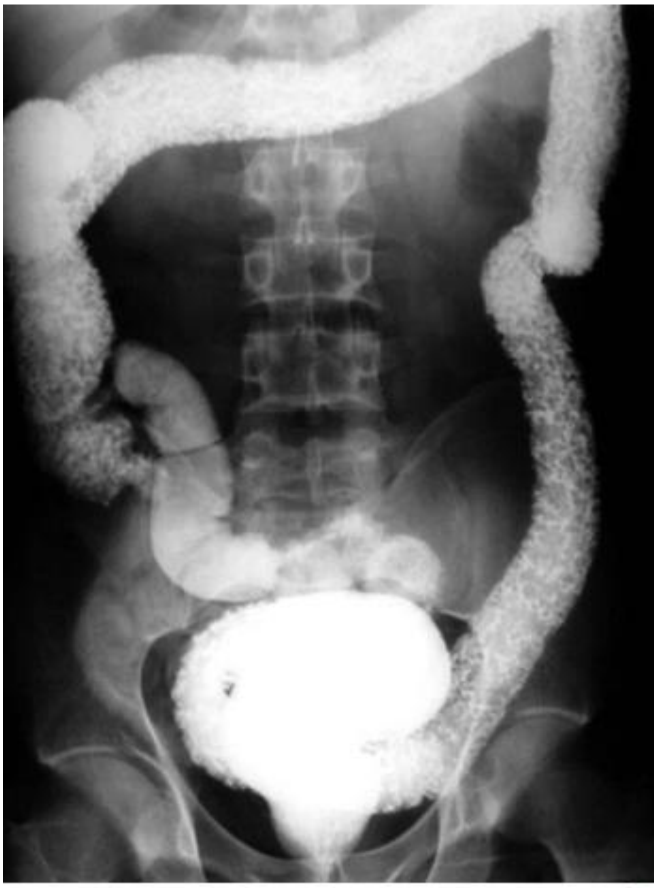
This patient has acute pancreatitis. While there are multiple causes of acute pancreatitis, the most common causes include gallstones and alcohol intake. Less common causes include recent endoscopic retrograde cholangiopancreatography (ERCP), infections such as mumps and Coxsackie virus, hypertriglyceridaemia, hypocalcaemia, medications and sphincter of Oddi dysfunction.

It would be very unusual for her alcohol intake or current triglyceride levels to trigger pancreatitis. In a significant proportion of cases of pancreatitis, no specific cause is found (idiopathic).

Question 129 of 235

A 40-year-old man is investigated for diarrhoea which has persisted for around 3 months. Prior to this he was fit and well with no past medical history of note. He initially had some abdominal pain but this has now settled. He is now passing around 5 loose, non-bloody stools per day.

His past medical history includes lower back pain for which he takes regular ibuprofen. A barium enema is requested:



What is the most likely diagnosis?

A. Colon cancer
B. Perforated duodenal ulcer
C. Ulcerative colitis
D. Infective gastroenteritis
E. Crohn's disease

C. Ulcerative colitis

The whole colon, without skip lesions, is affected by an irregular mucosa with loss of normal haustral markings.

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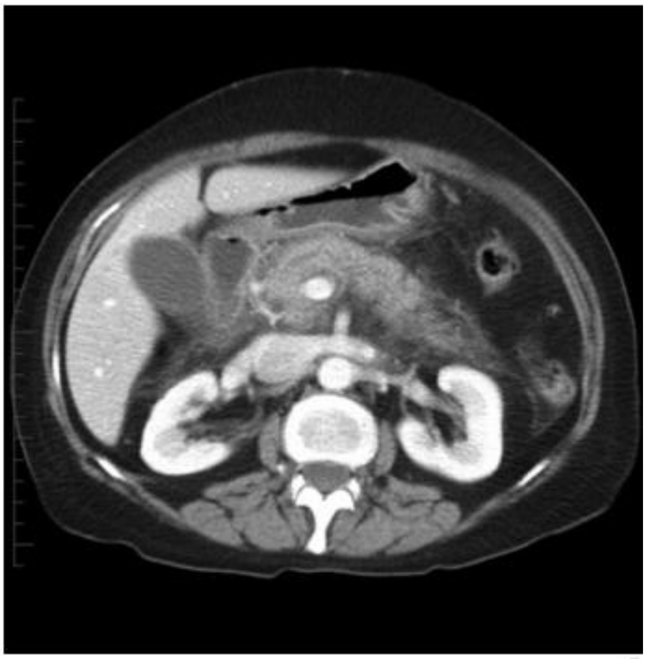
A 54-year-old woman who has recently been diagnosed with autoimmune hepatitis presents to the Emergency Department with central abdominal pain. This has been getting worse for the past 48 hours and is now 8/10 in terms of severity.

On examination she is tender in the epigastrium.

For the past 3 weeks she has been taking prednisolone 40mg od. Her long term medication includes amlodipine and lisinopril for hypertension. She is a non-smoker and drinks 10 units of alcohol/week.

A CT abdomen is requested:



What is the most likely diagnosis?

- A. Acute cholecystitis
- B. Perforated duodenal ulcer
- C. Hepatocellular carcinoma
- D. Psoas abscess
- E. Acute pancreatitis

E. Acute pancreatitis

The CT is consistent with acute pancreatitis. Note the diffuse parenchymal enlargement with oedema and indistinct margins.

Her recent steroid use is the likely precipitant of this episode.

Question 131 of 235

A 76-year-old man presents with jaundice. He is a heavy drinker, consuming approximately 35-40 units per week. There is no history of abdominal pain, and pain is not elicited on abdominal examination. However, examination does reveal a palpable gallbladder. Bloods are taken and the results are:

Albumin	30 g/L
Alk Phos	342 U/L
ALT	95 U/L
Bilirubin	102 mol/L
INR	1.4
GGT	123 U/L

Which of the following is the likely diagnosis?

- A. Alcoholic hepatitis
- B. Pancreatic cancer
- C. Primary biliary cirrhosis
- D. Paracetamol overdose
- E. Gallstones

B. Pancreatic cancer

The correct answer for this question is pancreatic cancer.

Courvoisier's sign states that in a patient with a painless, enlarged gallbladder and mild jaundice the cause is unlikely to be gallstones. Furthermore, it is more likely to be a malignancy of the pancreas or biliary tree.

Alcoholic hepatitis and primary biliary cirrhosis are a reasonable differentials. However, an examination finding of a painless, enlarged gallbladder makes both of these differentials less likely.

The scenario does not suggest paracetamol overdose, as this would not cause a painless, palpable gallbladder. Furthermore, jaundice is not usually seen in paracetamol overdose.

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A 59-year-old male with known liver cirrhosis presents to the acute medical unit with a raised creatinine from baseline of 100µmol/L to 180µmol/L. He denies any reduced oral fluid intake. On examination, he is well perfused. There are no signs of dehydration. His abdomen is distended and shifting dullness is positive.

CRP	10mg/L

Hepatorenal syndrome is suspected. Which of the following is the first line treatment?

- A. Bolus of normal saline fluid
- B. Bolus of Hartmann's solution
- C. Terlipressin
- D. Terlipressin with albumin infusion
- E. Intravenous antibiotics

D. Terlipressin with albumin infusion Next question

There are two types of hepatorenal syndrome. Type one is more aggressive with a worse prognosis. Regardless which type of hepatorenal syndrome, initial treatment is the same, which is intravenous terlipressin with albumin infusion. Simply giving fluid is not going to solve the problem because of the splanchnic vasodilation. There are no signs to suggest the acute kidney injury is secondary to sepsis which would require intravenous antibiotics.

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A 48-year-old gentleman presents to the Emergency Department with a 3-day history of fever, nausea, anorexia, and malaise. His past medical history is unremarkable but he admits to drinking heavily for many years. He estimates his present consumption at half-bottle of vodka on 5-6 days of the week.

Examination reveals an unwell and generally tremulous middle-aged man. His temperature is 38.2ºC, his pulse is 113bpm and his blood pressure is 95/57mmHg. Scleral icterus is clearly evident, even from the end of the bed. His abdomen is mildly distended but soft. There is tender hepatomegaly and a bruit is audible on auscultation of the right upper quadrant.

His blood tests are as follows:

Hb	97 g/l	Na +	142 mmol/l	Bilirubin	173 µmol/l

MCV	103.1 fl	K +	3.1 mmol/l	ALP	184 u/l
Platelets	46 * 10 /l 9	Urea	2.1 mmol/l	ALT	109 u/l
WBC	15.1 * 10 /l 9	Creatinine	51 µmol/l	AST	276 u/l
Neuts	13.9 * 10 /l 9	γGT	412 u/l		
Lymphs	0.6 * 10 /l 9	Albumin	24 g/l		
INR	1.4				

You calculate his Maddrey Discriminant Score (MDS) as 37.7.

Given the likely diagnosis, which of the following is the most appropriate treatment strategy?

A. Pabrinex, nutritional support, prednisolone and pentoxifylline
B. Pabrinex, nutritional support and pentoxifylline
C. Pabrinex and nutritional support only
D. Pabrinex, nutritional support and prednisolone
E. Pabrinex, nutritional support and N-acetylcysteine

D. Pabrinex, nutritional support and prednisolone

The patient has presented with alcoholic hepatitis, as evidenced by the history of excessive alcohol consumption, macrocytosis, and AST:ALT ratio >2:1. Alcoholic hepatitis almost always presents with clinical jaundice and the presence of a hepatic bruit is particularly suggestive.

An MDS of > 32 is associated with 50% mortality within 2 months and indicates the need for treatment. All patients should receive vitamin replenishment and nutritional support. Patients with alcoholic hepatitis should be encouraged to consume >2000 kcal per day with supplementation via NG feeding if necessary.

Prednisolone has been shown in meta-analyses to confer a significant reduction in 28-day mortality in patients with an MDS >32 or hepatic encephalopathy, however, there was no demonstrable benefit at 90 days or at one year. Pentoxifylline may reduce mortality in the presence of hepatorenal syndrome (HRS) but evidence of efficacy in patients without HRS is lacking.

Combination therapy with prednisolone and pentoxifylline confers no additional benefit over treatment with prednisolone alone, whilst the use of N-acetylcysteine as monotherapy in alcoholic hepatitis is not recommended. In one randomised controlled trial, combination therapy with prednisolone and N-acetylcysteine was shown to be superior to prednisolone monotherapy in reducing mortality at 28 days; largely due to a reduction in the incidence of HRS and superimposed infection.

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A 44-year-old gentleman is referred to the gastroenterology clinic with deranged LFTs. His only symptom is mild fatigue, which does not interfere with his daily activities. He denies itch, nausea, vomiting and recent foreign travel.

His past medical history is remarkable for depression, which is controlled with 20mg citalopram once daily. He has been taking this for the last 6 years. He works long hours as a pastry chef and often eats at unusual times. He admits to drinking a 'glass of wine or two most nights' but denies any intravenous drug use.

Examination reveals an overweight gentleman with no obvious jaundice. His abdomen is soft with mild right upper quadrant tenderness on deep palpation. There is no obvious hepatomegaly. Bowel sounds are present.

A set of blood tests are requested:

Hb	143 g/l	Na +	142 mmol/l	Bilirubin	24 µmol/l
Platelets	317* 10 /l 9	K +	3.9 mmol/l	ALP	176 u/l
WBC	6.4* 10 /l 9	Urea	7.1 mmol/l	AST	225 u/l
Neuts	4.2* 10 /l 9	Creatinine	109 µmol/l	ALT	112 u/l
Lymphs	1.7* 10 /l 9	γGT	108 u/l		
Eosin	0.1* 10 /l 9	Albumin	37 g/l		

What is the most likely diagnosis?

A. Haemochromatosis
B. Non-alcoholic fatty liver disease (NAFLD)
C. Cholelithiasis
D. Viral hepatitis
E. Alcoholic liver disease (ALD)

E. Alcoholic liver disease (ALD)

The patient is overweight and his occupation offers him access to a number of rich foods, making NAFLD a plausible diagnosis. However, closer inspection of his LFTs reveals that the patient's AST:ALT ratio is > 2:1 and his γGT is double the upper limit of normal. This is more in keeping with ALD, and this is the correct answer.

Haemochromatosis is a common cause of liver dysfunction, but there is no history of skin pigmentation, arthralgia or diabetes mellitus. There is nothing in the history to suggest viral hepatitis whilst one would expect cholelithiasis to present with episodic, severe right upper quadrant pain rather than an incidental finding of deranged LFTs.

Question 135 of 235

A 65-year-old woman is admitted to the medical assessment unit for evaluation of ascites. She has a history of excess alcohol consumption for many years and admits to previous illicit drug use. Other history of note includes a hysterectomy with preservation of ovaries, and symptoms of irritable bowel syndrome during the past six months.

On examination her blood pressure is 110/70 mmHg, the pulse is 87 beats per minute and regular. There are no heart murmurs and the chest is clear. The abdomen is distended with obvious ascites and her body mass index is 26 kg/m².

Hb	100 g/l	Na +	134 mmol/l	Bilirubin	24 µmol/l
Platelets	112 * 10 /l 9	K +	3.9 mmol/l	ALP	190 u/l

WBC	8.0 * 10 /l 9	Urea	6.3 mmol/l	ALT	112 u/l
Neuts	4.5 * 10 /l 9	Creatinine	85 µmol/l	γGT	135 u/l
Lymphs	2.1 * 10 /l 9	Albumin	32 g/l		
Eosin	0.5 * 10 /l 9	Serum ascites albumin gradient	7.5 g/l		

Which of the following is the most likely diagnosis?

A. Hepatic cirrhosis
B. Ovarian carcinoma
C. Viral hepatitis
D. Nephrotic syndrome
E. Right heart failure

B. Ovarian carcinoma

A low serum-ascites albumin gradient (SAAG <11 g/L) indicates causes of ascites not associated with increased portal pressure. These include tuberculosis, other causes of peritoneal sepsis, pancreatitis, serositis, nephrotic syndrome and peritoneal carcinomatosis. Clues towards a diagnosis of ovarian carcinoma include the history of irritable bowel syndrome and the fact that she still has her ovaries post hysterectomy.

The lack of history of any previous renal disease counts against nephrotic syndrome, cirrhosis, right heart failure and viral hepatitis would be associated with an elevated SAAG.

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A 49-year-old man with a history of alcoholism has been admitted to the ward for a detoxification program. He was brought to the hospital by police in an intoxicated state. He has been treated with B vitamin replacement therapy and has now begun re-feeding. You are asked to see him because of acute shortness of breath and generalised muscle weakness. On examination his blood pressure is 100/80 mmHg, pulse is 86 beats per minute and regular. He has bilateral crackles on auscultation of his chest consistent with heart failure, and global 4/5 power weakness.

Which of the following is the most likely cause of his symptoms?

A. Low thiamine
B. Low phosphate
C. Low potassium
D. Low folate
E. Low magnesium

B. Low phosphate

Globalised muscle weakness coupled with symptoms of heart failure is consistent with re-feeding syndrome. This occurs because of intra-cellular movement of phosphate, thought to be related to be related to hyperinsulinaemia because of carbohydrate loading. Heart failure and muscle weakness can be avoided by ensuring that any feeds used contain adequate phosphate when used in the treatment of patients at risk of re-feeding syndrome, such as those with alcoholism and / or eating disorders.

Low thiamine results in Wernicke-Korsakoff syndrome. Although low potassium can result in muscle weakness and is seen in reactive hyperinsulinaemia, feeds normally contain adequate potassium to avoid this, hence it's less likely than hypophosphataemia in this situation. Folate deficiency is manifest by anaemia, glossitis, and neuropathy, and magnesium deficiency presents with similar symptoms to hypocalcaemia.

Question 137 of 235

A 42-year-old man has been diagnosed with diabetes following a resection for chronic pancreatitis. He has been initiated on insulin therapy and comes to the clinic to discuss a target HbA1c. His GP has been striving for a target HbA1c of 42, although the patient has pushed back, saying that this is resulting in significant hypoglycaemia. Clinical examination is unremarkable, his blood pressure is 142/82 mmHg, pulse is 80 and regular. His chest is clear. He is thin with a midline scar on his abdomen, his body mass index is 21 kg/m².

Which of the following is an appropriate target HbA1c for him?

A. 31
B. 37
C. 42
D. 48
E. 53

E. 53

It's important to remember in diabetes related to pancreatic resection, both alpha cells producing glucagon, and beta cells producing insulin, are removed in the pancreatectomy. This reduces the intensity of any counter regulatory response to hypoglycaemia and thus impacts on prospects of recovery and increases the severity of individual events. For this reason a more lax HbA1c target is instigated for patients who have a history of pancreatectomy, of 53. HbA1cs of 31 and 37 are actually within the normal range.

Question 138 of 235

You are asked to review a 29-year-old man who is an inpatient on the orthopaedic ward having undergone emergency surgery for a fractured femur some 36hrs earlier. Surgery was uneventful, there were no significant episodes of hypotension. The staff are concerned as they have seen a marked rise in his transaminases since the surgery. He has no past medical history of note and takes no regular medication. On examination his blood pressure is 115/80 mmHg, his pulse is 80 beats per minute and regular. He is apyrexial and looks comfortable at rest. Abdomen is soft and non-tender with no masses. His leg is elevated in a cast.

Investigations reveal:

Bilirubin	22 µmol/l
ALP	220 u/l
ALT	1150 u/l
γGT	520 u/l
Albumin	32 g/l

Which of the following is the most likely diagnosis?

A. Anaesthetic related hepatitis
B. Hepatic ischaemia
C. Staphylococcal septicaemia
D. Transfusion reaction
E. Viral hepatitis

A. Anaesthetic related hepatitis

Although the incidence of anaesthetic related hepatitis has decreased over recent years with the introduction of modern agents such as desflurane and sevoflurane, cases are still reported. The rapid rise in transaminases, gamma GT and alkaline phosphatase so soon after surgery fits well with the diagnosis. Supportive therapy is the only option, no therapeutic interventions, (including corticosteroids), have proved effective.

Hepatic ischaemia is unlikely given there were no episodes of hypotension during the course of surgery. Staphylococcal septicaemia would be unlikely to present only with symptoms of hepatitis, and a transfusion reaction would be seen in much closer proximity to the transfusion itself. Viral hepatitis would be subject to an interval of 2-3 weeks before symptoms occur.

Question 139 of 235

A 72-year-old woman is admitted with diarrhoea having recently been an inpatient with urinary sepsis. She is having 8-10 episodes per day of watery, foul-smelling diarrhoea. Stool is sent for testing and *Clostridium difficile* toxin is detected.

She is treated with a 10-day course of oral vancomycin. At the end of this course, she describes no improvement and still has diarrhoea 8-10 times per day. Observations are as follows.

- Respiratory rate 18 /min
- Oxygen saturations 97% on air
- Heart rate 94 beats /min
- Blood pressure 141/72mmHg
- Temperature 38.1ºC

On examination, she appears dehydrated but the abdomen is soft and non-tender. An abdominal X-ray is requested and is unremarkable.

What is the best course of action?

A. Add IV metronidazole
B. Faecal microbiota transplant
C. Refer to general surgery
D. Switch to oral fidaxomicin
E. Switch to vancomycin enema

D. Switch to oral fidaxomicin

If *C. difficile* does not respond to first-line vancomycin , oral fidaxomicin should be used next, except in life-threatening infections

The correct answer is **switch to oral fidaxomicin**. Vancomycin is generally an effective first-line treatment but some patients may fail to respond. In such cases, NICE recommends switching to oral fidaxomicin for a 10-day course. In this case, the ongoing fever and the lack of decrease in stool frequency suggest non-resolving infection rather than other causes such as post-infectious irritable bowel syndrome.

Add IV metronidazole is incorrect. Oral vancomycin in combination with IV metronidazole has a role in life-threatening infection or in mild/moderate/severe infection that has not responded to first- or second-line treatment. In the case described, there are no life-threatening features and the patient has so far only received first-line treatment.

Faecal microbiota transplant is incorrect. This is recommended as a treatment option in recurrent *Clostridium difficile* infection where a patient has 2 or more previous episodes. It is not recommended in acute infection as in the case described.

Refer to general surgery is incorrect. Surgical management is only warranted in the context of life-threatening infection. As above, there are no features of life-threatening infection in the case described.

Switch to vancomycin enema is incorrect. Vancomycin via the nasogastric or rectal route is only indicated if the oral route is not available. In the case described, the patient's oral route is suitable and an alternative oral treatment should be offered.

Question 140 of 235

A 58-year-old female was referred to the gastroenterology clinic. Her GP had detected on routine blood investigations deranged liver function testing which despite repeated testing was found to be persistent. Her past medical history comprised diabetes mellitus type 2 diagnosed twelve years ago. Unfortunately, this was poorly controlled, despite the use of metformin 500mg TDS, glitazone 160mg OD, pioglitazone 15mg OD, aspirin 75mg OD and Lantus 42 units OD. She also suffered from diabetic nephropathy for which she was using ramipril 10mg OD as well as diabetic retinopathy requiring laser treatment. No other past medical history was noted. She strongly denied consuming alcohol and did not smoke. She admitted to a brief period of intravenous drug use many years ago lasting a few months but as far as she was aware of did not suffer from any sequelae arising from this.

On examination, she was noted to be obese but otherwise systemically well. Blood pressure was recorded as 138/86 mmHg. The cardiovascular and respiratory examination was otherwise normal. Gastrointestinal examination revealed a palpable mass in the right upper quadrant with a smooth border, 3 fingerbreadths from the lower costal margin. There were no other palpable masses and no other stigmata of chronic liver disease. Neurological examination was unremarkable.

Investigations revealed the following:

Hb	141g/l
Platelets	212 * 10 /l9
WBC	6.4 * 10 /l9
Na +	144 mmol/l
K +	3.9 mmol/l
Urea	5.8 mmol/l
Creatinine	81 µmol/l
Bilirubin	18 µmol/l
ALP	74 u/l

ALT	162 u/l
AST	28 u/l
Albumin	34 g/l
Calcium	2.12 mmol/l
Adjusted calcium	2.38 mmol/l
Total protein	76 g/l
HbA1c	66 mmol/mol

Fasting cholesterol	3.8 mmol/l

HBsAg	negative
Anti-HBs antibody	positive
Anti-HBc antibody	negative
HBeAG	negative
Anti-HBe antibody	negative
Anti Hep C antibody	negative
HIV serology	negative

Iron studies	normal
Copper studies	normal
ANA	positive
AMA	negative
SMA	negative

Ultrasound: bright hyper-echogenic image What is the single most likely diagnosis?

A. Autoimmune hepatitis
B. Nonalcoholic steatohepatitis
C. Chronic hepatitis B infection
D. Alcoholic hepatitis
E. Drug induced hepatitis

NOT ANSWERED

Question 141 of 235

A 40-year-old man has been found to have a liver abscess. Given that he had spent extended periods of time in South East Asia and the rusty brown colour of the pus drained, microbiologic testing included looking for parasites. It has since been found that the causative agent was Entamoeba histolytica.

Which medication is most appropriate?

A. Metronidazole
B. Cefepime
C. Chloroquine
D. Ceftriaxone
E. Diloxanide furoate

A. Metronidazole Next question

Entamoeba histolytica is more common in people who have lived in tropical areas with poor sanitary conditions. First-line treatment is with metronidazole. Chloroquine may be used but it is not first line.

Diloxanide furoate should be given after the completion of metronidazole or tinidazole to kill remaining amoeba in the gut. It is also the drug of choice when E.histolytica is found in the faeces of asymptomatic patients as metronidazole and tinidazole are relatively ineffective.

Broad spectrum antibiotics may be used where there is a concern of bacterial superinfection but they are not recommended as the first line.

Question 142 of 235

An 80-year-old lady was admitted with two episodes of 'black watery diarrhoea'. On the way to the hospital, she collapsed in the ambulance, losing consciousness for 5 minutes. She does not remember losing consciousness in the ambulance, but is otherwise orientated, and denies any preceding symptoms before the episode of diarrhoea. Her past medical history includes hypertension, arthritis and gastritis.

On examination she is alert, her heart rate is 80 bpm, blood pressure is 126/76 mmHg, respiratory rate of 18 breaths per minute and saturating at 98% on air. Her abdomen is soft with a mildly tender epigastrium. Rest of the examination is unremarkable. Rectal examination reveals an empty rectum, no melena or bleeding.

Na+	141 mmol/l
K+	4.8 mmol/l
Urea	11.1 mmol/l
Creatinine	80 µmol/l
Serum bilirubin	21 µmol/l
Serum alkaline phosphatase	97 IU/l
Serum aspartate aminotransferase	22 IU/l
C Reactive protein (CRP	<1 mg/l
Haemoglobin	116 g/l (prev 11.8)
White cell count	7.2 x 10^9/L
INR	1.0

According to the NICE guidelines, how soon should this lady undergo an oesophageal-gastroduodenoscopy (OGD)?

A. Immediately
B. Within 6 hours
C. Within 12 hours
D. Within 24 hours
E. Within 1 week

D. Within 24 hours

This lady has presented with signs and symptoms of an upper gastrointestinal (GI) bleed. Her Blatchford score is 5, which signifies a high-risk GI bleed that is likely to require medical intervention: either transfusion, endoscopy or surgery.

However, she is haemodynamically stable and does not appear to be actively bleeding on assessment. According to NICE guidelines, endoscopy should be offered to unstable patients with severe acute upper GI bleeding immediately after resuscitation, and endoscopy should be offered within 24hrs of admission to all other patients with an upper GI bleed.

This patient should be fluid resuscitated, and unless a variceal bleed is suspected, proton pump inhibitors should be held until after the endoscopy. If the patient is on aspirin, clopidogrel or other anti-platelet agents they should be held too.

Question 143 of 235

A 56-year-old gentleman presents to the emergency department with worsening shortness of breath. He has a past medical history of oesophageal cancer which was treated six months ago with an oesophagectomy and postoperative chemotherapy and radiotherapy. He has been in remission for three months. His shortness of breath has progressed over one week and has been associated with distension of his abdomen. On examination, there is dullness to percussion of both lung bases with reduced air entry as well. His abdomen is tense with shifting dullness, but not very tender. His skin is warm and well perfused. His legs are not swollen and there is no tenderness or pitting oedema. A chest X-ray demonstrates small bilateral pleural effusions.

Which treatment is most appropriate to relieve his shortness of breath?

A. Unilateral chest drain
B. Bilateral chest drains
C. IV furosemide
D. Abdominal paracentesis
E. Treatment dose low molecular weight heparin

D. Abdominal paracentesis

The correct answer is abdominal paracentesis. This is a patient with an aggressive type of malignancy presenting with ascites and pleural effusions. Both of these can cause shortness of breath as tense ascites will make it difficult for the diaphragm to properly ventilate the lungs. Since the ascites is tense and the pleural effusions are small paracentesis is more likely to be helpful than a chest drain. The most likely cause is malignant ascites and effusions as well. Low molecular weight heparin is a treatment option for pulmonary embolism, but there is little evidence for this as a cause.

Question 144 of 235

A 54-year-old gentleman is reviewed on the ward two hours following a liver biopsy. He was admitted three days ago with jaundice and diagnosed with acute hepatitis. He has a background of hypertension, anxiety and cholecystectomy. He has minimal alcohol intake and an acute liver screen has found no obvious cause for his hepatitis and therefore he underwent the biopsy. On review, the patient is complaining of severe right upper quadrant pain and feeling generally unwell. The nurse has noted dark stool which he thinks is consistent with melaena. On examination his blood pressure is 94/56 mmHg, heart rate is 113/min, oxygenation is 94% and respiratory rate is 26/min. On examination he is jaundiced, has a tender right upper quadrant and a PR examination reveals melaena.

What is the most likely cause of his deterioration?

A. Duodenal rupture
B. Haemobilia
C. Pneumothorax
D. Variceal bleed
E. Peptic ulcer Next question

B. Haemobilia

The correct answer is haemobilia. This is a patient who develops right upper quadrant pain and melaena following liver biopsy. The most likely explanation is that the biopsy needle has hit the splanchnic vein and this has resulted in bleeding into the biliary tree.

Haemobilia

A condition in which there is bleeding into the biliary tree following connection between splanchnic circulation and either the intrahepatic or extrahepatic biliary system. Characterized by right upper quadrant pain, evidence of upper GI bleeding and jaundice.

Question 145 of 235

A 45-year-old woman is reviewed in rheumatology clinic. She has rheumatoid arthritis and has been on methotrexate for many years. Over the past 6 months her LFTs have become abnormal.

Bilirubin	13 µmol/l
ALP	179 u/l
ALT	63 u/l
γGT	101 u/l
Albumin	31 g/l

Which blood test will help determine the underlying pathology?

A. Ferritin
B. Anti-LKM antibody
C. Anti-smooth muscle antibody
D. Anti-mitochondrial antibody
E. Alpha fetoprotein

D. Anti-mitochondrial antibody

Primary biliary cholangitis - the **M** rule

Ig**M**

anti-**M**itochondrial antibodies, **M**2 subtype

Middle aged females

Primary biliary cirrhosis (PBC) is a chronic cholestatic, autoimmune liver disease . There is progressive inflammatory destruction of the intrahepatic bile ducts. It is more common in middleaged females. Autoimmune diseases such as Sjogrens syndrome, systemic sclerosis, autoimmune thyroiditis and rheumatoid arthritis, are frequently found to co-exist with PBC. Diagnosis is aided by autoantibody detection (anti-mitochondrial antibodies).

The blood tests suggest a more cholestatic picture, which is in keeping with PBC. She also has a past medical history (rheumatoid arthritis) that would suggest concomitant PBC. Her blood tests are not in keeping with haemochromatosis, which would affect the ALT more than the ALP. She also has no risk factors for developing HCC and, that often doesn't affect liver function tests until advanced disease.

Question 146 of 235

A 63-year-old man is referred for an OGD after a 5 month history of epigastric pain after eating. He is also suffering from significant reflux and dyspepsia. His OGD shows 2 duodenal ulcers, and a CLO test taken at the time is positive. He also suffers from hypertension and type 2 diabetes, which are controlled with amlodipine and metformin. He is allergic to penicillin.

What is the current gold standard treatment for H. pylorieradication for this patient?

A. Pantoprazole, metronidazole and levofloxacin
B. Amoxicillin, clarithromycin and omeprazole

C. Omeprazole, clarithromycin and metronidazole
D. Bismuth, tetracycline, metronidazole, omeprazole
E. Clarithromycin, levofloxacin and lansoprazole

C. Omeprazole, clarithromycin and metronidazole

Helicobacter pylori (*H. pylori*) is a gram positive bacteria which is commonly found in the stomach lining. Infection has been associated with gastritis, gastric ulceration and duodenitis. At OGD a CLO (*Campylobacter*-Like Organism) test is performed to detect *H. pylori* using a biopsy sample of gastric mucosa, and a reagent that acts as a pH indicator.

If *H. pylori* infection is confirmed then eradication is needed to prevent further damage and ulceration of the gastric and duodenal mucosa. The standard first line treatment for someone who has not received eradication therapy before is one week of 'triple therapy' with a PPI, amoxicillin and clarithromycin. If a patient is allergic to penicillin then metronidazole is a good substitute. The other options listed are sometimes used as second and third line options if previous treatment has failed.

Question 147 of 235

A 62-year-old male presents with a 5-month history of epigastric discomfort and two episodes of black tarry stools in the past 4 weeks. Over the past year, he reports a change in his bowel habit of almost predominantly loose bowel motions with no mucous or blood. He has no past medical history but acknowledges that he does not lead the healthiest lifestyle: he is an active smoker with a 50 pack year history and drinks at least 40 units of alcohol per week. However, he has been actively trying to improve a sedentary lifestyle as a lorry driver by using the gym twice a month, reporting 1.5 stones weight loss over the past 4 months. He had been started on increasing doses of omeprazole by his GP with no improvements in his symptoms. His mother died of pancreas adenocarcinoma aged 55. On examination, his abdomen is soft and non-tender with no organomegaly. Abdominal XR was unremarkable. Stool culture revealed no organisms on microscopy. An oesophageal-gastric endoscopy demonstrated multiple gastric and duodenal ulcers, biopsies of which were H Pylori negative. His blood tests are as follows:

Hb	128 g/l
Platelets	299 * 10 /l9
WBC	9.4 * 10 /l9
Na +	139 mmol/l
K +	4.5 mmol/l
Urea	7.9 mmol/l
Creatinine	60 µmol/l
Bilirubin	22 µmol/l
ALP	30 u/l
ALT	120 u/l
γGT	82 u/l
Adj calcium	2.22 mmol/l
Phos	1.31 mmol/l
Random blood glucose	7.1 mmol/l

Which of the following tests would make a definitive diagnosis?

A. CT chest/abdomen/pelvis with contrast
B. Faecal elastase
C. Serum gastrin and secretin stimulation
D. Endoscopic retrograde cholangiopancreatography (ERCP)
E. Magnetic resonance cholangiopancreatography (MRCP)

C. Serum gastrin and secretin stimulation on

A middle-aged male with evidence of upper GI tract bleeding, multiple gastric and duodenal ulcers, weight loss and diarrhoea with no history of NSAID use, family history of multiple endocrine neoplasia syndrome 1 (MEN1) and refractory to proton pump inhibitor treatment should make you suspicious of Zollinger-Ellison syndrome, characterised by a gastrin-secreting tumour leading to peptic ulceration and excess stomach acid secretion. The family history of pancreatic adenocarcinoma is a red herring; chronic pancreatitis, where faecal elastase helps to demonstrate malabsorption, may cause chronic diarrhoea but does not explain widespread peptic ulcerations. MRCP can demonstrate structural abnormalities such as strictures while ERCP allows therapeutic interventions and biopsies within the biliary system.

Diagnosis of Zollinger-Ellison syndrome is made by raised levels of gastrin. Secretin stimulation test differentiates between ZES and other causes of raised gastrin: secretin increases gastrin secretion in gastrinomas but inhibits gastrin secretion in gastric G cells. CT chest/abdomen/pelvis with contrast may be useful in locating the gastrinoma but does not make the initial diagnosis.

Question 148 of 235

A 53-year-old man presents with recurrent episodes of dysphagia to solids and fluids, resolving completely after 2 weeks. This is his third episode in 4 months. He reports weight loss of over two stones since the problem started and is fearful of further oral intake in case he vomits up any food he tries to ingest. He denies haematemesis or melaena. His past medical history includes angina, for which he currently takes minimal doses of GTN. He stopped taking isosorbide mononitrate prescribed to him by his cardiologist as it caused unbearable headaches. His mother died of pancreatic cancer and his uncle recently had an anterior resection for sigmoid colonic carcinoma. His GP initially improved his swallowing symptoms with nifedipine but they now have no effect. An OGD was unable to pass an obstruction at the proximal oesophagus. A barium swallow demonstrated a 'corkscrew appearance'.

What is the most appropriate next step in management?

A. Change ISMN to diltiazem
B. Oral imipramine
C. Percutaneous endoscopic gastrotomy (PEG) tube insertion
D. Sildenafil
E. Widespread pneumatic dilation

E. Widespread pneumatic dilation

The patient presents mainly with symptomatic dysphagia with barium swallow appearance suggestive of diffuse oesophageal spasm (DES). He appears intolerant of long-acting nitrates. The management of DES is controversial beyond this point but it is generally accepted that calcium channel blockers are optimal for those presenting primarily with chest pain, dysphagia resistant to pharmacological therapies require more invasive or surgical treatments. Endoscopic Botox is beneficial but may require multi-

level injections and repeat treatments. Widespread pneumatic dilation, though appearing drastic, is the most appropriate management for this patient, who reports significant weight loss due to severe dysphagic symptoms.

Question 149 of 235

A 70-year-old male presents with 3 days of vomiting and profuse watery diarrhoea, up to 6 times a day, with abdominal pain and a fever. He denies any haematemesis or melaena. He returned from a holiday to India 6 weeks ago and also visited his niece in the Lake District, where he had a barbecue about a week prior. He has minimal medical history except a 'cough and cold' treated by his GP with 3 days of oral augmentin last week. His past medical history includes hypertension, gastric reflux and type 2 diabetes mellitus, for which he takes metformin, ramipril and lansoprazole. On examination, abdomen generally tender and distended, resonant to percussion. Bowel sounds are absent. Stool cultures were initially sent three days ago but the results are still awaited, the laboratory reports that the sample has been lost. A flexible sigmoidoscopy was performed, with the report stating yellow membranes in an inflamed sigmoid colon.

What is the most likely diagnosis?

A. Bacillus cereus infection from undercooked barbecue meats
B. Giardiasis
C. Clostridium difficile infection
D. Diverticulitis
E. E Coli 0157 gastroenteritis

C. Clostridium difficile infection

Fever, abdominal pain, diarrhoea in the context of recent antibiotic use and lansoprazole should raise suspicion for Clostridium difficile infection. The colonoscopic description of yellow membranes is suggestive of pseudomembranous colitis as a result of exudates and cell debris from C diff infection. Bacillus cereus usually occurs within 24 hours, E Coli within 72 hours (with bloody stool) and giardiasis within 3 weeks. Diverticulitis would be seen on flexible sigmoidoscopy.

Question 150 of 235

A 54-year-old lady with known Sjogren's syndrome presents to her GP with lethargy. There are no abnormal findings on physical examination. The patient takes no regular medication apart from artificial tears. She drinks 10 units of alcohol a week and doesn't smoke. Her GP refers her to your clinic as her routine bloods come back showing the following:

Hb	140 g/l	Na +	134 mmol/l	Bilirubin	6 µmol/l
Platelets	225 * 10 /l 9	K +	3.7 mmol/l	ALP	650 u/l
WBC	6.7 * 10 /l 9	Urea	4.2 mmol/l	ALT	36 u/l
Neuts	4 * 10 /l 9	Creatinine	90 µmol/l		

What is the most likely diagnosis?

A. Gall stones
B. Primary sclerosing cholangitis
C. Sarcoma
D. Paget's disease
E. Primary biliary cirrhosis

E. Primary biliary cirrhosis

Sjogren's syndrome is associated with primary biliary cirrhosis (PBC). All the options could lead to a raised alkaline phosphatase but PBC has the greatest association with Sjogren's syndrome.

Question 1 of 86

A 40-year-old man presented for the first time in a GP surgery. He had recurrent abdominal pain which improved with defecation, and mushy stools up to three times a day for the last 4 months. He denied weight loss, rectal bleeding, or a family history of colorectal cancer. Stool microscopy, culture and sensitivity were negative for ova, cysts and parasites.

Which of the following is the next most appropriate investigation to be done?

A. Abdominal ultrasound
B. Colonoscopy
C. Flexible sigmoidoscopy
D. Abdominal X-ray
E. Tissue transglutaminase

E. Tissue transglutaminase

Full blood count, ESR, CRP, antibody testing for coeliac disease - endomysial antibodies or tissue transglutaminase are first-line investigations which are expected to be normal in patients with suspected irritable bowel syndrome.

Irritable bowel syndrome is common; approximately 5-11% of the population are affected. Chronic abdominal pain, bloating, and altered bowel habit are the main characteristics. Irritable bowel syndrome cannot be cured, and treatment should focus on the relief of symptoms.

If the patient meets the diagnostic criteria for irritable bowel syndrome, the following tests should be undertaken to exclude other diagnoses (These blood tests would be expected to be normal in irritable bowel syndrome).

- Full blood count
- Erythrocyte sedimentation rate
- C-reactive protein
- Antibody testing for coeliac disease - endomysial antibodies or tissue transglutaminase.

Question 2 of 86

A 17-year-old female presents having noticed yellowness to her skin in recent weeks and symptoms of myalgia and fatigue. She is normally fit and well, drinks ten units of alcohol per week and does not report any high risk behaviour. On examination she is clinically jaundiced and has a tender hepatomegaly. Blood tests taken in the acute medical unit are as follows:

Haemoglobin	123 g/l
White cell count	10.0 *10^9/l
Neutrophils	7.0 *10^9/l
Platelets	358 *10^9/l
Bilirubin	85 µmol/l
Aspartate transaminase (AST)	140 u/l
Alanine aminotransferase (ALT)	180 u/l
Alkaline phosphatase	120u/l
Albumin	36 g/l
Anti-nuclear antibody (ANA)	positive
Anti-Liver Kidney Microsomal-1 antibody (anti-LKM-1)	positive
Anti-mitochondrial antibody	negative
Hepatitis B surface antigen (HBsAg)	negative
Hepatitis B surface antibody (anti-HBs)	positive
Hepatitis B core antibody (anti-HBc)	negative

What is the most likely diagnosis?

A. Primary biliary cirrhosis
B. Primary sclerosing cholangitis
C. Autoimmune hepatitis type I
D. Autoimmune hepatitis type II
E. Chronic hepatitis B

D. Autoimmune hepatitis type II

Autoimmune hepatitis is a chronic disease of unclear cause which leads to progressive fibrosis and cirrhosis. It presents non-specifically and there are no pathognomonic features but typically there are raised transaminases with a normal or minimally raised alkaline phosphatase. Diagnosis rests on autoantibodies and liver biopsy.

It is sub-divided into type I and type II depending upon the antibodies present:

- Type I Anti-smooth muscle antibodies or anti-nuclear antibodies; this constitutes 75% of patients
- Type II Anti-LKM-1 or anti-liver cytosolic-1 antibodies

The AST/ALT ratio can give clues to the cause of hepatitis. If the ratio is greater than 2 it is suggestive of alcoholic liver disease whereas less than 1 suggests non-alcoholic liver disease.

Her hepatitis B serology results are indicative of immunity.

Question 3 of 86

A 45-year-old man with know alcoholic cirrhosis is admitted under the acute medical take with upper abdominal pain. This pain occurs mainly on eating his meals. He has not noticed any change in bowel habits, and denies any dark sticky or particularly offensive smelling stools. The patient is booked for an oesophagogastroduodenoscopy (OGD), the results of which are shown below:

Oesophagus: Grade 1 varices with no evidence of recent or active bleeding Stomach: Moderate non-haemorrhagic gastritis

Duodenum: Nil of note

He is not previously known to have oesophageal varices.

What is the most appropriate management with regards to this gentleman's oesophageal varices?

A. Variceal band ligation
B. Propranolol 40mg BD and titrate up as tolerated
C. Repeat OGD in 1 year
D. Sclerotherapy
E. Repeat OGD in 2-3 years

C. Repeat OGD in 1 year

In patients with known cirrhosis the management of varices is as follows.

No varices	Rescope 2-3 years
Grade 1 varices	Rescope 1 year
Grade 2 or 3 varices or signs of bleeding	Non-cardio selective beta blocker

The history given by the patient is most consistent with gastritis, and the lack of melaena or evidence of bleeding on OGD means that this patient has non-bleeding grade 1 varices, and thus needs a rescope in 1 year.

Question 4 of 86

A 23-year-old man is seen two weeks after an emergency open appendicectomy. He presents with increasing right upper quadrant pain which does not radiate. This has continued for 5 days becoming worse and is constant in nature. He feels unwell and has been in bed today shaking with his bed sheets soaked in sweat. His bowel habits have returned to a normal consistency but he has vomited twice with the contents of his food. His appetite is poor.

Asides from the recent admission, he takes citalopram for depression and is otherwise well. He drinks 14 units of alcohol a week and does not smoke.

On examination, he appears mildly jaundiced with tenderness and guarding in the right upper quadrant. He is Murphy's test negative but a palpable 2-3cm mass can be felt on inspiration under the right costal margin. There is no rigidity and his abdomen is soft. His observations show respiratory rate 25/min, temperature 37.6ºC, blood pressure 90/62mmHg, heart rate 120/min. His chest is clear and there are normal heart sounds. He is warm with a bounding pulse.

Hb	130 g/l	Na +	139 mmol/l
Platelets	278 * 10 /l 9	K +	4.2 mmol/l
WBC	18.0 * 10 /l 9	Urea	5.4 mmol/l
Neuts	15.6 * 10 /l 9	Creatinine	86 µmol/l
Lymphs	1.3 * 10 /l 9	CRP	230 mg/l
Eosin	0.5 * 10 /l9		

Bilirubin	56 µmol/l
ALP	340 u/l
ALT	120 u/l
Albumin	27 g/l

Chest X-ray	no consolidation or pneumoperitoneum seen
Abdominal X-ray	dilated ascending colon filled with gas

What is the likely diagnosis?

A. Liver abscess

B. Pericolic abscess
C. Ascending cholangitis
D. Cholecystitis
E. Crohn's disease

A. Liver abscess

Raised LFTs + recent appendicitis -> liver abscess

This gentleman has presented with right upper quadrant pain with localised tenderness. Crohn's disease is unlikely as there is no bowel change and it does not explain the liver function test (LFT) changes. Cholecystitis does present with any jaundice or LFT derangement. Of liver abscess and cholangitis, the recent surgery and the palpable mass point towards an abscess as the most likely diagnosis. A pericolic abscess is possible but this presentation is clearly linked to the liver.

Question 5 of 86

A 40-year-old woman with a background of hypothyroidism and Sjogren's Syndrome presents to the acute medical take with worsening jaundice and pruritus. She is otherwise well and reports the symptoms increasing over the past few months. She has been nauseous and struggled to manage any oral fluids in the past three days. She has soft loose stools that are pale.

On examination, she is markedly jaundiced. Her abdomen is soft with no masses and bowel sounds are present. She is haemodynamically stable and there are no bruises or bleeding. She has a mildly distended abdomen.

Bilirubin	89 µmol/l	ALT	150 u/l
ALP	450 u/l	gamma#GT	270 u/l
Albumin	27 g/l	Platelets	300 *10^9/l
PT	17.7s	APTT	39.9s

Hepatitis A IgM/IgG	negative
HBsAg	negative
Hepatitis C DNA PCR	negative
Liver ultrasound	dilated biliary ducts seen, no focal lesion, normal echogenicity, small volume ascites around the liver

What contraindication to biopsy is present?

A. Coagulopathy
B. Ascites
C. Cholestasis
D. Thrombocytopenia
E. Diagnosis uncertain

C. Cholestasis

Biliary obstruction in cholestasis is a contraindication to liver biopsy

Liver biopsy has many contraindications. Platelets should be over 80 and PT/APTT should not be grossly deranged, they are only mildly so here. These are required to ensure there is a low risk of bleeding. Ascites can cause the liver and abdominal wall to not appose and heal properly but here the volume is small and this is unlikely to be significant. Biliary obstruction or cholestasis is important as there can be bile leak following biopsy and a secondary peritonitis. At this point, the obstruction may need relieving and the cause determined before the biopsy is carried out. Uncertainty of diagnosis does not contraindicate but a cystic or malignant lesion should be excluded as biopsy can seed the lesion across the abdomen causing sepsis or dissemination respectively.

Question 6 of 86

A 60-year-old man is admitted with severe upper abdominal pain, nausea and dizziness lasting for the last day. On further questioning he admits to experiencing intermittent mild upper abdominal pain for the last month, typically after meals. His past medical history includes hypertension, type 2 diabetes, osteoarthritis and myocardial infarction 5 years ago for which he had a stent placed. His current medications include aspirin, ramipril, amlodipine, metformin, naproxen and paracetamol.

Whilst in the department he develops diarrhoea and examination of the stool shows melaena.

His blood pressure is 110/55 mmHg and heart rate is 95 beats per minute. On examination, he is tender in the epigastrium with no peritonism and normal bowel sounds. Examination of other systems is normal.

Blood results:

Hb	95 g/l	Na +	145 mmol/l
Platelets	200 * 10 /l 9	K +	4.5 mmol/l
WBC	8 * 10 /l 9	Urea	12 mmol/l
Neuts	3 * 10 /l 9	Creatinine	102 µmol/l

He is treated given intravenous fluids and analgesia. All his regular analgesia aside from paracetamol is withheld. and taken to endoscopy later that day. A 1cm ulcer is seen in the gastric antrum with an adherent clot. This is clipped and injected with adrenalin. He recovers well from sedation and on return to the ward his blood pressure is 135/70 mmHg and heart rate 80 beats per minute. He has no further diarrhoea or vomiting and repeat haemoglobin is 121 g/l.

On discharge, what advice should he be given regarding his non-steroidal anti-inflammatory drugs?

A. Continue both aspirin and naproxen and add a proton pump inhibitor
B. Stop naproxen and aspirin. Start a proton pump inhibitor.
C. Stop naproxen, change aspirin to clopidogrel and add a proton pump inhibitor
D. Stop naproxen, change aspirin to dalteparin and add a proton pump inhibitor
E. Stop naproxen, continue aspirin and add a proton pump inhibitor

E. Stop naproxen, continue aspirin and add a proton pump inhibitor

steroidal anti-inflammatory drugs (NSAIDS) aspirin and naproxen, NICE guidelines recommend continuing aspirin for secondary prevention of vascular events (as in the gentleman with previous myocardial infarction) providing haemostasis has been achieved. All other NSAIDS should be discontinued and so naproxen should be stopped.

Proton pump inhibitors are recommended for all those with non-variceal bleeding and stigmata of recent haemorrhage at endoscopy.

National Institute for Health and Clinical Excellence. Acute upper gastrointestinal bleeding. (2012) NICE guideline CG141.

Question 7 of 86

A 55-year-old man is seen in the gastroenterology clinic for follow-up of his alcoholic liver disease. He states he has not had an alcoholic drink for many months but following some bad news at work, he went on a binge two days ago and is now feeling very unwell.

On examination he appears mildly jaundiced and clammy. His heart rate is 119 beats per minute and his blood pressure is 105/66 mmHg. He is afebrile. Cardiovascular and respiratory examinations are normal. Examination of the abdomen reveals a tender 2cm liver edge and shifting dullness.

His blood tests are as follows:

Hb	105 g/l	Na +	132 mmol/l	Bilirubin	33 µmol/l
Platelets	167 * 10 /l 9	K +	4.4 mmol/l	ALP	220 u/l
WBC	10.5 * 10 /l 9	Urea	7 mmol/l	AST	137 u/l
Neuts	5.7 * 10 /l 9	Creatinine	105 µmol/l	γGT	505 u/l
INR	1.9	CRP	47 mg/l	Albumin	29 g/l

He is diagnosed with alcoholic hepatitis and admitted to hospital. Which treatment is most likely to improve his survival?

A. Antibiotics
B. Chlordiazepoxide
C. Human albumin solution
D. Pentoxyphylline
E. Prednisolone

E. Prednisolone

This gentleman has no evidence of infection and so antibiotics are not indicated at this point.

Although this gentleman claims to have been abstinent prior to his recent alcohol binge, he should be monitored for signs of withdrawal and given chlordiazepoxide if indicated.

Human albumin solution is useful for rehydration in patients with liver disease, particularly in cases of hepatorenal syndrome (HRS). However, this patient does not meet the criteria for HRS and should be rehydrated with crystalloid in the first instance.

Question 8 of 86

A 63-year-old man is seen in the Emergency Department with fevers and abdominal pain. He has a history of alcoholic liver disease with cirrhosis, hypertension and oesophageal varices. He has noticed increasing abdominal distension over the last week, associated with fatigue and malaise. In the last day he has developed generalised abdominal pain and has been feeling hot and cold. His temperature at triage was 38.2 ºC. He feels nauseated but has not vomited. He has not opened his bowels in 2 days but is passing flatus. He has had no dysuria, cough or shortness of breath.

On examination he is tachycardic at 120 beats per minute and has a blood pressure of 111/78 mmHg. He looks dehydrated and is jaundiced. His chest is clear and heart sounds are normal. He has a distended abdomen with generalised tenderness but no guarding and normal bowel sounds. There is shifting dullness and pitting oedema to the mid-shin.

His chest x-ray demonstrates clear lung fields but poor inspiration due to ascites.

His ECG shows sinus tachycardia. His urine dip shows 1+ of protein. His bloods are as follows:

Hb	110 g/l	Na +	146 mmol/l	Bilirubin	37 µmol/l
Platelets	205 * 10 /l 9	K +	4.6 mmol/l	ALP	272 u/l
WBC	17.3 * 10 /l 9	Urea	8.1 mmol/l	ALT	62 u/l
Neuts	14.6 * 10 /l 9	Creatinine	141 µmol/l	γGT	591 u/l
Lymphs	2.1 * 10 /l 9	CRP	150 mg/l	Albumin	29 g/l

He is started on intravenous normal saline and broad spectrum antibiotics. Blood cultures are sent. An ascitic tap is performed which demonstrates cloudy fluid with a neutrophil count of 300 per mm³. This is sent for gram stain and culture. He is diagnosed with spontaneous bacterial peritonitis and resultant acute kidney injury and antibiotics are switched to cefotaxime.

Which further management has been shown to reduced mortality in this condition?

A. Dialysis
B. Human albumin solution
C. Repeat ascitic tap in 48 hours
D. Rifaximin
E. Steroids

B. Human albumin solution

This gentleman has sepsis secondary to spontaneous bacterial peritonitis (SBP). Use of human albumin solution to rehydrate patients with cirrhosis and SBP has been shown to reduce mortality.

Although this gentleman has an acute kidney injury, he is not yet at the threshold for dialysis and should be rehydrated.

All patients with SBP should have repeat ascitic tap at 48 hours to ensure neutrophil count of fluid is improving on antibiotics. However, this has not been proven to reduce mortality.

Rifaximin is an antibiotic licensed for use in patients with alcoholic cirrhosis with previous hepatic encephalopathy. It can be used prophylactically to prevent ammonia-releasing bacteria in the gut precipitating further episodes, However, it is not used in SBP.

Steroids may be used in acute alcoholic liver failure to improve outcome but are not used in SBP.

Question 9 of 86

A 38-year-old ex-intravenous drug user attends the GUM clinic for review. He describes a history of needle sharing in the past and is no longer abusing intravenous drugs. He also describes unprotected sexual intercourse with a number of partners who he no longer is in touch with, most recently 3 days prior to attending clinic. He has come to clinic after hearing a recent awareness campaign around the dangers of undiagnosed hepatitis C. He is currently well and cannot recall ever being jaundiced.

He has a wide range of investigations as detailed below.

Hepatitis B core antibody (HBcAb) - positive Hepatitis C antibodies - positive

HIV antibody negative

p24 antigen negative

Further testing was carried out based on the results above

Hepatitis B surface antigen (HbsAg) negative Hepatitis B surface antibody (HbsAb) positive Hepatitis B envelope antigen (HbeAg) negative Hepatitis C RNA - negative

What is the most accurate profile of the patient detailed above?

A. HIV negative, Hepatitis B chronic carrier, Hepatitis C chronic infection
B. HIV negative, Hepatitis B chronic carrier, Hepatitis C active infection
C. HIV negative, Hepatitis B active infection, Hepatitis C spontaneously cleared
D. HIV negative, Hepatitis B previous infection, Hepatitis C spontaneously cleared
E. HIV negative, Hepatitis B previous infection, Hepatitis C chronic infection

D. HIV negative, Hepatitis B previous infection, Hepatitis C spontaneously cleared

In this case, the patient has been exposed and cleared both hepatitis B and C infections. In this instance, he is not a carrier and therefore not infectious to others. During this visit to the GUM clinic, he should also be tested for infections such as syphilis, chlamydia and gonorrhoea.

HIV testing should be done with HIV antibody and p24 antigen testing. It is not appropriate to use HIV viral loads as a screening test. The antibody and p24 tests are 4th generation tests that have helped reduce the time from exposure to being able to prove infection from one month (with previous 3rd generation tests) to two weeks.

The understanding of both hepatitis B and C serology is important as both are commonly ordered tests, both in pregnancy screening, screening for chemotherapy, or for patients with abnormal liver function tests. The hepatitis C serology is suggestive of exposure to the virus and subsequent spontaneous clearing. However, it must be noted that Hep C Antibody testing can throw up false positives (up to 10%). Where Hep C antibodies are positive and RNA viral load is undetected it may be appropriate to repeat the antibody test using a different antibody assay. The false positive rate is thought to be secondary to other similar antibodies in the circulation. If the patient has Hep C antibodies and is Hep C RNA positive for more than 2 months they should be considered for treatment.

Question 10 of 86

A 52-year-old man on allopurinol for gout takes 20 mg of prednisolone per day for Crohn's disease, which was diagnosed 12 months ago. He has no other comorbidities of note. At present, he has a normal bowel habit with no loose stools or abdominal pain. He has been on the 20 mg dose of steroid for 12 months and is gaining significant amounts of weight. Each time you have reduced his dose to 15 mg, he has developed diarrhoea up to four times per day and required hospital admission for intravenous steroid therapy. This has happened three times in total. You wish to add in a steroid-sparing agent to enable the taper

Which agent should be avoided owing to significant interaction with allopurinol?

A. Mesalazine
B. Infliximab
C. Methotrexate
D. Azathioprine
E. Adalimumab

D. Azathioprine

Allopurinol enhances the effects and increases the toxicity of azathioprine. According to the British National Formulary (BNF), one should reduce the dose of azathioprine to one-quarter of usual dose, but in practice, I would recommend talking to your consultant before combining these two medications. The interaction is significant. Sometimes, specialists do actually combine the two purposefully in order to be able to use a lower dose of azathioprine...but this is again a very specialist area. If you see these two drugs together, alarm bells should ring and further enquiries should be made.

Incidentally, remember to always check serum thiopurine methyltransferase (TPMT) levels prior to commencing azathioprine. Azathioprine is a prodrug of mercaptopurine, a substance that is subsequently metabolised by several alternative pathways, one of which involves the enzyme TPMT. Because of our natural genetic variability, some healthy people will just naturally have a deficiency of TPMT (around three in a thousand). Such people are at a significant risk of developing severe, potentially life-threatening, bone marrow toxicity when treated with conventional doses of azathioprine.

Question 11 of 86

A 55-year-old man presents to the emergency department with a 2-week history of feeling generally unwell. He is a known alcoholic and reports consuming around 1L whiskey per day for the last two months. On further questioning, he reports progressive shortness of breath with some dizziness and palpitations but denies any haematemesis or melaena. He has had no chest or abdominal pain. He states that he takes no regular medications and aside from alcohol dependency has no other medical problems.

Examination reveals scleral icterus but no asterixis and unremarkable respiratory and cardiovascular systems with some right upper quadrant tenderness on abdominal assessment.

Bloods are performed and demonstrate:

Hb	80 g/L	Male: (135-180)Female: (115 - 160)
Platelets	112 * 10 /L 9	(150 - 400)
WBC	10.1 * 10 /L 9	(4.0 - 11.0)
Reticulocytes	5%	(0.5 - 2.5%)
LDH	500 u/L	(140 - 280)
Na +	140 mmol/L	(135 - 145)
K +	5.0 mmol/L	(3.5 - 5.0)
Urea	1.5 mmol/L	(2.0 - 7.0)
Creatinine	70 µmol/L	(55 - 120)
Bilirubin	35 µmol/L	(3 - 17)
ALP	150 u/L	(30 - 100)
ALT	50 u/L	(3 - 40)
Triglycerides	5.6g/dL	(<1.7)
Blood film	polychromasia, macrocytosis, teardrop cells, spur cells and s	

An ultrasound abdomen shows steatohepatitis and splenomegaly.

Endoscopic examination of the upper and lower gastrointestinal systems shows no signs of active bleeding with no varices.

What is the unifying diagnosis?

A. Alcoholic hepatitis
B. Zieve's syndrome
C. Cholecystitis
D. Decompensated alcoholic liver disease
E. Myelodysplastic syndrome in chronic liver disease Next question

B. Zieve's syndrome

Zieve syndrome is a triad of symptoms: haemolytic anemia, cholestatic jaundice, and transient hyperlipidemia

This patient has Zieve's syndrome - a rare condition characterized by haemolytic anaemia, cholestatic jaundice, and hyperlipidaemia.

Cholecystitis should remain in the differential for patients with Zieve's syndrome and there are cases of patients undergoing assessment for cholecystectomy who are subsequently diagnosed with Zieve syndrome, however, it would not explain this patient's hyperlipidaemia or haemolytic anaemia.

This patient may have chronic liver disease, however, the presence of haemolytic anaemia and hyperlipidaemia would not be explained by simple decompensated liver disease.

Myelodysplasia would be in the differential, but would not explain the hyperlipidaemia.

Question 12 of 86

A 35-year-old man attends Gastroenterology clinic with some new and troubling symptoms. Seven years previously he had undergone total colectomy after presenting with an acute severe episode of ulcerative colitis that had failed to respond to intravenous glucocorticoids and biological therapy. Seven months after colectomy, the patient underwent restorative proctocolectomy with ileal pouch-anal anastomosis. The patient had made a good recovery from his surgeries and had experienced minimal gastrointestinal problems in the following years. Medical history was otherwise unremarkable and the patient took no regular medications.

However, over the past two months the patient had noticed an increase in stool frequency (up to 7 times per day), increasing urgency of defecation and on several occasions some seepage of stool over night. He denied any history of fevers, weight loss or bloody stool. The patient denied having contact with any individuals with gastrointestinal illnesses and had no recent travel history.

Abdominal examination demonstrated a soft and non-tender abdomen with active bowel sounds. Basic investigations arranged by the patients General Practitioner are listed below.

Stool microscopy, culture and sensitivity: no organisms seen; no growth Clostridium difficile toxin: not detected

Haemoglobin	138 g / dL
White cell count	9.9 * 10 /l9
Neutrophils	7.8 * 10 /l9
Platelets	336 * 10 /l9
Urea	5.6 mmol / L
Creatinine	87 micromol / L
Sodium	140 mmol / L
Potassium	5.0 mmol / L
Erythrocyte sedimentation rate	40 ml / h

What is the appropriate first line management for the patients condition?

A. Metronidazole
B. Vancomycin
C. Prednisolone
D. Infliximab
E. Pouch excision with permanent ileostomy

A. Metronidazole Next question

Up to 30 % of patients develop pouchitis following ileal pouch-anal anastomosis after total colectomy in ulcerative colitis. This presents with increased stool frequency, urgency, incontinence and nocturnal seepage.

First line treatment is with antibiotics such as metronidazole or ciprofloxacin. Probiotics are used in some cases. In 5 % of cases, pouchitis can become chronic, ultimately leading to pouch failure and requiring pouch excision.

Prednisolone and infliximab are important treatments of ulcerative colitis but are not used in the treatment of pouchitis.

Question 13 of 86

A 35-year-old woman with known Crohns disease is reviewed in gastroenterology clinic. Following her initial diagnosis 2 years previously she has been treated with azathioprine as a immunomodulating agent. This medication has been well tolerated and the patient reported no significant side effects. However, the patients disease had not been well controlled over the previous 6 months with frequent exacerbations of bloody diarrhoea and abdominal pain. Each flare had been treated with a tapered course of corticosteroids, however the patient had been unable to fully wean herself off steroids due to recurrent symptoms.

A severe recent exacerbation had involved the patient suffering significant nausea and vomiting necessitating admission to hospital. The report from a gastroscopy performed at this time is given below.

Following discussion with the patient it was agreed that additional therapy was needed in order to maintain disease remission. The patient felt that the azathioprine she had been taking was effective and was keen to continue this medication in conjunction with infliximab.

Gastroscopy: normal oesophagus and lower oesophageal sphincter; small areas of patchy inflammation affecting antrum; further multiple areas of inflammation throughout first part of duodenum

What additional risk of combination immunomodulator and biological therapy compared to biological therapy alone is it necessary to discuss with the patient?

A. Reactivation of latent tuberculosis
B. Aplastic anaemia
C. Increased risk of non-melanoma skin cancer
D. Congestive cardiac failure
E. Hypersensitivity reaction

C. Increased risk of non-melanoma skin cancer

Anti-tumour necrosis factor agents used in combination with immunomodulating agents are more effective at maintaining steroid-free clinical remission than when used as mono-therapy (56.8 % vs 30 % $P < 0.001$).

However, compared with mono-therapy, combination therapy carries an increased risk of nonmelanoma skin cancer (standardised incidence ratio 3.46) and other cancers (standardised incidence ratio 2.82). Of particular concern are cases of hepatosplenic T-cell lymphoma reported in adolescent and young adult patients with Crohns disease treated with infliximab and azathioprine or mercaptopurine.

The other answers are all risks factors of infliximab treatment with or without concomitant immunomodulator therapy.

Question 14 of 86

A 50-year-old woman with a history of polycythaemia rubra vera develops abdominal pain and gross ascites over the past 2 months. Due to her busy work as a lawyer she mentions that her diet is mostly takeaways, however she is a lifelong teetotaler.

On examination: mild jaundice with smooth hepatomegaly. Hepatojugular reflex is negative. Abdominal wall is grossly oedematous with an associated tortuous venous pattern. There is prominent lower limb oedema.

Which of the following is the most likely diagnosis?

A. Budd-Chiari syndrome
B. Hepatic cirrhosis
C. Hepatocellular carcinoma
D. Primary sclerosing cholangitis
E. Nonalcoholic steatohepatitis Next question

A. Budd-Chiari syndrome

Budd-Chiari syndrome presents with the triad of sudden onset abdominal pain, ascites, and tender hepatomegaly

Budd-Chiari syndrome occurs when hepatic venous outflow is obstructed secondary to thrombosis of the major hepatic veins. The thrombus can extend into the inferior vena cava, leading to the abdominal wall signs and leg oedema as noted in the presentation. The condition is rare, typically occurring in the setting of a coagulopathy due to haematologic disease, which in this cause is polycythaemia rubra vera. The impression from the patient's social history may lead you towards considering nonalcoholic steatohepatitis, however this does not cause ascities.

Question 15 of 86

A 33-year-old woman is referred to the gastroenterology clinic with diarrhoea for the last 2 months. The stool is very loose, but not quite watery. There is no blood present. Otherwise, she feels fairly well, denying weight loss or abdominal pain. None of her family has diarrhoea at present.

On examination, her abdomen is soft and non-tender. She has a past medical history of anxiety, Crohn's disease with ileal resection, and hypothyroidism.

Blood tests are performed.

Hb	133 g/L	Female: (115 - 160)
Platelets	190 * 10 /L $_9$	(150 - 400)
WBC	10.1 * 10 /L $_9$	(4.0 - 11.0)
Na $_+$	141 mmol/L	(135 - 145)
K $_+$	3.9 mmol/L	(3.5 - 5.0)
Urea	6.1 mmol/L	(2.0 - 7.0)
Creatinine	71 µmol/L	(55 - 120)
CRP	3 mg/L	(< 5)

What is the most appropriate management?

A. Oral cholestyramine
B. Oral mesalazine
C. Oral metronidazole
D. Oral prednisolone
E. Rectal prednisolone

A. Oral cholestyramine

Ongoing diarrhoea in Crohn's patient post-resection with normal CRP → cholestyramine

The patient in the scenario is experiencing malabsorption following bowel resection. The presentation and blood test results don't suggest a flare of her Crohn's disease and so alternate diagnoses should be sought. Having an ileal resection predisposes patients to malabsorption syndromes, particularly bile salt malabsorption, leading to persistent diarrhoea. The treatment is with **oral cholestyramine** which is a bile acid sequestrant. This acts by binding bile salts, making them insoluble, preventing them from creating an osmotic shift of water into the large bowel and so helping to prevent diarrhoea.

Oral mesalazine is used in the treatment of Crohn's disease, such as mild flares. The normal blood test results do not suggest a flare and therefore this answer is incorrect.

Oral metronidazole is an antibiotic that can be used for some gastrointestinal infections, such as *Clostridioides difficile* or giardiasis. There is no suggestion of these infections from the history, therefore this answer is incorrect.

Prednisolone may be used in flares of Crohn's disease, however, the normal blood test results mean this diagnosis is less likely. Therefore, both **oral prednisolone** and **rectal prednisolone** are incorrect.

Question 16 of 86

A 58-year-old man presents to the emergency department after abnormal liver function tests were found by his general practitioner. This is his first presentation and other than type 2 diabetes mellitus, there is no relevant past medical history. He reports drinking roughly 16 units of alcohol per week. On examination there are no signs of respiratory distress, he is warm and well perfused, tenderness is noted in the right upper quadrant but otherwise, his abdomen is soft and nontender. He is mildly jaundiced in the sclera and his body mass index is 31kg/m².

Initial investigations showed the following:

Bilirubin	24 µmol/L	(3 - 17)
ALP	121 u/L	(30 - 100)
ALT	374 u/L	(3 - 40)
AST	136 u/L	(3 - 40)
Î³GT	71 u/L	(8 - 60)
Albumin	39 g/L	(35 - 50)

HBsAg	negative
Anti-HBs	positive
Anti-HBc	negative
Anti-HCV	negative

An abdominal ultrasound is performed and shows multiple gallstones in the gallbladder with no dilation of the biliary tree.

What is the most likely diagnosis?

A. Alcoholic liver disease
B. Biliary colic
C. Hepatitis B
D. Hepatitis C
E. Non-alcoholic fatty liver disease

E. Non-alcoholic fatty liver disease

Obesity with abnormal LFTs - ? non-alcoholic fatty liver disease

This man has presented with hepatic, rather than obstructive, liver function tests. In the context of obesity, non-alcoholic fatty liver disease is highly suspicious. This is further reinforced by the fact that this man's ALT is more markedly deranged than his AST.

Alcoholic liver disease is less likely as his drinking only slightly exceeds the recommended limit. Additionally, alcoholic liver disease is associated with a disproportionately deranged AST rather than ALT.

Biliary colic can cause pain and a raised bilirubin however the liver function tests are indicative of a hepatocellular pattern of injury rather than cholestatic (which would cause a larger rise in ALP and Î³GT). The ultrasound findings of gallstones in the gallbladder are often an incidental finding and the absence of bile duct dilatation further eliminates biliary colic as a diagnosis.

Hepatitis B is not the case here as HBsAg and anti-HBc are negative.

Hepatitis C is not the case here as anti-HCV is negative.

Question 17 of 86

A 35-year-old woman presents to hospital with a three-day history of acute abdominal pain and distension. Her past medical history includes asthma, coeliac disease and an unprovoked deep vein thrombosis in her left leg a year ago. Her medications include Symbicort 400/12 2 puffs BD and salbutamol PRN. She does not smoke and drinks approximately 12 units of alcohol a week. She works as a secretary in a legal firm. Her parents both suffer from type 2 diabetes mellitus.

On examination, her temperature is 37.7ºC, pulse rate is 110 beats per minute and blood pressure is 130/80 mmHg. There is evidence of icterus in her sclera. Inspection of her abdomen reveals gross distension. There is hepatomegaly on palpation and shifting dullness on percussion. There is marked tenderness on palpation of the right upper quadrant of her abdomen.

Her blood tests show:

Hb	100 g/l	Na +	138 mmol/l	Bilirubin	63 µmol/l
Platelets	740 * 10 /l 9	K +	5.2 mmol/l	ALP	295 u/l
WBC	14.2 * 10 /l 9	Urea	5.1 mmol/l	ALT	230 u/l
Neuts	9.5 * 10 /l 9	Creatinine	89 µmol/l	γGT	210 u/l
Lymphs	0.7 * 10 /l 9	Albumin	26 g/l		
INR	1.5	CRP	93 mg/L	Amylase	173 U/L

What is the most likely diagnosis?

A. Budd-Chiari syndrome
B. Hepatic abscess
C. Portal vein thrombosis
D. Acute pancreatitis
E. Sarcoidosis

A. Budd-Chiari syndrome

Budd-Chiari syndrome presents with the triad of sudden onset abdominal pain, ascites, and tender hepatomegaly

Next question

The most likely diagnosis, in this case, is Budd-Chiari syndrome (BCS) which is the occlusion of the hepatic veins draining the liver. Patients may present with the classical triad of abdominal pain, ascites and hepatomegaly but some patients with BCS may follow a chronic or asymptomatic course.

BCS can be caused by thrombosis of the hepatic veins (primary) or external compression causing occlusion of the hepatic veins (secondary). It is important to look for a cause for hypercoagulability in patients with primary BCS.

Treatment for BCS involves medical therapy with diuretics and anticoagulants, surgical shunt placement or a TIPSS procedure. In patients with fulminant BCS, liver transplantation should be considered.

Question 18 of 86

A 65-year-old retired woman with a history of diet-controlled diabetes and mild COPD is brought in by ambulance with 3 episodes of black stool followed by collapse. She describes a 2 day history of intermittent abdominal pain. Her regular medications are Seretide inhaler and paracetamol/ibuprofen when required for knee osteoarthritis.

On examination, she has conjunctival pallor and epigastric tenderness. Rectal examination confirms the presence of melaena.

Her observations are as follows:

- Temperature 36.4
- Respiratory 28/min
- Saturations 96% on air
- Heart rate 126 bpm
- Blood pressure 78/44 mmHg

Hb	67 g/l	Na +	140 mmol/l	Bilirubin	12 µmol/l
Platelets	88* 10 /l 9	K +	3.9 mmol/l	ALP	100 u/l
WBC	12.0* 10 /l 9	Urea	13.3 mmol/l	ALT	28 u/l
Neuts	6.0* 10 /l 9	Creatinine	63 µmol/l	Albumin	38 g/l
Lymphs	1.0* 10 /l 9	INR	1.2		
Eosin	0.3* 10 /l 9	APTT	26	Fibrinogen	4.0 g/l

What medical therapy should be urgently instituted prior to endoscopy?

A. Red blood cell transfusion
B. Red blood cell transfusion and IV omeprazole
C. Red blood cell transfusion and fresh frozen plasma
D. Red blood cell and platelet transfusion and IV omeprazole
E. Red blood cell and platelet transfusion

A. Red blood cell transfusion

This patient presents with upper GI bleeding and haemodynamic compromise. The history of abdominal pain and NSAID use are suggestive of peptic ulceration. There is nothing in the history/examination to suggest variceal bleeding.

According to NICE guidelines (updated August 2016), platelets/FFP are not indicated unless platelets<50 and INR/APTT>1.5 x normal. PPIs are not recommended prior to endoscopy. Red cell transfusion should occur according to local protocol but with Hb<7 and low BP,

Question 19 of 86

A 39-year-old female presents with subacute onset, itching, severe abdominal pain and distension. The patient is normally relatively fit and well with a past medical history of hypothyroidism and board line type 2 diabetes. Other than these she occasionally has episodes of lower back pain following long days standing at work where she is a waiter.

A list of her medications are:

- Levothyroxine 75mcg OD
- Metformin 500mg OD
- Combined oral contraceptive pill
- Paracetamol 1g QDS PRN
- Ibuprofen 200mg TDS PRN

On examination, the patient has obvious pain, has yellowing of the sclera, and abdominal examination reveals tender hepatomegaly and shifting dullness. The examination is otherwise normal.

Some of the patient's blood investigations shown below:

Patient's results	Reference ranges	
Na+	139	136-146 mmol/L
K+	4.0	3.5-5.0 mmol/L
Urea	5.0	2.5-6.4 mmol/L
Creatinine	100	53-106 µmol/L
Total Bilirubin	35	2-17 µmol/L
ALT	88	10-40 U/L
ALP	180	25-100 U/L

Which of the following investigation is most appropriate to confirm the likely diagnosis?

A. Abdominal ultrasound with doppler flow studies
B. Ascitic aspiration and fluid analysis
C. CT abdomen and pelvis
D. Coagulation blood test

A. Abdominal ultrasound with doppler flow studies

Combined oral contraceptive pill use + acute abdominal pain/distension → ?Budd-Chiari syndrome

The patient has presented with features of hepatic vein thrombosis (Budd-Chiari syndrome) with acute abdominal pain, tender hepatomegaly, ascites, and deranged LFTs. The patient has likely developed this condition secondary to the combined oral contraceptive pill use resulting in a procoagulant state. Abdominal ultrasound with doppler flow is the correct answer as this is the most specific test to confirm the presence of hepatic vein thrombosis.

Ascitic aspiration will likely confirm an abdominal transudate however this will not confirm the causative pathology of hepatic blood flow obstruction secondary to Budd-Chiari syndrome.

CT abdomen can assist with the diagnosis of hepatic vein thrombosis (Budd-Chiari syndrome) however it is less sensitive and specific than ultrasound with doppler flow studies.

Although a coagulopathy may have resulted in the patient's Budd-Chiari syndrome it is most likely due to the use of the combined oral contraceptive pill and therefore coagulation test would be normal.

Endoscopic retrograde cholangiopancreatography is incorrect as there is no specific biliary or pancreatic ductal system pathology.

Question 20 of 86

A 40-year-old female is referred to the clinic with progressive increased tiredness and itching. She is currently also under investigation at a dermatology clinic with a working diagnosis of Sjogren's syndrome having suffered for several years with dry skin, which preceded this new itching.

Today on examination the patient appears jaundiced, with globally dry skin and hyperpigmentation noted over the elbows and knees. Her abdominal exam is normal, and all observations are within the normal range.

Blood tests from the patient's previous clinic attendance are shown below.

Patient's results	Reference ranges	
Haemoglobin	120	130-180 g/L
White cell count	11.0 Ã—10 /L 9	4.0-11.0 Ã—10 /L9
Platelet count	200 Ã—10 /L 9	150-400 Ã—10 /L9
Na+	140	136-146 mmol/L
K+	3.9	3.5-5.0 mmol/L
Creatinine	100	53-106 µmol/L
Total Bilirubin	50	2-17 µmol/L
ALT	60	10-40 U/L
ALP	400	25-100 U/L

Antinuclear antibody	+ve
Anti-mitochondrial antibodies	+ve
Anti-smooth muscle antibodies	+ve
Anti-neutrophil cytoplasmic antibodies	-ve
α1-antitrypsin, α-fetoprotein	-ve

What is treatment may slow the progression of this patient's most likely diagnosis?

A. Ciclosporin
B. Glucocorticoids plus azathioprine
C. Methotrexate
D. Low cholesterol diet
E. Ursodeoxycholic acid

E. Ursodeoxycholic acid

Ursodeoxycholic acid is the first-line medication for primary biliary cholangitis

The patient has a likely diagnosis of primary biliary cholangitis (PBC) presenting with pruritus and cholestatic jaundice on the background of the associated Sjogren's syndrome. On review of the patient's investigations, the highly specific anti-smooth muscle antibodies M2 is positive which is present in 98% of PBC cases. Ursodeoxycholic acid is therefore the correct answer as this is firstline management in PBC and is believed to possibly slows disease progression and improves symptoms.

A number of immunosuppressant agents have been trialled in the management of PBC including cyclosporin however they have been shown to be ineffective in slowing the progression of the disease.

Glucocorticoids plus azathioprine are immunosuppressants used in the management of autoimmune hepatitis. The patients predominately raised ALP and positive anti-smooth muscle antibodies M2 means primary biliary cholangitis is the more likely diagnosis.

Methotrexate is incorrect as this is normal reserved for second-line management in autoimmune hepatitis, not primary biliary cholangitis as seen in this case with the positive, highly specific antismooth muscle antibodies M2.

Although a low cholesterol diet has been associated with a reduction in cholelithiasis complications there is no evidence that it can reduce the progression of PBC.

Question 21 of 86

You see a 45-year-old patient with Crohn's disease in clinic. They had an extensive terminal ileal and small bowel resection in 2011 for active disease with strictures after the failure of medical therapy. Since then he has been well controlled on 8 weekly Infliximab infusions and mesalazine. They come to see you with increasing stool frequency, up to 10 times per day. It is watery and loose and he has been having some nocturnal episodes as well. His observations are within normal limits and his BMI is 21. On examination, his abdomen is soft and non-tender. Rectal examination is unremarkable.

Blood tests are as follows;

Hb	125 g/l
Platelets	298 * 10 /l9
WBC	8.3 * 10 /l9
Na +	141 mmol/l
K +	4.4 mmol/l
Urea	4.1 mmol/l
Creatinine	95 µmol/l
Bilirubin	7 µmol/l
ALP	74 u/l
ALT	23 u/l
Albumin	37 g/l
CRP	7 mg/l
Faecal calprotectin	13 µg/g (normal < 50)
Faecal MC&S	NAD

He goes on to have a colonoscopy which is macroscopically normal and the biopsies show no active disease.

He has a SeHCAT (selenium homocholic acid taurine) scan which shows <5% Selenium retention at 7 days. He is diagnosed as having bile acid malabsorption secondary to terminal ileal resection.

What is the most appropriate treatment to start him on?

A. Loperamide
B. Codeine phosphate
C. Octreotide
D. Cholestyramine
E. Increase infliximab frequency

D. Cholestyramine

Ongoing diarrhoea in Crohn's patient post-resection with normal CRP → cholestyramine

This patient has bile acid malabsorption following extensive terminal ileal and small bowel resection for his Crohn's disease. A complication of extensive terminal ileal resection is bile acid diarrhoea which presents with watery loose stools including nocturnal episodes. The diagnostic test is the SeHCAT scan (nuclear medicine) which used radiolabelled selenium and measures the retention of the labelled molecule. BAD is diagnosed by a <15%

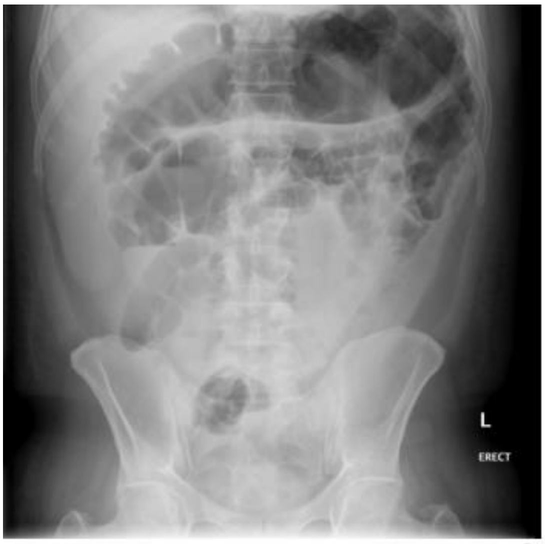
retention of SeHCAT at 7 days. The treatment is with bile acid sequestrants including cholestyramine (Questran). This is the correct answer here, the patient does not have an acute flare so Infliximab frequency does not need altering. Loperamide and codeine will not treat the underlying cause and octreotide can be used to treat diarrhoea in VIPomas.

Question 22 of 86

A 50-year-old man is admitted to hospital with a 5-day history of worsening abdominal pain. Over the past 24 hours, the pain has become very severe. On examination, his pulse is 110/min, blood pressure 110/62 mmHg and temperature 37.7ºC. Bloods show the following:

Hb	105 g/l
Platelets	452 * 10 /l9
WBC	16.3 * 10 /l9
CRP	263 mg/l

The abdominal film is shown below:



What is the most likely underlying disorder?

A. Perforated diverticulum
B. Sigmoid volvulus
C. Ulcerative colitis with toxic megacolon
D. Spontaneous bacterial peritonitis
E. Crohn's disease

C. Ulcerative colitis with toxic megacolon

This patient had toxic megacolon secondary to underlying ulcerative colitis - note the dilated transverse colon. The abdomen demonstrates a markedly dilated transverse colon (9 cm) with an impression of slight dilatation of the descending colon with some 'thumb printing' in the wall. No free subphrenic gas is seen.

They went on to have a subtotal colectomy.

Question 23 of 86

A 56-year-old man has had a recent colonoscopy for per rectal bleeding which found 2 small adenomas <10mm in size. He has been otherwise well and does not have a family history of colorectal cancer

. What is the best option for colonoscopic surveillance in this patient?

A. Offer colonoscopy at 1 year
B. Offer colonoscopy at 3 years
C. Does not require colonoscopic surveillance
D. Offer colonoscopy at 5 years
E. Offer colonoscopy at 6 months

D. Offer colonoscopy at 5 years

The guidance for colonoscopic surveillance is dependent on the patient's risk profile and can be divided into low risk, intermediate risk, and high risk. A patient with 2 small adenomas <10mm in size is at a low risk of developing colorectal cancer. Therefore a repeat colonoscopy should be offered at 5 years.

Patients at intermediate risk should be offered a colonoscopy at 3 years and patients at high risk should be offered a colonoscopy at 1 year.

Question 24 of 86

A 62-year-old retired school teacher attended the diabetes clinic on a routine follow-up visit. He had undergone bariatric surgery for his morbid obesity associated with type 2 diabetes mellitus and obstructive sleep apnoea about 6 months previously. He had noticed a significant amount of weight loss during this period, although he complained of visual symptoms that were more noticeable during late evening hours.

Deficiency of which one of the following vitamins/nutrients is the probable explanation for his visual symptoms?

A. Magnesium
B. Vitamin D
C. Vitamin A
D. Vitamin B12
E. Zinc

C Vitamin A

Vitamin A is essential to maintain visual acuity, immunological functions and cellular proliferation

Vitamin A is essential to maintain visual acuity, immunological functions and cellular proliferation. The incidence of vitamin A deficiency has been reported between 5 and 10% after bariatric surgery. Vitamin A deficiency is associated with night blindness, xerophthalmia, and occasionally complete blindness. Night blindness usually manifests as an inability in adjustment to dimmed light and is an early feature of vitamin A deficiency.

Question 25 of 86

A 38 year-old man, who has a job as a pastry chef is referred by his GP with pale, offensive stools and weight loss of 5 kg over the last two months. He has a past medical history of episodic acid reflux disease for which he was recently prescribed omeprazole. He drinks several glasses of wine a day and has a five year pack history.

Blood tests reveal:

Hb	9.9 g/dL
Mean corpuscular volume (MCV)	115 fl
Platelets	280 * 10 /l9
WBC	7.1 * 10 /l9
Na +	136 mmol/l
K +	4.0 mmol/l
Urea	3.2 mmol/l
Creatinine	52 µmol/l
Bilirubin	15 µmol/l
ALP	140 u/l
ALT	50 u/l
γGT	210 u/l
Albumin	39 g/l
Vitamin B12	120 ng/l
Faecal elastase	98 g/g (normal > 200)

What is the most appropriate next investigation?

A. Abdominal ultrasound scan
B. Liver biopsy
C. Magnetic resonance cholangiopancreatography (MRCP)
D. Colonoscopy
E. Endoscopic retrograde cholangiopancreatography (ERCP)

A. Abdominal ultrasound scan Next question

Abdominal ultrasound is the most appropriate next investigation in this patient's diagnosis. It helps rule out a mass lesion, especially as the patient has suffered from weight loss. Further, it can help detect calcification, given the most likely diagnosis is probably chronic pancreatitis, considering his alcohol history and low levels of faecal elastase. Further investigations may be CT scan or MRCP as this can image the biliary and pancreatic ducts in a non-invasive manner.

Question 26 of 86

A 30-year-old woman presents to the gastroenterology clinic with a flare of ulcerative colitis. She reports that for the month she has been experiencing diarrhoea, with bowels opening up to 10 times per day. She denies any fevers. She has been compliant with mesalazine orally and has continued her rectal mesalazine variably as she finds the enema preparations very difficult to administer. She is a non-smoker and works as a school teacher. Her symptoms are causing her significant difficulties and she has had several accidents at work, which she found very distressing.

On clinical assessment, she is afebrile, basic observations are within normal limits and her abdomen is soft with moderate diffuse tenderness.

Blood tests show:

Hb	100 g/L	Male: (135-180)Female: (115 - 160)
Platelets	600 * 10 /L 9	(150 - 400)
WBC	11.5 * 10 /L 9	(4.0 - 11.0)
ESR	35 mm/hr	(<10)

An abdominal X-ray shows some thickening of her bowel wall but no bowel dilatation.

A flexible sigmoidoscopy is arranged for the following day and shows moderate inflammation and is terminated due to patient discomfort.

The patient is admitted and commences a course of intravenous steroids, which markedly improve her symptoms and within 48 hours she is stepped down to oral steroids and discharged on day three post-admission.

Given the clinical history, which of the following interventions should be considered for this patient once she has recovered from her acute flare?

A. Continue on oral and rectal mesalazine, no changes
B. Long term prednisolone
C. Referral for surgical review
D. Azathioprine
E. Infliximab

D. Azathioprine

If a patient with ulcerative colitis has had a severe relapse or >=2 exacerbations in the past year they should be given either oral azathioprine or oral mercaptopurine to maintain remission

This patient has suffered 2 exacerbations in the past year and thus her current treatment with oral and rectal mesalazine is likely insufficient to maintain remission.

Long term prednisolone has a huge list of side effects and is not recommended as risks often outweigh benefits.

The patient's abdominal X-ray showed no features of toxic megacolon or acute severe inflammation and symptoms settled well with steroids, thus surgical intervention would not be warranted at this stage.

Infliximab could be considered but carries the risk of immune responses and significant immunosuppression, thus is generally reserved for patients with a severe flare not responding to steroids, or in cases that are unresponsive/intolerant to azathioprine or 6-mercaptopurine.

Remember TPMT should be checked in all patients ideally prior to commencement of either azathioprine or 6-mercaptopurine to reduce the risk of toxicity.

Question 27 of 86

A 24-year-old female presents to general practice with a several week history of diarrhoea, passage of mucus, lethargy and abdominal discomfort relieved by defecation. A blood test is arranged showing the following:

Na +	138 mmol/l
K +	4.0 mmol/l
Urea	4.5 mmol/l
Creatinine	80 µmol/l
Hb	11 g/dl
Platelets	320 * 10 /l9
WBC	4.0 * 10 /l9

CRP	1.0 mg/l
Tissue transglutaminase antibody	neg

Which one of the following agents would be most suitable for her?

A. Linaclotide
B. Codeine
C. Sertraline
D. Loperamide
E. Amitriptyline

D. Loperamide

This is a clinical diagnosis of irritable bowel syndrome, supported by relief on defaecation as well as a panel of normal blood tests. The first-line anti-motility agent for this presentation of diarrhoea would be loperamide, as recommended by NICE guidelines. Whilst codeine may be effective in minimising diarrhoea, it has no established roles in the management of IBS.

Linaclotide is a laxative that is recommended in use of IBS if symptoms of constipation predominate, if the patient has had constipation for 12 months and if a trial of the more conventional laxatives fail.

Sertraline and amitriptyline are second-line pharmacological treatments if anti-motility treatment, laxatives and antispasmodics have failed to improve symptoms.

Question 28 of 86

A 25-year-old man who is normally fit and well presents with a 2-week history of crampy abdominal pain and bloody diarrhoea. A colonoscopy shows erythema and oedema in the distal colon and evidence of proctitis. He is started on mesalazine.

What blood test(s) must be done prior to its commencement?

A. Renal function and full blood count
B. Thiopurine methyltransferase activity
C. Amylase
D. Hepatitis screen
E. Liver function test and full blood count

A. Renal function and full blood count

Renal function should be monitored before starting an oral aminosalicylate, at 3 months and then annually thereafter. This should be done more often in the presence of renal impairment. Blood disorders can also occur with mesalazine, and patients should be asked to look out for bruising, bleeding, purpura, fever and sore throat.

TPMT levels should only be needed on commencement of mercaptopurine or azathioprine.

Amylase, hepatitis screen and liver function tests are not required in aminosalicylate therapy monitoring.

Question 29 of 86

A 28-year-old male presents with frank haematemesis. The patient admits to drinking 1L of vodka daily. He has a background of alcoholic liver disease. On examination, there is evidence of gross abdominal ascites, flapping tremor and jaundice. His blood pressure is 96/50mmHg, pulse rate 105 bpm. You presume variceal bleeding as the cause of his symptoms.

Bilirubin	83 µmol/l
ALP	52 u/l
ALT	253 u/l
γGT	151 u/l
Albumin	28 g/l
Hb	98 g/l
Platelets	51 * 10 /l9
WBC	2.5 * 10 /l9

Apart from urgent endoscopic assessment and starting terlipressin which further initial management will reduce mortality?

A. Propranolol
B. Blood transfusion
C. Tranexamic acid
D. Broad spectrum intravenous antibiotics
E. Urgent referral for transjugular intrahepatic portosystemic shunt (TIPSS)

D. Broad spectrum intravenous antibiotics

Bacterial infections occur in about 20% of patients with cirrhosis with upper gastrointestinal bleeding within 48 hours of admission. Prognosis both in terms of re-bleeding, failure to control bleeding, and in-hospital outcome are closely related to bacterial infections. BSG guidelines advocate the use of prophylactic antibiotics in acute variceal bleeding.

Question 30 of 86

A 60-year-old woman with a known history of alcohol dependency presents with a 4-hour history of haematemesis and abdominal discomfort. She has no history of upper GI bleed but does report several previous episodes of passing black stools. She has asthma and a family history of type II diabetes mellitus. A previous abdominal ultrasound has shown hepatic cirrhosis.

Today, she appears unwell. She continues to vomit small amounts of blood. On examination, her blood pressure is 120/85mmHg and pulse rate is 90 beats per minute. Her chest is clear and her abdomen is soft, though she has some epigastric tenderness. Rectal examination reveals melaena.

An intravenous cannula is inserted and blood is taken. Blood tests show:

Hb	98 g/l
Platelets	180 * 10 /l9
WBC	6.3 * 10 /l9
Fibrinogen	2.3 g/L
APTT	30 seconds
PT	12.5 seconds
Na +	142 mmol/l
K +	4.9 mmol/l
Urea	10 mmol/l
Creatinine	100 µmol/l

Fluids are started and arrangements are made to send the patient to the endoscopy suite.

What should be administered prior to endoscopy?

A. Terlipressin
B. Octreotide
C. Platelets
D. Packed red cells
E. Fresh frozen plasma (FFP

A. Terlipressin

This patient has oesophageal varices secondary to portal hypertension. She has a history of alcoholic liver disease and may have had small bleeds in the past. The most appropriate treatment is terlipressin, a synthetic vasopressin analogue. It causes splanchnic vasoconstriction, which reduces portal pressure and so eases variceal bleeding. NICE guidance recommends offering terlipressin on presentation and stopping treatment either after definitive haemostasis is achieved, or after 5 days. Prophylactic antibiotics should also be given to these patients, due to the high incidence of bacterial infection.

Octreotide is a vasoactive agent that has been used to treat variceal bleeding, but unlike terlipressin has not been shown to reduce mortality. Platelets and FFP are not appropriate treatments for this patient, as her platelet count and fibrinogen are normal. The haemoglobin should be closely monitored, but the clinical picture at this juncture does not warrant blood transfusion.

Question 31 of 86

A recovering alcoholic patient on the ward has developed confusion two days after admission to hospital. In addition he reports difficulty standing up from sitting and increasing shortness of breath.

Further questioning reveals he normally drinks 5-6 pints of medium strength lager most days but his last drink was 7 days ago. His diet is poor. He smokes 30 rolled up cigarettes per day. On examination it is noted his chest is clear but he he has generalised muscle weakness.

What investigation will most likely reveal the underlying abnormality?

A. CT brain
B. Full blood count
C. Urea and electrolytes, including phosphate and magnesium
D. Muscle biopsy
E. Electromyography (EMG)

C. Urea and electrolytes, including phosphate and magnesium

Alcoholic patients are often malnourished. In hospital the patient has (hopefully!) no access to alcohol but also three meals a day provided. Re-feeding syndrome is the only condition responsible for the above symptoms that can be readily diagnosed from the above tests. Blood tests will reveal electrolyte disturbances particularly hypophsphataemia accompanying the neurological and pulmonary manifestations. Treatment involves replacing the depleted electrolytes and slow build up of diet.

Question 32 of 86

A 40-year-old man who recently had an appendicectomy presents to hospital unwell. He has generalised abdominal pain and pyrexia. Initial bloods are presented below.

Hb	134 g/l	Na +	139 mmol/l	Bilirubin	18 µmol/l	B12 900ng/l
Platelets	490 * 10 /l 9	K +	4.1 mmol/l	ALP	120 u/l	Folate 2.1µg/l
WBC	23.2 * 10 /l 9	Urea	7.6 mmol/l	ALT	35 u/l	Iron 18µmol/l
Neuts	19.8 * 10 /l 9	Creatinine	101 µmol/l	γGT 30 u/lAlbumin 41 g/l		
Lymphs	3.2 * 10 /l 9					
Eosin	0.1 * 10 /l9					

Automated lab haematinics show a grossly elevated serum B12 with normal folate and iron. Blood culture confirms gram-negative septicaemia

. What is the likely diagnosis?

A. Atypical respiratory tract infection
B. Infectious mononucleosis
C. Liver abscess
D. Catheter associated urinary tract infection
E. Infective endocarditis Next question

C. Liver abscess

The best answer is a liver abscess. A history of recent appendicectomy provides a clue, a liver abscess may form consequently to any form of intra-abdominal sepsis e.g. perforated appendix, biliary sepsis. Most commonly the causative organism is *E. Coli* or anaerobic organisms. Vitamin B12 is stored in the liver and can be released during infection.

Infectious mononucleosis is unlikely to cause a neutrophilia being a virus.

Question 33 of 86

A 52-year-old woman presents with several months of widespread itching. She denies any rashes and feels well in herself, aside from feeling more fatigued than usual. She has a past medical history of Hashimoto's thyroiditis, for which she takes levothyroxine.

Examination is unremarkable. Blood tests results are:

Na +	138 mmol/L	(135 - 145)
K +	4.7 mmol/L	(3.5 - 5.0)
Urea	5.6 mmol/L	(2.0 - 7.0)
Creatinine	67 µmol/L	(55 - 120)
Bilirubin	32 µmol/L	(3 - 17)
ALP	178 u/L	(30 - 100)
ALT	58 u/L	(3 - 40)

Î³GT	154 u/L	(8 - 60)
Albumin	38 g/L	(35 - 50)

Given the likely diagnosis, which of the following is the most appropriate management?

A. Cetirizine
B. Cholestyramine
C. Fexofenadine
D. Prednisolone
E. Ursodeoxycholic acid

E. Ursodeoxycholic acid

Ursodeoxycholic acid is the first-line medication for primary biliary cholangitis

The history of fatigue and itching in an otherwise-well middle-aged female patient, with a background of autoimmune disease, strongly points towards primary biliary cholangitis. This is supported by the elevated LFTs, in particular the ALP and gamma-GT. Subsequent testing would involve screening for anti-mitochondrial antibodies (AMA), which are highly specific, present in 98% of patients. First-line treatment involves the use of ursodeoxycholic acid, which slows disease progression and improves symptoms.

Cetirizine is an antihistamine. Whilst generally used to relieve symptoms of itching in numerous conditions, given that the underlying diagnosis here is primary biliary cholangitis, ursodeoxycholic acid is the more definitive management, and cetirizine may be used as an adjunct for symptom relief.

Cholestyramine may be used as an adjunct to help with the relief of pruritus. However, it does not play a role in the management of disease progression - ursodeoxycholic acid is used for this initially.

Fexofenadine is another antihistamine, similar to cetirizine. Unlike cetirizine, it is not available overthe-counter and requires a prescription. Again, it would not routinely be used in the management of primary biliary cholangitis.

Prednisolone is a corticosteroid and is sometimes used if there are overlapping features of autoimmune hepatitis present alongside primary biliary cholangitis. There is nothing in this scenario to warrant its use.

Question 34 of 86

A 75-year-old man was admitted to hospital with a three-day history of right sided abdominal pain and vomiting. He has a history of ischaemic heart disease, chronic obstructive pulmonary disease and peripheral vascular disease.

On examination, he appears unwell. His temperature is 38.5ºC, heart rate is 120 beats per minute, respiratory rate is 24 breaths per minute and blood pressure is 90/60 mmHg. He appears jaundiced. Examination of his abdomen reveals tenderness over the right upper quadrant on palpation.

Blood tests show:

Hb	115 g/l	Na +	146 mmol/l	Bilirubin	225 µmol/l
Platelets	650 * 10 /l 9	K +	5.8 mmol/l	ALP	894 u/l
WBC	24.3 * 10 /l 9	Urea	10.5 mmol/l	ALT	160 u/l
Neuts	20.5 * 10 /l 9	Creatinine	190 µmol/l	γGT	279 u/l
CRP	348 mg/L	Albumin	27 g/l		

An urgent ultrasound of his abdomen reveals sludge in his gallbladder and a dilated common bile duct at 13mm. His spleen and kidneys appear normal. He was fluid resuscitated and started on piperacillin-tazobactam after blood cultures were taken. After 6 hours of treatment, his blood pressure improves to 130/70 mmHg, his heart rate has decreased to 95 beats per minute and he is passing 50 millilitres of urine an hour.

What is the next appropriate management for this patient?

A. Watch and wait
B. Endoscopic retrograde cholangiopancreatography
C. Percutaneous transhepatic cholangiogram
D. Urgent cholecystectomy
E. Refer to intensive care

B. Endoscopic retrograde cholangiopancreatography

seen in patients with ascending cholangitis. His LFTs show a cholestatic picture and his high serum bilirubin is indicative of an obstruction. Sludge in his gallbladder and a dilated common bile duct is indicative of choledocholithiasis.

The most important management for this patient after initial resuscitation would be to urgently decompress the biliary system and remove the obstructing gallstone via an endoscopic retrograde cholangiopancreatography (ERCP) and sphincterotomy. Intensive care input is not indicated at this stage as the patient has shown signs of clinical improvement. However, in severe septic shock, inotropic support may be needed to stabilise the patient prior to an ERCP. In patients who are unfit for an ERCP, a percutaneous transhepatic cholangiogram could be done.

Question 35 of 86

A 55-year-old man presents to the emergency department with shortness of breath.

He reports several months of worsening diarrhoea and is now passing over 10 watery motions per day.

On examination he is dyspnoeic at rest. The blood pressure is 104 / 77mmHg and the pulse rate is 90bpm and regular. Oxygen saturations are 93% on room air. Chest auscultation reveals widespread mild polyphonic wheeze and bibasal crackles. Jugular venous pressure is elevated and C-V waves are present. There is a pansystolic murmur at the left lower sternal border louder in inspiration. There is a palpable liver edge 2cm below the costal margin.

Which investigation is most likely to confirm the diagnosis?

A. Growth hormone day curve
B. 24 hour urine 5-HIAA
C. Faecal calprotectin
D. 24 hour urine VMA
E. Stool sample for ova, cysts and parasites

B. 24 hour urine 5-HIAA

A long history of diarrhoea along with signs consistent with tricuspid regurgitation is suggestive of carcinoid syndrome. This is a a paraneoplastic syndrome that is caused by endogenous secretion of serotonin (chiefly) from carcinoid tumours.

About 50% of patients have cardiac abnormalities caused by serotonin-induced fibrosis of the valvular endocardium, notably the tricuspid and pulmonary valves. The majority of patients with cardiac involvement present with signs of right heart failure secondary to severe dysfunction of these right-sided valves.

The most useful initial test is the 24-hour urine levels of 5-HIAA (5-hydroxyindoleacetic acid) - the end product of serotonin metabolism.

Growth hormone day curves are used to diagnose acromegaly.

24 hour urine VMA (Vanillylmandelic acid) is used in the investigation of pheochromocytoma. Stool culture could be used to exclude infective causes of diarrhoea but will not confirm the diagnosis in this case.

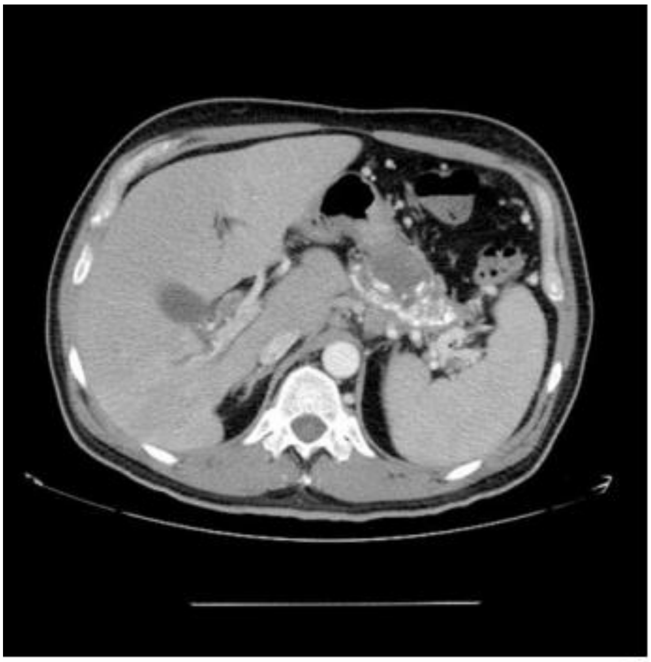
Faecal calprotectin is a non-specific marker of gut inflammation and will not confirm the diagnosis in this case.

Question 36 of 86

A 62-year-old woman is investigated for chronic diarrhoea. She is currently opening her bowels 5- 6 times a day. This is associated with episodes of central abdominal pain. Some of these episodes are severe and do not seem correlated with the episodes of diarrhoea. Her symptoms started around 6 months ago and have slowly been getting worse.

Clinical examination is unremarkable.

She is a non-smoker and drinks around 6 units of alcohol per week. Her past medical history includes depression and asthma.



What is the most likely diagnosis?

A. Crohn's disease
B. Pancreatic cancer
C. Chronic pancreatitis
D. Ulcerative colitis
E. Multiple endocrine neoplasia

C. Chronic pancreatitis

The CT shows classic changes consistent with chronic pancreatits. Note the irregular shaped pancreas with the typical calcifications.

Whilst a history of alcohol excess is a common cause of chronic

Question 37 of 86

A 32-year-old pregnant woman presents to the GP with jaundice and itchy skin for the past 2 weeks. She claims that is a lot worse during this pregnancy compared to her last one. History reveals that she is currently 30 weeks pregnant with no complications up until presentation. On examination, the only notable findings are mild jaundice seen in the sclerae, as well as excoriations around the umbilicus and flanks. She denies any tenderness in her abdomen during the examination. Blood tests show the following:

ALT	206 U/L
AST	159 U/L
ALP	796 umol/l
GGT	397 U/L
Bilirubin (direct)	56 umol/L
Bile salts	34 umol/L
Bile salts reference range	0 - 14 umol/L

What is the most likely diagnosis?

A. Obstetric cholestasis
B. Budd-Chiari syndrome
C. Acute hepatitis
D. HELLP syndrome
E. Acute fatty liver of pregnancy

A. Obstetric cholestasis

Obstetric cholestasis, also known as intrahepatic cholestasis of pregnancy, is a condition caused by the impaired flow of bile. This, in turn, causes a build up of bile salts which can then deposit in the skin (causing pruritus) as well as the placenta. It is thought that the aetiology of this condition is a combination of hormonal, genetic and environmental factors.

Although the pruritic symptoms can be distressing for the mother, the build up of bile salts can also be detrimental to foetal wellbeing. The combination of the immature foetal liver's ability to cope with breaking down the excessive bile salt levels as well as the vasoconstricting effect of bile salts on human placental chorionic veins has been theorised to be the cause of sudden asphyxia events in the foetus leading to anoxia and death.

Question 38 of 86

A 45-year-old man is reviewed on the gastroenterology ward. He has a background of alcoholic liver disease and had a transjugular intrahepatic portosystemic shunt (TIPS) performed yesterday due to recurrent variceal haemorrhage. The nursing staff report that he is uncharacteristically confused and combative.

What complication of TIPS has he most likely developed?

A. Cerebral venous sinus thrombosis
B. Air embolism
C. Wernicke's encephalopathy
D. Hepatic encephalopathy
E. Biliary sepsis

D. Hepatic encephalopathy uestion

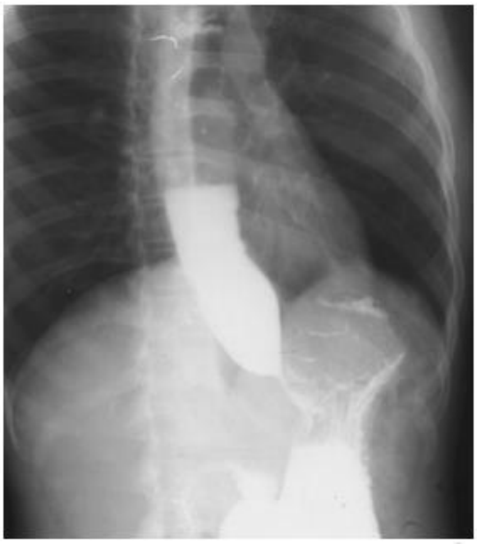
Transjugular intrahepatic portosystemic shunt may precipitate hepatic encephalopathy

Hepatic encephalopathy is a common complication of TIPS, occurring in up to 25% of patients caused by the increased portosystemic passage of nitrogen from the gut.

Whilst Wernicke's encephalopathy can cause confusion in alcoholics

Question 39 of 86

A barium swallow is arranged for a 40-year-old man with dysphagia.



What is the most likely diagnosis?

A. Pharyngeal pouch
B. Retrosternal goitre
C. Oesophageal cancer
D. Hiatus hernia
E. Achalasia

E. Achalasia

This film demonstrates the classical 'bird's beak' appearance of the lower oesophagus that is seen in achalasia. An air-fluid level is also seen due to a lack of peristalsis.

Question 40 of 86

A 47-year-old lady presents to the Emergency Department with large-volume haematemesis. The blood is bright red and she reports experiencing 3 separate episodes over the preceding 12 hours. Despite a previous diagnosis of alcoholic liver disease, she continues to drink a bottle of vodka per day.

On examination, she is pale and clammy. Her pulse is 128bpm and her blood pressure is 87/58mmHg. Her chest is clear and her heart sounds are normal. Her abdomen is distended with mild right upper quadrant tenderness and evidence of shifting dullness.

She is resuscitated with intravenous fluids and 4 units of cross-matched blood are requested. Terlipressin and intravenous ceftriaxone are administered whilst an emergency endoscopy is arranged. She subsequently undergoes variceal band ligation and returns to the ward, where she passes an uneventful night.

Over the next few days, she has two further episodes of large-volume haematemesis which are only partly controlled by repeat endoscopic intervention. She is reviewed by the Gastroenterology Consultant and a plan to refer the patient for a transjugular intrahepatic portosystemic shunt (TIPSS) is made.

Which of the following is the greatest contraindication to a TIPSS procedure?

A. Child-Pugh class A liver disease
B. Hepatic encephalopathy
C. INR 1.7
D. Refractory ascites
E. Pleural effusion

B. Hepatic encephalopathy

A TIPSS procedure involves the percutaneous creation of a low-pressure tract between the intrahepatic portal vein and the hepatic vein. This allows blood to bypass the liver and lowers portal pressure. The primary indications for TIPSS include uncontrolled variceal haemorrhage, refractory ascites, and hepatic pleural effusion.

Absolute contraindications for TIPSS include:

- Severe and progressive liver failure (Child-Pugh score >12 is associated with a high risk of early death)
- Uncontrolled hepatic encephalopathy
- Right-sided heart failure
- Uncontrolled sepsis
- Unrelieved biliary obstruction

Relative contraindications:

- Severe uncorrectable coagulopathy (INR >5)
- Thrombocytopenia <20 * 10 /l
- Portal and hepatic vein thrombosis
- Pulmonary hypertension
- Central hepatoma

Taking the above into consideration, we can eliminate options 4 and 5 as they are actually indications for a TIPSS procedure. Child-Pugh class A liver disease is not a contraindication to TIPSS and it is debatable whether an INR of 1.7 would need to be corrected prior to the procedure. This leaves option 2 as the correct answer.

Question 41 of 86

A 65-year-old woman comes to the gastroenterology clinic with persistent iron deficiency anaemia. she has had two colonoscopies and one upper GI endoscopy without finding a significant lesion, and haemoglobin continues to drop despite iron replacement. On examination she has a blood pressure of 142/105 mmHg, her pulse is 75 and regular. She looks pale. You note an ejection systolic murmur.

Which of the following is the most appropriate way to investigate her?

A. Capsule endoscopy
B. CT colonoscopy
C. Labelled white cell scan
D. Repeat upper GI endoscopy
E. Repeat colonoscopy

A. Capsule endoscopy

The suspicion here is a diagnosis of Heyde's syndrome, with aortic stenosis and small bowel angiodysplasia. The pathogenesis of the angiodysplasia is unknown but may be related to local circulatory changes because of the valve stenosis. Further conventional endoscopy is not likely to be useful, capsule endoscopy that can adequately visualise the small bowel. Angiodysplasia usually resolves once the valve is repaired.

Further conventional endoscopy investigations have potentially a less than 5% chance of picking up a previously undetected lesion. Labelled white cell scan is most useful for large volume bleeding, and CT colonoscopy is less sensitive than conventional endoscopy.

Question 42 of 86

A 25-year-old woman with a long history of Crohns' disease comes to the clinic for review. She is 12 weeks pregnant. Current medication for Crohns' includes 100mg BD of azathioprine and her disease is currently stable. She is opening her bowels 2-3 times per day with a normal, formed motion. Clinical examination is unremarkable, abdomen is soft and non tender, with no palpable masses, bowel sounds are normal.

Hb	110 g/l	Na +	139 mmol/l
Platelets	180 * 10 /l 9	K +	4.0 mmol/l
WBC	6.7 * 10 /l 9	Urea	7 mmol/l
Neuts	3.2 * 10 /l 9	Creatinine	80 µmol/l
Lymphs	1.4 * 10 /l 9	CRP	10 mg/l
Eosin	0.7 * 10 /l9		

Which of the following is the most appropriate way to manage her Crohn's?

A. Stop all immunosuppressives
B. Add prednisolone
C. Continue azathioprine
D. Switch to methotrexate
E. Switch to prednisolone

C. Continue azathioprine

Considerable data has now accumulated on use of azathioprine in women during pregnancy who have an underlying diagnosis of inflammatory bowel disease, arthritis or skin disease. Although this is registry data, and hence not as robust as a randomised controlled trial, it does not suggest a significant increase in risk of teratogenicity associated with azathioprine use. For this reason guidance from the BGS supports it's continued use.

Methotrexate is teratogenic and therefore cannot be recommended for use in pregnancy. Given adverse effects of prednisolone use such as weight gain and reduced bone mineral density, long term use of corticosteroids in pregnancy isn't recommended, although they would still be used to manage an exacerbation whilst on azathioprine. Stopping all immunosuppressives risks worsening Crohns' and is clearly not advisable.

Question 43 of 86

A 38-year-old woman comes to the follow up clinic some 6 weeks after a small bowel resection for Crohn's disease. She is currently prescribed azathioprine and a tapering dose of corticosteroids. She smokes 10 cigarettes per day and endeavours to eat a normal diet.On examination her blood pressure is 115/78 mmHg, pulse is 70 beats per minute and regular. Cardiac and respiratory systems are unremarkable, abdomen is soft and non tender, with a midline scar consistent with the recent laparotomy. Her body mass index is 22 kg/m². Routine bloods are unremarkable.

Which of the following is most important with respect to reducing risk of future exacerbations?

A. Anti-TNF biological therapy
B. Gluten free diet
C. Lactose free diet
D. Oral mesalazine
E. Smoking cessation

E. Smoking cessation

A prospective study suggests that continued smoking raises the risk of Crohn's exacerbation by approximately 50%.The study examined 573 Crohn's disease patients for a follow up period of 4 years. In comparison with nonsmokers, continuing smokers relapsed more frequently with an incidence rate ratio of 1.53 (95% confidence interval : 1.102.17). Former smokers and quitters had similar relapse incidences compared with nonsmokers.

Mesalazine in Crohn's disease has debatable efficacy. A meta-analysis conducted in 2004 revealed a statistically significant but probably clinically insignificant effect on disease progression. Anti-TNF agents are effective in maintaining remission in fistulating Crohn's disease, but must be continued, as relapse rates are high, and come with not insignificant risks from immunosuppression. These interventions contrast with smoking cessation, which has a positive effect independent of background therapy.

Gluten free diet impacts on the clinical course of coeliac disease, and a lactose free diet often improves recovery after gastroenteritis.

Question 44 of 86

A 40-year-old woman with a background of Sjogren's syndrome presents with fatigue and itchy skin. She has not been commenced on any new medications or travelled recently. On examination, she is found to have a palpable liver edge. Further investigations reveal the following results:

Bilirubin	22 µmol/l
ALP	400 u/l
ALT	35 u/l

Anti-mitochondrial antibodies positive, anti-smooth muscle antibodies negative

Anti-HBs positive, HBsAg negative, HBcAg negative Hepatitis C negative

What is the most likely diagnosis?

A. Primary biliary cirrhosis
B. Primary sclerosing cholangitis
C. Autoimmune hepatitis
D. Acute Hepatitis B infection
E. Hepatitis B carrier

A. Primary biliary cirrhosis

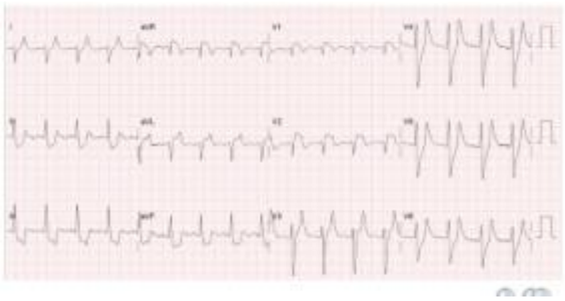
Given the fact that she is a middle-aged woman, with a background of Sjogren's and a positive antimitochondrial antibody test, the best answer is primary biliary cirrhosis.

Negative anti-smooth muscle antibodies make autoimmune hepatitis less likely. Primary sclerosing cholangitis is frequently associated with ulcerative colitis and does not fit the clinical picture here. Her hepatitis B results indicate previous vaccination and not an infection or carrier status.

Question 45 of 86

A 65-year-old woman with a known history of hypertension, ischaemic heart disease and diabetes was brought in by ambulance after the family found her collapsed on the bedroom floor. She lives alone and the family is unsure about the duration the patient was on the floor. Her vital signs are stable with a pulse rate of 100 bpm and a spot hs troponin was normal. Other blood tests were pending.

Her 12 lead ECG shows:



What is the next most important step in her management?

A. Calcium gluconate
B. Magnesium sulphate
C. Normal saline with added potassium chloride infusion
D. Percutaneous coronary intervention
E. Transvenous cardiac pacing

A. Calcium gluconate

The ECG shows peaked or 'tall-tented' T waves, loss of P waves and broad QRS complexes with a sinusoidal wave pattern suggestive of hyperkalaemia.

Calcium gluconate is the correct answer. The ECG changes are consistent with hyperkalaemia likely secondary to acute renal failure from a fall, long lie and myoglobinuria. Patients with hyperkalaemia and ECG changes are at high risk of developing ventricular fibrillation (VF). Calcium gluconate is a cardiac membrane stabiliser that reduces the risk of VF and should be promptly administered in this patient.

Magnesium sulphate is the treatment of choice for torsades de pointes and may be considered in high burden of recurrent premature ventricular complexes in ventricular bigeminy or trigeminy which are not seen in this patient.

Normal saline with added potassium chloride infusion is the treatment of choice in hypokalaemia. Hypokalaemia causes typical ECG changes of widespread ST depression, T wave inversion, and prominent U waves which are not seen in this patient.

Percutaneous coronary intervention is the treatment of choice for ST elevation myocardial infarction. ECG changes in hyperkalaemia can mimic STEMI as seen in this ECG but the normal hs troponin makes STEMI less likely.

Transcutaneous pacing is used in patients with symptomatic bradyarrhythmia while they await a permanent pacemaker insertion. This patient does not have bradyarrhythmia and has a perfusion pulse rate of 100bpm. Hence pacing is not indicated in this patient.

Question 46 of 86

A 54-year-old gentleman present to the general medical clinic with feelings of bloatedness, rising acid in his chest and pain associated with mealtime. He is investigated with endoscopy and found to have a peptic ulcer and further testing demonstrates evidence of Helicobacter pylori. He has a past medical history of depression. He does not take any regular medications. He specifically denies any use of NSAIDs. He is known to be allergic to penicillin having suffered a systemic rash as a child following treatment for an inner ear infection

. How should he be treated?

A. Proton pump inhibitor, metronidazole and clarithromycin
B. Proton pump inhibitor, amoxicillin and clarithromycin
C. Proton pump inhibitor, bismuth, metronidazole and tetracycline
D. Metronidazole and clarithromycin
E. Proton pump inhibitor, and metronidazole

A. Proton pump inhibitor, metronidazole and clarithromycin

The correct answer is a proton pump inhibitor, metronidazole and clarithromycin. This is a patient with a peptic ulcer associated with Helicobacter pylori. NICE advises an eradication strategy, which should take allergies and recent antibiotic use into account.

FIrst-line treatment over seven days:

- If no allergies: PPI, amoxicillin and clarithromycin or metronidazole
- If penicillin allergic: PPI, clarithromycin and metronidazole
- If penicillin allergic and previous clarithromycin exposure: PP, bismuth, metronidazole and tetracycline

As he may be penicillin allergic he should be treated as per the second option.

Question 47 of 86

A 42-year-old woman presents to the emergency department generally unwell. She has been feeling progressively worse over the last five days, complaining of a productive cough, worsening abdominal swelling and severe constipation. A friend who is with her as also noticed she has been increasingly confused, and also mentions to you that she has an extensive alcohol history. She has a past medical history of cirrhosis and COPD. She does not remember what her medications are called, but she does take several tablets in the morning and evening and uses a red inhaler regularly.

Examination shows her to look generally unwell, feel hot and clammy. She appears jaundiced. There are crepitations in the left lower lobe. Her abdomen is distended and there is evidence of ascites. She also has distended veins on her chest.

Observations

Saturations	93%
Respiratory rate	18/min
Blood pressure	121/58mmHg
Heart rate	109/min
Temperature	38.2°C

Blood tests:

Hb	121 g/l
Platelets	52 * 10 /l^9
WBC	16 * 10 /l^9
Na +	147 mmol/l
K +	4.8 mmol/l
Urea	11.1 mmol/l
Creatinine	153 µmol/l
Bilirubin	57 µmol/l
ALP	173 u/l
ALT	121 u/l
Albumin	26 g/l
INR	2.3
PT	25s

Further to his blood tests a CXR demonstrates left lower lobe consolidation. Blood cultures are sent, alongside calcium, phosphate, magnesium and glucose. An ultrasound scan of her abdomen is requested, as is a urine dip.

What other investigation is most appropriate in the acute setting?

A. CT chest
B. CT head
C. Abdominal X-ray
D. Lumbar puncture
E. Ascitic tap

E. Ascitic tap

The correct answer is ascitic tap. This is a case of acute decompensated cirrhosis secondary to lower respiratory tract infection. A CT head and lumbar puncture can both be useful in excluding causes of confusion, and a CT head would definitively have been indicated if there was any evidence of head trauma. However; there is a clear cause for confusion in the context of sepsis and likely hepatic encephalopathy. An abdominal X-ray and CT chest are unlikely to be helpful, but an ascitic tap can give valuable information about the nature of the ascites by looking at the serumascites albumin gradient (SAAG). It is also important to obtain a sample prior to starting antibiotics.

Question 48 of 86

A 54-year-old Caucasian female presents with a 3-month history of dysphagia to bread and solid meats, with no problems with swallowing oral liquids. She has lost about 2 stones in weight unintentionally during this period. She denies any night sweats or fevers, haematemesis, change in bowel habit or melaena. She reports no change in her voice or aspiration episodes. On examination, you note koilonychia and an enlarged tongue on examination with conjunctival pallor. Her blood tests are as follows:

Hb	76 g/l
MCV	62 fl
Platelets	246 * 10 /l^9
WBC	7.2 * 10 /l^9
Ferritin	22 ng/ml
Na +	142 mmol/l
K +	4.2 mmol/l
Urea	5.6 mmol/l
Creatinine	55 µmol/l
CRP	20 mg/l

Her GP refers her for an urgent upper oesophageal-gastric endoscopy (OGD) as part of the 2-week wait of suspected cancer. OGD reveals narrowed distal oesophageal lumen caused by webs that are ruptured as the scope is passed.

What is the most likely diagnosis?

A. Plummer-Vinson syndrome
B. Oesophageal squamous cell carcinoma
C. Oesophageal adenocarcinoma
D. Pharyngeal carcinoma
E. Oesophageal rings

A. Plummer-Vinson syndrome

Oesophageal webs in association with iron deficiency anaemia (resulting in clinical glossitis and koilonychia) and dysphagia is Plummer-Vinson syndrome, particularly prevalent in middle-aged white Caucasian females. The post cricoid oesophageal webs are premalignant for oesophageal squamous cell carcinoma (SCC) and pharyngeal SCC, which instead of webs on OGD, present as plaques, nodules or ulcerated masses instead. However, the agreed frequency of follow-up OGD monitoring is debated in Plummer-Vinson syndrome. Frequently, the webs recede on iron replacement and break on the scope passing through, improving dysphagia. Infrequently, dilation may be required, as in oesophageal rings, caused by mucosal out folds most frequently as a result of chronic oesophageal reflux and hence, treated in conjunction with proton pump inhibitors.

Question 49 of 86

A 55-years-old male presents with a 4 week history of persistent diarrhoea and transient shortness of breath, which he describes to have been very similar to his childhood asthma. He has no other past medical history, is a builder and smokes 10 cigarettes per day for the past 30 years. He drinks minimal alcohol. On examination, you note the patient has a very red face, no clubbing and soft and non-tender abdomen. Cardiovascular examination reveals a mid diastolic murmur loudest at the left sternal border. Respiratory examination is unremarkable. blood pressure is 86/49 mmHg, heart rate 112 beats/min, sats 96% on air and respiratory rate 20 breaths/ min.

Which test is most likely to be diagnostic?

A. Urinary serotonin
B. Lung function tests
C. 24 hour urinary 5HIAA
D. Plasma chromogranin A
E. Echocardiogram

C. 24 hour urinary 5HIAA

The patient presents with the diarrhoea, tricuspid stenosis, facial flushing, bronchospasm and hypotension, clearly pointing towards a unifying diagnosis of carcinoid syndrome. However, screening for carcinoid syndrome should be performed with 24 hour urinary 5HIAA, the end product of serotonin metabolism. Although well-differentiated carcinoid tumours secrete elevated levels of plasma chromogranin A, it is increasingly recognised that falsely positive elevated levels can be produced by other neuroendocrine tumours, hyperthyroidism, hyperparathyroidism, pituitary tumours, colon carcinoma, IBD, hypertension, COPD, ACS amongst many others. The low specificity of the test means that it is recommended that it is not used on its own as a screening test.

Urinary serotonin may also be useful but is criticised for low sensitivity for foregut carcinoid tumours, which often lack aromatic amino acid decarboxylase to produce 5HIAA. Although current guidelines recommend the measurement of both 24hr urinary 5HIAA and plasma chromogranin A1, 24 urinary 5HIAA is therefore most likely to be diagnostic.

Question 50 of 86

A 60-year-old lady originally presented with diarrhoea and flushing 8 months ago and was diagnosed with carcinoid syndrome. She was managed medically with Octreotide and had been stable but her concerned daughter brought her today in with confusion, she seems to be seeing things and her diarrhoea was becoming a problem again. She also had an itchy rash predominantly on the back of her hands and around her neck. The GP had suggested emollients and topical corticosteroids but this had made no improvement. On examination, there was an erythematous, scaly rash on the dorsum of both hands and around the neck, she had an abbreviated mental test score (AMTS) of 4/10 with no other positive findings.

What is the most likely diagnosis?

A. Irritant contact dermatitis
B. Brain metastases
C. Dermatomyositis
D. Beriberi
E. Pellagra

E. Pellagra

Pellagra is a rare condition in the United Kingdom but it is seen in patients with carcinoid syndrome. It is caused by a deficiency in vitamin B3, (Niacin) and classically presents as the '4 Ds' dermatitis, dementia, diarrhoea and death. Niacin can be absorbed through the diet or converted in vivo from tryptophan. Patients with carcinoid syndrome are at increased risk as tryptophan stores are metabolised to serotonin.

Dermatomyositis characteristically presents with a heliotrope rash, Gottron's papules and muscle weakness but does not cause confusion or diarrhoea typically, carcinoid tumours rarely metastasise to the brain.

Beriberi is caused by thiamine deficiency and typically causes a peripheral neuropathy, highoutput cardiac failure and is associated with weight loss.

Question 51 of 86

A 32-year-old man presents with loose stools or diarrhoea on all occasions for almost a year now, associated with intermittent, mild, generalised, crampy abdominal pain that has no obvious triggers. He has lost three stone (19 kilogrammes) in weight over the year despite not trying to. For nearly 10 years he has had episodes of arthralgia and fever which he has never thought to consult about. He once travelled to Gambia on his gap-year aged 18 and recalls several mosquito bites. He took full malaria prophylaxis as directed for the trip. His partner notices that as of the last 2-3 years he has had lapses in memory and is performing badly in his day-to-day tasks at work as a stoke broker such that he has now been made redundant. This has encouraged him to consult. On clinical examination, you find an emaciated gentleman with mild polyarthralgia and an ataxic gait. His Mini-Mental State Examination (MMSE) score is 22/30. Below are some of his blood test results:

Hb	90 g/l
Platelets	300 * 10 /l9
WBC	10 * 10 /l9
MCV	70 fL
Ferritin	8 micrograms/l
Na +	140 mmol/l
K +	2.9 mmol/l
Urea	11 mmol/l
Creatinine	110 µmol/l
Calcium	1.9 mmol/l
Magnesium	0.5 mmol/l
Rheumatoid factor	negative

You organise a small bowel biopsy. What abnormality is the histology likely to show?

A. Increased increased intraepithelial lymphocytes with villous atrophy and crypt Hyperplasia
B. Deposition of macrophages containing PAS-positive granules within villi
C. Transmural inflammation with multiple lymphoid aggregates
D. Adenocarcinoma
E. Carcinoid tumour cells with trabecular cell arrangements

B. Deposition of macrophages containing PAS-positive granules within villi

The onset of Whipple's disease features is insidious. Suspect it in persons with abdominal complaints, particularly malabsorption, plus neurological features. Incidentally, your differential for abdominal complaints and neurological features should include acute intermittent porphyria (AIP, which you can read about elsewhere on the site); but specifically, in this history, you would have expected a history of abdominal pain rather than malabsorption in AIP. The main point of the question is for you to recognise three key histological features they will commonly bring up in the exam in association with certain conditions, as below:

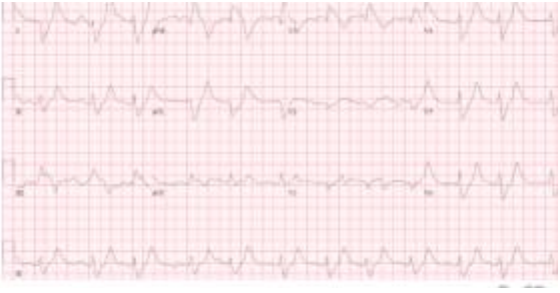
- Increased intraepithelial lymphocytes with villous atrophy and crypt hyperplasia = coeliac disease
- Deposition of macrophages containing PAS-positive granules within villi = Whipple's disease
- Transmural inflammation with multiple lymphoid aggregates = Crohn's disease

Question 52 of 86

A 55-year-old man attends the emergency department following a collapse at a family barbecue. Prior to the collapse, he complained of feeling dizzy with a fluttering feeling in his chest. He has a past medical history of hypertension for which he takes amlodipine and ramipril.

On examination, he is alert and oriented. Peripheral pulses are intact bilaterally and his lying and standing blood pressures are 155/89mmHg and 147/80mmHg, respectively. On auscultation, his chest is clear and heart sounds are normal. His neurological examination is unremarkable, although he still complains of feeling dizzy.

An ECG is taken, as shown below:



What is the next most immediate step in the management of this patient?

A. Amiodarone
B. Calcium gluconate
C. Synchronised electrical cardioversion
D. Transcutaneous pacing
E. Transfer to percutaneous coronary intervention

B. Calcium gluconate

This patient's ECG shows peaked T waves and QRS widening in the majority of the leads in keeping with hyperkalaemia. Additional ECG findings include P wave flattening, PR interval prolongation, conduction blocks and bradyarrhythmias. Hyperkalaemia can cause collapse and dizziness as experienced by this patient. Furthermore, the medication history of ramipril puts this patient at a higher risk of developing hyperkalaemia. The treatment of choice is an infusion of calcium gluconate to stabilise the cardiac membrane.

Amiodarone is used in the treatment of tachyarrhythmias including atrial fibrillation and ventricular tachycardia. Whilst this patient's ECG features broad QRS complexes, this patient is not tachycardic with a heart rate of around 66bpm. Furthermore, the significantly peaked T waves are not suggestive of a tachyarrhythmia. Rather, they indicate hyperkalaemia.

Synchronised electrical cardioversion is used in the management of tachyarrhythmias with adverse features (e.g. shock, myocardial ischaemia, heart failure). Not only does this patient's ECG not represent a tachyarrhythmia, but there are also no adverse features at present and this patient is haemodynamically stable.

Transcutaneous pacing would be indicated if this patient's ECG demonstrated a bradyarrhythmia, particularly one with a significant risk factor for asystole (e.g. Mobitz type II or complete heart block). It does not play a role in the treatment of hyperkalaemia.

Although there is an ambiguous QRS morphology on this patient's ECG, there is no clear STsegment elevation. Instead, there are significantly peaked T waves that may give a false impression of ST-segment elevation. With this in mind, it is unlikely that this patient would benefit from primary percutaneous coronary intervention.

Question 53 of 86

An 80 year-old man presents with a 3 month history of generalised abdominal pain and a change in bowel habit. The abdominal pain is colicky in nature and does not radiate anywhere. He also reports increasing distension of his abdomen and 2 episodes of blood in the rectum several weeks ago. He has recently undergone colonoscopy, which did not reveal anything abnormal. His past medical history includes diabetes type 2 and a heart attack two years ago, for which he needed three stents. His mother died of a stroke when he was 60 and his father died of a heart attack at the age of 55. His current medications include ramipril, aspirin, atenolol, atorvastatin and metformin. He has a 40 year pack history and drinks on average 15 units per day.

Blood tests reveal:

Hb	12.0 g/dL
Mean corpuscular volume (MCV)	80 fl
Platelets	198 * 10 /l9
WBC	13.1 * 10 /l9
Na^+	132 mmol/l
K^+	5.1 mmol/l
Urea	9.0 mmol/l
Creatinine	145 µmol/l

Other than an abdominal x-ray, what is the most appropriate investigation?

A. Contrast-enhanced computed tomography (CT) of the abdomen with angiography
B. Colonoscopy
C. Endoscopy
D. MRI of the abdomen
E. Angiography

A. Contrast-enhanced computed tomography (CT) of the abdomen with angiography

Given this man's cardiac history, age and the length of clinical features, the most likely diagnosis is chronic mesenteric ischaemia - the cause is probably atheroma of the mesenteric vessels, as opposed to embolism. The patient's creatinine levels start to rise in response to bowel wall ischaemia, hence the apparent affect on renal function.

A plain-film abdominal x-ray is often valuable in excluding certain abdominal conditions, although CT of the abdomen with angiography is the preferred investigation. Angiography gives information regarding a proximal defect of a mesenteric vessel or vasoconstriction of all mesenteric arcades and the CT scan provides information on bowel wall thickening, bowel dilation and the classic thumb-printing sign suggestive of submucosal oedema or haemorrhage.

Question 54 of 86

An 81 year-old woman presents with a 2 year history of dysphagia for both solids and liquids. She now has to chew her food a lot before swallowing and often has to drink water with every bite. Frequently she coughs after eating and has experienced pain on several occasions behind her sternum when swallowing.

Barium swallow reveals narrowing at the gastro-esophageal junction, producing a 'bird's beak' appearance.

What is the most appropriate medical management for this condition?

A. Ramipril
B. Nifedipine
C. Losartan
D. Stenting
E. Bisoprolol

B. Nifedipine

The diagnosis in this case is achalasia, which characterized by difficulty in swallowing, regurgitation of food and liquids and episodes of chest pain. Diagnosis is achieved via oesophageal manometry and barium swallow radiographic studies. The most common form of achalasia is primary achalasia, which has no known cause. Medical treatment includes drugs that help reduce the lower oesophageal pressure, such as calcium channel blockers (e.g. nifedipine) or nitrates (isosorbide dinitrate and nitroglycerin).

Question 55 of 86

A 40 year-old man is referred by his GP with a two-day history of severe abdominal pain located in the lower left and lower right quadrants. Other symptoms involve diarrhoea, malaise and weight loss. His past medical history includes Crohn's disease, for which he has recently been treated with mycophenolate mofetil for the past 4 months, however this is starting to lose its effect. Other treatments for the Crohn's disease include steroid therapy, 6-mercaptopurine and a course of azathioprine, although all have failed to control his disease. Surgical history includes an ileocaecal resection 7 years ago.

Blood test results reveal:

Hb	9.8 g/L
Platelets	190 * 10 /l9
WBC	14.9 * 10 /l9
Na +	135 mmol/l
K +	3.8 mmol/l
Urea	6.8 mmol/l
Creatinine	90 µmol/l
CRP	58 mg/l
ESR	64 mm/hr

This episode is diagnosed as a severe flare-up of his Crohn's disease. What is most appropriate next management?

A. Oral ciclosporin
B. Intravenous infliximab
C. Beta-interferon therapy
D. Oral Sulfasalazine
E. Intravenous cyclophosphamide

B. Intravenous infliximab

Infliximab should be considered (and is licensed) for corticosteroid or immunomodulator refractory disease or intolerance to the above drugs. It works as a monoclonal antibody to the cytokine tumour necrosis factor (TNF) alpha.

The National Institute for Health and Clinical Excellence (NICE) recommends infliximab for severe active or active fistulising Crohn's disease until treatment failure (including surgery) or for 12 months, whichever is the shorter. Treatment should only be continued after that if there is evidence of active disease. Adalimumab can be used as an alternative.

Question 56 of 86

A 40-year-old man is referred by his GP to the outpatient department with a 4 month history of tiredness and itching. He currently sleeps excessively during the daytime and reports he is having trouble concentrating at work, where he is an auditor. The itching becomes worse when he is in hotter climates and if he sleeps with a blanket over him. His past medical history includes asthma and dry eyes. His regular medications include beclometasone and antihistamine that he takes for the itching. He is not allergic to any medication and there is no family history of any known conditions. He does not smoke and drinks 5-10 units of alcohol per week.

Examination reveals mild jaundice of the sclera and scratch marks over his upper torso and arms. On examination of his abdomen, he has a slightly enlarged spleen and liver, both by 1cm.

Blood tests reveal raised bilirubin and alkaline phosphatase (ALP). Autoantibody screening reveals antimitochondrial antibodies (AMA) and antinuclear bodies (ANA) both being present.

What is the most appropriate management to help control his symptoms?

A. Hydroxyzine
B. Cetirizine Hydrochloride
C. Ursodeoxycholic acid
D. Fexofenadine
E. Corticosteroid therapy

C. Ursodeoxycholic acid

The most likely diagnosis in this case is primary biliary cirrhosis, as evidenced by the patients severe tiredness, itching and positive autoantibodies. The ratio of male:female is around 1:9, however it is important to remember this disease does affect men as well. Treatments are aimed at alleviation of symptoms and at slowing the disease, although liver transplant can offer a cure. Ursodeoxycholic acid has been shown to slow progression of the disease and improve the symptoms of jaundice.

Question 57 of 86

A 54 year old gentleman who is a known heavy drinker, presents with hematemesis.

What is recognised as the most effective treatment strategy, at the point of endoscopy, for oesophageal varices?

A. Proton pump inhibitors
B. Endoscopic banding
C. Transjugular intrahepatic portosystemic shunt
D. Endoscopic sclerotherapy
E. Transexamic acid

B. Endoscopic banding

Management of acute upper gastrointestinal bleeding secondary to varices can be divided up into acute vs prophylactic.

- Resuscitate the patient!. Transfuse with blood, platelets, clotting factors in line with local protocols. Nice advise only using platelets if the platelet count is <50x10^9/Litre.
- Offer fresh frozen plasma for individuals with a fibrinogen level of < 1g/Litre or a prothrobin time or activated partial thromboplastin time > 1.5 times the norm.
- In patients who are on warfarin, consider prothrombin complex concentrate.
- Vasoactive agents such as Terlipressin is the only licensed vasoactive agent. It has been shown to be of benefit in initial hemostasis and preventing rebleeding. It acts by constriction of the splanchnic vessels (contraindicated in IHD, use Octreotide as alternative).
- Prophylactic antibiotics have been shown to be of benefit in patients with liver cirrhosis.
- Variceal banding is superior to sclerotherapy at endoscopy.
- Only consider a Sengstaken-Blakemore tube for uncontrolled haemorrhage and where access to endoscopy is delayed/difficult*
- Transjugular intrahepatic portosystemic shunt if the above measures fail.

Prophylaxis:

- Propranolol
- endoscopic band ligation at regular intervals, proton pump inhibitors are given at this stage to prevent ligation induced ulceration.

Common causes of upper gastrointestinal bleeding:

- peptic ulcer disease
- gastritis
- oesophagitis
- Mallory-Weiss tears
- oesophageal/gastric varices

*Many hospitals do not run an out of hours endoscopy service, candidates are encouraged to familiarise themselves with the correct use of a Sengstaken-Blakemore tube as it may be the ONLY option when someone presents at 4am!.

Question 58 of 86

A 42-year-old woman attends the gastroenterology clinic for outpatient review approximately one month following a recent admission to hospital. She had presented with a 6 week history of frequent bloody diarrhoea and cramping abdominal pain. In addition, she reported a longstanding tendency to suffer from multiple mouth ulcers. She had lost approximately, 8 kilograms of weight between the onset of diarrhoea and presentation to hospital.

The results of investigations performed during the hospital admission are given below. Following investigation, a short course of intravenous steroids had been given followed by oral prednisolone (initial dose 40 mg daily, slowly tapered over 8 weeks).

The patient reported near complete resolution of her previous symptoms and welcome relief from her oral ulceration. She had regained some of the weight she had previously lost and reported her appetite to be good. A discussion was had with the patient about future therapy and she indicated that she would prefer to consider the initiation of further medication to attempt to maintain her symptom remission.

Stool microscopy: no organisms seen

Stool culture: no organisms grown

Colonoscopy: patchy inflammation with 'cobblestone' appearance affecting ascending and transverse colon and terminal ileum

Colonic histology: multiple samples demonstrating signs of chronic transmural inflammation, crypt abscesses and submucosal fibrosis

CT abdomen: no evidence of intra-abdominal collection, structuring or abnormal fistulation

What is the most appropriate medication to maintain disease remission in this patient?

A. Methotrexate
B. Infliximab
C. Budesonide
D. Azathioprine
E. Metronidazole

D. Azathioprine xt question

The patients presentation and investigation results are consistent with a new diagnosis of Crohns disease. She appears to have had a good response to initial treatment with corticosteroids, however introduction of an immunomodulating agent to maintain remission and limit steroid exposure is appropriate, especially given the patients risk factors for a severe Crohns disease phenotype (female sex; need for steroids to treat first flare).

Once remission is obtained then a thiopurine such as azathioprine or mercaptopurine is first line treatment to maintain remission.

Methotrexate is an alternative agent but tends to be less well tolerated than thiopurines.

Biological therapies such as infliximab are considered to maintain remission only after failure of treatment with the immunomodulating agents above. Budesonide is an oral corticosteroid and is therefore not an appropriate choice for maintenance of remission. Metronidazole has efficacy in perianal Crohns disease only.

Question 59 of 86

A 25-year-old gentleman presents with bloody diarrhoea, fevers and lower abdominal pain. An initial colonoscopy shows extensive ulceration in his distal colon with some mild active inflammation. His dad developed colorectal cancer at the age of 52. Following the procedure the gastroenterologist discusses the necessity of colonic surveillance.

What is the risk that he will develop colorectal cancer?

A. Low risk (offer colonoscopy at 5 years)
B. High risk (offer colonoscopy at 1 year)
C. Intermediate risk (offer colonoscopy at 3 years)
D. Too early to ascertain
E. No risk

C. Intermediate risk (offer colonoscopy at 3 years)

In anyone with inflammatory bowel disease, be it Crohn's or ulcerative colitis, a baseline colonoscopy with chromoscopy and target biopsy is necessary to determine the risk of developing colorectal cancer.

According to NICE guidance on the colonoscopic surveillance of colorectal cancer, this patient fulfils intermediate risk: extensive ulcerative or Crohn's colitis with mild active inflammation (confirmed endoscopically or histologically) or post-inflammatory polyps or family history of colorectal cancer in a first-degree relative aged 50 or over.

Question 60 of 86

A 26-year-old man comes to the outpatient clinic. He describes a 2-month history of weight loss, cramping lower abdominal pain with increasing stool frequency. His stools often have blood and mucus in them and occasionally he passes pure blood with no faeces. He is normally fit and well and takes no regular medications. On examination, he has a low-grade fever at 37.5 and pale. His abdomen is soft but tender across the lower half. His bloods checked by the GP earlier in the week are as follows:

Hb	110 g/l
Platelets	400 * 10 /l9
WBC	12.0 * 10 /l9
Neuts	9.0 * 10 /l9
Na +	139 mmol/l
K +	4.5 mmol/l
Urea	4.0 mmol/l
Creatinine	89 µmol/l
CRP	60 mg/L (<10)
Bilirubin	8 µmol/l
ALP	78 u/l
ALT	34 u/l
Albumin	36 g/l

Stool cultures sent by the GP are negative.

He is admitted the same day for a flexible sigmoidoscopy which shows mild colitis extending to the mid-descending colon. Biopsies are taken which are show mild colitis but of indeterminate cause.

He is transferred to the ward and started on IV hydrocortisone 100mg QDS and he clinically improves and is started on regular mesalazine. He is found to be positive for anti-Saccharomyces cerevisiae antibodies but negative for pANCA

. What is the likely cause of his colitis?

A. Ulcerative colitis
B. Microscopic colitis
C. C.difficile infection
D. Behcet's disease
E. Crohn's disease

E. Crohn's disease ion

This patient has Crohn's disease. Anti-Saccharomyces cerevisiae antibodies are more likely to be positive in Crohn's disease. Ulcerative colitis patients are more likely to be pANCA positive.

The patient has colitis on flexible sigmoidoscopy, excluding microscopic colitis which has a normal appearance on endoscopy.

Behcet's usually present in Middle Eastern men with oral and genital ulcers, conjunctivitis and colitis.

Question 61 of 86

A 72-year-old man was diagnosed with colorectal cancer after a positive faecal immunochemical test (FIT) and microcytic anaemia on a routine health check. Following further investigations and resection, he is found to have an adenocarcinoma confined to the mucosa and submucosa.

What is his 5-year survival based on his Dukes' stage?

A. 95%
B. 75%
C. 65%
D. 50%
E. 30%

A. 95%

This patient has Dukes' A bowel cancer which means he has a 95% 5-year survival. Please see the table below for further information on Dukes' stages and survival rates.

Dukes' classification and 5-year survival rates

Stage	5-year survival
A - confined to mucosa and submucosa	95% in men and 100% in women
B - extends through the muscularis propria	> 80% in men and 90% in women

C - regional lymph nodes involved	65% in men and 65% in women
D - distant spread	> 5% in men and 10% in women

Question 62 of 86

A 50-year-old man with known alcoholic hepatitis is admitted to the Emergency Department generally unwell. He has had vague abdominal pain and general malaise for 4 days. His wife tells you he has been eating and drinking much less in that time and confirms he has been abstinent from alcohol for over a year.

On examination he appears clinically dehydrated and is drowsy. His heart rate is 121 beats per minute and his blood pressure is 102/55 mmHg. His temperature is 37.4 ºC. His chest is clear. His abdomen is soft with suprapubic tenderness but no organomegaly. There is some dullness in the flanks.

His urine dip is positive for nitrites and 1+ leucocytes. His blood tests are as follows:

Hb	115 g/l	Na +	125 mmol/l	Bilirubin	24 µmol/l
Platelets	189 * 10 /l 9	K +	4.9 mmol/l	ALP	250 u/l
WBC	14 * 10 /l 9	Urea	11 mmol/l	ALT	124 u/l
Neuts	10 * 10 /l 9	Creatinine	230 µmol/l	γGT	255 u/l
Lymphs	2.5 * 10 /l 9	CRP	75 mg/dl	Albumin	30 g/l

He is treated with antibiotics and normal saline but after 24 hours his sodium is 124 mmol/l and creatinine is 229 µmol/l.

His urine sodium is 12 mmol/l and an ultrasound of the abdomen shows mild ascites, a cirrhotic liver and a normal renal tract.

What is the next most appropriate step?

A. 5% dextrose
B. Fluid restriction
C. Hartmann's solution
D. Human albumin solution
E. Hypertonic saline

D. Human albumin solution

This gentleman has hepatorenal syndrome secondary to his alcoholic liver disease. This is evidenced by his renal failure with hyponatraemia, low urine sodium and poor response to rehydration with normal saline.

Current guidelines recommend the next most appropriate step is rehydration with human albumin solution.

Question 63 of 86

You review a 30-year-old male who was seen in the gastroenterology department with a first presentation of ulcerative colitis symptoms He was having diarrhoea 5 times a day with small amount of blood in his stool. He had been systemically well and haemodynamically stable and thus did not require hospital admission. He was started on daily Pentasa.

Today, he reports his symptoms have not improved despite treatment. He is still opening his bowels 5 times per day and is concern about it.

He is mildly dehydrated clinically, but haemodynamically stable, with a blood pressure of 120/76 mmHg and a heart rate of 70 beats per minute. His abdomen is soft and not distended.

What is the most appropriate management?

A. Stop 5 ASA and start infliximab
B. Add prednisolone oral therapy
C. Topical 5 ASA
D. Oral ciclosporin
E. Oral mercaptopurine

B. Add prednisolone oral therapy

This gentleman has symptoms of moderate ulcerative colitis which was not managed with oral 5 ASA. He is not on topical therapy following an initial assessment which gives you the clue that his disease is not isolated to proctitis.

His symptoms are still that of moderate ulcerative colitis and given his failure to respond to treatment he should now be started on oral prednisolone with a follow up in 2 weeks for re-review of symptoms.

Mild to moderate ulcerative colitis (up to 6 motions per day with mild bleed. ESR <30) should be managed using a stepwise approach.

- Proctitis: Induce remission with a topical 5-ASA (aminosalicylic acid). If no response or cant tolerate, 5-ASA the next step would be to add topical steroids. Oral should be considered if remission not achieved or patient presents with left-sided extensive ulcerative colitis.
- Left-sided extensive disease should be managed using oral 5-ASA initially with or without topical 5-ASA. If no improvement within 4 weeks oral prednisolone therapy should be added. If no response to oral prednisolone, tacrolimus should be started to induce remission.

Question 64 of 86

A 42-year-old woman has been referred due to persistent iron deficiency anaemia despite oral iron supplements. She has a history of menorrhagia, depression, and gastro-oesophageal reflux disease.

Medications include ferrous fumarate, tranexamic acid, sertraline, and amitriptyline. She occasionally takes over-the-counter paracetamol, ibuprofen, and an antacid solution.

Recent investigations have been normal including endoscopy and imaging. Up-to-date blood tests confirm ongoing iron deficiency anaemia.

What is the likely cause for her ongoing iron deficiency?

A. Amitriptyline
B. Antacid solution
C. Ibuprofen
D. Sertraline
E. Tranexamic acid

B. Antacid solution

Oral iron supplement absorption may be impaired by calcium, zinc or magnesium containing gastric coating suspensions causing treatment failure. An alternative route of administration should be considered

Antacid solutions, such as peptac, act by coating the stomach with an alkaline solution containing magnesium and calcium, thus improving heartburn symptoms. Unfortunately, this can lead to failed absorption of oral iron replacement and is, therefore, the correct answer.

Amitriptyline does not affect oral iron absorption.

Ibuprofen can increase the risk of gastrointestinal (GI) ulceration which may lead to iron deficiency via bleeding, however, recent endoscopy is normal and so this is unlikely to be the cause.

Sertraline, as with ibuprofen, carries an increased risk of GI ulceration and therefore may lead to iron deficiency via bleeding, however again, recent endoscopy is normal and so this is unlikely to be the cause.

Tranexamic acid is used to control heavy bleeding but is not associated with impaired iron absorption.

Question 65 of 86

A 75 year old women was referred for urgent upper gastrointestinal endoscopy after a two month history of recurrent post-prandial vomiting. She also reported a 10 kg weight loss over a similar period. Examination revealed cachexia but was otherwise unremarkable. Past medical history includes hypertension and a right-sided carotid endarterectomy following a transient ischaemic attack 8 years previously. Regular medications are bendroflumethiazide, clopidogrel and simvastatin. The patient is a retired schoolteacher has never smoked and drinks minimal amounts of alcohol.

Endoscopy demonstrated a 3 cm ulcer in the body of the stomach. Histology confirmed adenocarcinoma with penetration of the tumour into the subserosal connective tissue. The results of further staging investigations are given below.

CT abdomen: 3 cm mass in body of stomach along greater curve of stomach; enlargement of the right and left gastro-epiploic lymph nodes; liver, gallbladder, spleen and kidneys unremarkable.

PET: no evidence of distant metastasis or nodal involvement Staging laparoscopy: no evidence of peritoneal or metastatic disease

What is the appropriate initial treatment for this patient?

A. Neoadjuvant chemotherapy
B. Total gastrectomy
C. Partial gastrectomy
D. Endoscopic mucosal resection
E. Palliative radiotherapy

A. Neoadjuvant chemotherapy

This patient's investigations indicate that they have T3N1M0 gastric carcinoma (Stage IIB disease). Standard of care within the UK for stage II / III disease would be for neoadjuvant chemotherapy prior to radical surgery. The MAGIC trial demonstrated significant improvement in 5 year survival for three cycles of pre-operative chemotherapy followed by three cycles of post-operation chemotherapy compared to surgery alone (36 % vs 23 %; P = 0.009).

Primary surgical resection is only appropriate for stage 1 tumours. Endomucosal resection can be considered in some early gastric cancers (T1a).

Extent of surgical resection depends on tumour location with subtotal gastrectomy feasible for some distal tumours.

Curative treatment is typically not possible in tumours with local invasion (T4) or distant metastasis (M1).

Question 66 of 86

A 60-year-old lady on the gastroenterology ward develops an acute episode of haematemesis in the night, the nurse who witnessed the event described the bleed as about one cupful of bright red blood. The patient has a known history of alcohol abuse, drinking 2 bottles of wine a day, and has a past medical history of ascites secondary to alcoholic liver cirrhosis, hypertension and type two diabetes mellitus. She was admitted initially for an ascitic drain earlier in the afternoon which has not occurred due to staff shortages in the medical team.

On examination she was afebrile, her heart rate was 110bpm, blood pressure was 104/81mmHg, respiratory rate of 20 breaths per minute and had an oxygen saturation of 97% on air. Cardiovascular and respiratory examination was unremarkable. Abdominal examination revealed a tensely distended abdomen with shifting dullness present. There are marked distended superficial veins on her abdominal surface. There was mild epigastric tenderness, and on rectal examination, there was a small amount of tarry black stool. She was not actively vomiting during the examination.

Intravenous fluid resuscitation was already started by the house officer on call, and blood samples including a cross-match were sent. The gastroenterology registrar on call has been informed and was arranging an emergency endoscopy for the patient.

Her previous blood results and a current venous blood gas (VBG) results are shown: Blood results (earlier in the afternoon)

Na+	134 mmol/l
K+	4.8 mmol/l
Urea	10.9 mmol/l
Creatinine	100 µmol/l
Serum bilirubin	30 µmol/l
Serum alkaline phosphatase	165 IU/l
Serum aspartate aminotransferase	68 IU/l
C Reactive protein	6 mg/l
Haemoglobin	126 g/l
White cell count	7.6 x 10^9/L
Platelets	122 x 10^9/L
INR	1.8

VBG (Current)

pH	7.368
Lac	1.8 mmol/l
Base Excess	-2.4 mmol/l
Bicarbonate	26.9 mmol/l
Hb	11.0 g/dL

What is the next most appropriate immediate course of action to take?

A. Begin transfusion of O-negative blood
B. Intravenous proton pump inhibitor (PPI) bolus
C. Intravenous terlipressin bolus
D. Platelet transfusion
E. Intravenous ciprofloxacin

C. Intravenous terlipressin bolus

This clinical picture is strongly suspicious for a variceal upper gastro-intestinal (GI) bleed, and the principles of management should be initial resuscitation with intravenous fluids with two large bore cannulas. A blood transfusion should be arranged, however, although the patient is tachycardic she is still maintaining her blood pressure and the VBG result suggests she could wait for a cross-matched sample to be ready instead of requiring immediate O-negative blood which is limited in supply. An endoscopy should be offered immediately after resuscitation to all unstable patients for band ligation of the bleed.

NICE guidelines for variceal bleeds recommend both intravenous terlipressin and antibiotic prophylaxis prior to endoscopy. Of the two, there is a significant improvement in the rate of patients achieving initial haemostasis in patients treated with terlipressin due to its vasopressive effect in decreasing portal hypertension and would be the more appropriate drug to give first. Terlipressin should be stopped after definitive haemostasis has been achieved, or after 5 days of therapy. As this patient has a platelet count of greater than 50 x 109/L a platelet transfusion is not indicated, however, she is coagulopathic and would benefit from both vitamin K and fresh frozen plasma.

Intravenous proton pump inhibitors have a limited evidence base in the pre-endoscopy management of upper GI bleeds in decreasing the mortality from variceal bleeds, and are not included in the current NICE guidelines for the management of variceal bleeds.

Question 67 of 86

A 23-year-old man presents with four months of abdominal bloating and diarrhoea. He is opening his bowel 2-3 times a day. He also gets recurrent aphthous ulcers, but denies weight loss and has never passed mucus or blood in his stools. He has not travelled anywhere in the last year and there is no family history of inflammatory bowel disease.

On examination, his abdomen was soft with no organomegaly.

Coeliac serology is negative.

What is the next appropriate test?

A. Faecal calprotectin
F. Pylori serology
B. Faecal elastase
C. Sigmoidoscopy
D. Small bowel capsule endoscopy

A. Faecal calprotectin

Faecal calprotectin is a highly sensitive and specific test for inflammatory bowel disease

Faecal calprotectin is increased in inflammatory conditions of the bowel including crohns disease, ulcerative colitis, infectious colitis and malignancy. The faecal calprotectin level is normal in irritable bowel syndrome the most likely diagnosis in this case. If normal, the patient would not require further investigation such as a sigmoidoscopy or colonoscopy.

Faecal elastase is used to assess pancreatic exocrine function in pancreatic insufficiency.

Sigmoidoscopy would be an alternative, more invasive approach to investigate this gentlemans diarrhoea. However, if negative would not rule out inflammatory bowel disease as it only visualises the sigmoid.

A large proportion (around 20%) of the general population suffers from aphthous ulcers.

H.pylori serology would indicate whether the patient has been previously exposed to Helicobacter pylori; however it is associated with upper gastrointestinal symptoms.

Small bowel capsule endoscopy is used to visualize areas of the bowel unreachable by conventional endoscopy. This would not be used without initially performing a colonoscopy to rule out large bowel pathology.

Question 68 of 86

A 21-year-old woman presents to gastroenterology clinic with a 6 months history of abdominal bloating and diarrhoea. She is a university student and finds that her symptoms are exacerbated by periods of stress. She does not take any regular medications and has no drug allergies. Her uncle was diagnosed with ulcerative colitis at the age of 30 and is currently on biologic therapy for it.

On examination, her temperature is 36.7ºC, blood pressure is 100/50 mmHg and heart rate is 90 beats per minute. Her body mass index is 20.5 kg/m². There were no abnormalities to find on an examination of her cardiovascular, respiratory or abdominal systems.

Her GP had organised some blood tests prior to referring her to clinic:

Hb	130 g/l	Na +	135 mmol/l	Bilirubin	12 µmol/l
Platelets	280 * 10 /l 9	K +	4.6 mmol/l	ALP	85 u/l
WBC	6.5 * 10 /l 9	Urea	3.5 mmol/l	ALT	40 u/l
Neuts	4.0 * 10 /l 9	Creatinine	50 µmol/l	γGT	35 u/l
CRP	<1 mg/l	Albumin	40 g/l		

Which is the next most appropriate investigation for this patient?

A. Faecal elastase
B. Colonoscopy
C. Flexible sigmoidoscopy
D. Faecal calprotectin
E. CT abdomen

D. Faecal calprotectin

Faecal calprotectin is a highly sensitive and specific test for inflammatory bowel disease

This young patient has features of diarrhoea-predominant IBS. Patients with IBS often find that their symptoms are exacerbated by stress. Although there is some evidence patients with IBD may have a genetic predisposition, it does not follow a mendelian pattern of inheritance. Despite her family history of IBD, the nature of her symptoms and normal blood tests make the diagnosis of IBD unlikely and it would not be appropriate to proceed with a lower GI endoscopy or CT scan at this stage.

Faecal calprotectin is a test for intestinal inflammation and has been recommended by NICE as a screening tool for IBD for GPs. It can also be used to monitor the response to treatment in IBD patients.

Question 69 of 86

A 36-year-old man attends gastroenterology clinic with an 8-month history of watery diarrhoea. He opens his bowels up to 15 times a day and this is not associated with any rectal bleeding. His symptoms are not relieved by loperamide. He has a history of Crohn's disease which was diagnosed 5 years ago and had an ileal resection 12 months ago following an inflammatory stricture. He does not smoke and drinks approximately 35 units of alcohol a week. His medications include omeprazole 20mg OD, paracetamol 1g QDS and ibuprofen 400mg TDS PRN.

His blood tests are:

Hb	115 g/l	Na +	139 mmol/l	Bilirubin	12 µmol/l
Platelets	395 * 10 /l 9	K +	3.9 mmol/l	ALP	100 u/l
WBC	10.5 * 10 /l 9	Urea	6.5 mmol/l	ALT	42 u/l
Neuts	5.5 * 10 /l 9	Creatinine	100 µmol/l	γGT	65 u/l
CRP	4 mg/L	Albumin	37 g/l		
Amylase	124 U/L				

Which investigation is most likely to confirm the diagnosis?

A. Faecal elastase
B. CT abdomen
C. Faecal calprotectin
D. Secretin stimulation test
E. SeHCAT test

E. SeHCAT test

SeHCAT is the investigation of choice for bile acid malabsorption

Bile acid malabsorption can occur in patients with Crohn's disease affecting the ileum or following ileal resection. Bile salts are mostly absorbed in the ileum and the inability to do so results in chronic diarrhoea. Treatment with bile acid sequestrants such as cholestyramine is often effective. SeHCAT test is a form of nuclear imaging test which is used to investigate patients suspected of having bile acid malabsorption.

Question 70 of 86

A 55-year-old man with a history of alcohol excess is brought in by ambulance with coffee ground vomiting. He is drowsy and confused and unable to provide a history. Notes from previous admission state that he has a history of diabetes for which he takes metformin. On examination, he is thin, has spider naevi across the chest and has abdominal distension. Rectal examination confirms the presence of melaena.

His observations are as follows:

- Temperature 36.2
- Respiratory rate 25/min
- Saturations 95% on air
- Heart rate 125 bpm
- Blood pressure 80/40 mmHg

Hb	105 g/l	Na +	130 mmol/l	Bilirubin	30 µmol/l
Platelets	40* 10 /l 9	K +	3.8 mmol/l	ALP	200 u/l
WBC	13.2* 10 /l 9	Urea	15.3 mmol/l	ALT	98 u/l
Neuts	10.0* 10 /l 9	Creatinine	86 µmol/l	Albumin	32 g/l
Lymphs	1.0* 10 /l 9	INR	1.8		
Eosin	0.3* 10 /l 9	APTT	50	Fibrinogen	1.4 g/l

What medical therapy should be urgently instituted prior to endoscopy?

A. Red blood cell and platelet transfusion
B. Platelet transfusion, fresh frozen plasma, terlipressin and tazocin
C. Red blood cell and platelet transfusion, fresh frozen plasma, terlipressin and tazocin
D. Red blood cell transfusion, fresh frozen plasma, terlipressin and tazocin
E. Platelet transfusion and fresh frozen plasma

B. Platelet transfusion, fresh frozen plasma, terlipressin and tazocin

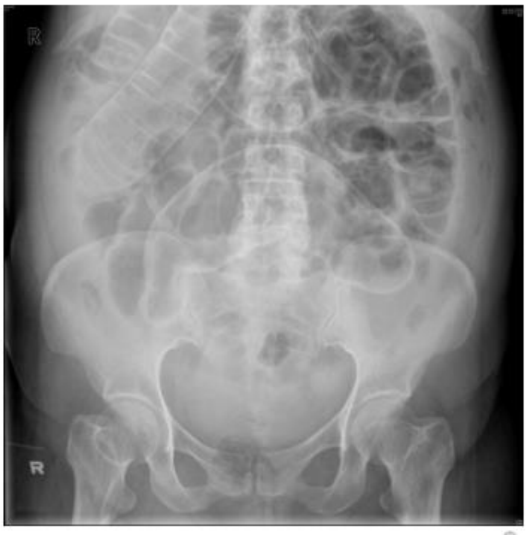
This patient has UGI bleeding and haemodynamic compromise. He needs urgent endoscopy once stabilised. The history of alcohol excess and examination findings mean variceal bleeding should be suspected.

This patient should therefore be managed as an acute upper GI variceal bleed according to NICE guidelines (updated August 2016) and receive terlipressin and broad spectrum antibiotics. Platelets are indicated as count is below 50 and FFP is indicated due to raised INR/APTT > 1.5 x normal. Cryoprecipitate is only required if fibrinogen remains below 1.5 after FFP. Red cell transfusion should follow local guidelines but with Hb>100 is certainly not indicated at this stage.

Question 71 of 86

An 80-year-old woman is admitted from a nursing home with a one day history of severe abdominal pain. She has a background of chronic obstructive pulmonary disease and recently completed a course of amoxicillin and prednisolone for an exacerbation.

On examination she is tender over her whole abdomen. Blood pressure is 98/66 mmHg and her pulse is 110/min. The abdominal radiograph is shown below:



What is the most likely diagnosis?

A. Intestinal perforation
B. Sigmoid volvulus
C. Leaking abdominal aortic aneurysm
D. Chronic pancreatitis
E. Clostridium difficile infection

A. Intestinal perforation

This lady had evidence of free air in the abdomen, caused by an intestinal perforation. Please see the annotated radiograph below for more details.

Question 72 of 86

A 33-year-old lady was referred to the outpatient gastroenterology clinic by her GP who had diagnosed Irritable Bowel Syndrome refractory to symptomatic management. Her symptoms commenced six months ago with central abdominal cramping bloating made worse with eating wheat and oat based foods. She also noticed that the onset of the abdominal cramping was often associated with faecal urgency. Despite a prolonged trial of mebeverine, hyoscine butylbromide and peppermint oil there was no improvement in her symptoms. Her symptoms were however alleviated by avoiding the precipitating foods. She had not lost any weight and did not pass any blood per rectum; other than a family history of maternal depression there was no family history of note. Her past medical history was comprised depression for which she was prescribed citalopram 40mg OD; this was increased recently following worsening of her mood over the last few weeks.

Examination revealed the presence of a well 33-year-old lady. Her blood pressure was 112/72mmHg, heart rate 78 bpm and temperature 36.6ºC. Examination of her cardiovascular and respiratory systems was unremarkable. Examination of her gastrointestinal system was similarly unremarkable with no organomegaly, normal bowel sounds and the absence of lymphadenopathy. Examination of her forearms revealed the presence of multiple superficial lacerations.

Investigations conducted by her GP are as shown:

Hb	105 g/l
MCV	81 fl
Platelets	224 * 10 /l9
WBC	8.2 * 10 /l9
Bilirubin	19 µmol/l
ALP	142 u/l
ALT	33 u/l
Protein	81 g/l
Albumin	39 g/l
ESR	9 mm/hr
CRP	12 mg/l
TSH	0.75 (NR 0.4-3.6mu/ml)
FT4	11.6 (NR 4.5-13.6 mcg/dl)
IgG	7.2 (NR 5.8-15.4g/l)
IgA	0.2 (NR 0.64-2.97g/l)
IgM	1.2 (0.75-2.20g/l)
B12	224 (NR 160-900 ng/l)
Anti-endomysial antibody	negative

What is the next most appropriate management option?

A. Organise oesophageal motility studies
B. Organise upper GI endoscopy and biopsy
C. Commence trial with low dose amitriptyline
D. Commence gluten exclusion diet
E. Commence trial with domperidone

B. Organise upper GI endoscopy and biopsy

This lady has a history strongly suggestive of coeliac disease which GP the erroneously diagnosed as IBS; a common mistake to make. Of note her anti-endomysial antibody screening test was negative. This test has a sensitivity rate of 90% but of significance here is the incidental finding of selective IgA deficiency; as anti-endomysial antibody is IgA based, deficiency of IgA will result in a false negative finding and therefore this test has no negative predictive capability in this scenario. The next step is therefore to proceed to OGD and biopsy; whilst a gluten exclusion diet will aid her symptoms it is important to make a definitive diagnosis where possible.

Question 73 of 86

A 42-year-old bar owner comes to the gastroenterology clinic for review. He has suffered from chronic diarrhoea which he says is difficult to flush away, coupled with anorexia and epigastric abdominal pain over the past few months. He claims to have lost 8 kg in weight over the past year. He is a non-smoker who drinks at least 32 units of alcohol per week. Examination reveals a blood pressure of 100/82 mmHg, his pulse is 80 beats per minute and regular. You note a number of spider naevi on examination of his chest and abdomen. He has no abdominal masses and his body mass index is 22 kg/m².

Investigations:

Hb	98 g/l
Platelets	101 * 10 /l9

WBC	5.9 * 10 /l9
Na +	134 mmol/l
K +	3.6 mmol/l
Urea	5.1 mmol/l
Creatinine	82 µmol/l
Bilirubin	12 µmol/l
ALP	168 u/l
ALT	64 u/l
γGT	134 u/l
Albumin	27 g/l

Which of the following tests is most likely to reveal the cause of his diarrhoea?

A. Serum amylase
B. Faecal calprotectin
C. Faecal elastase
D. Anti-tissue transglutaminase antibody testing
E. D-xylose absorption testing Next question

C. Faecal elastase

Faecal elastase is the most useful initial investigation in patients with possible chronic pancreatitis to determine exocrine pancreatic insufficiency

This patient is a bar owner, and he is potentially under-estimating his alcohol consumption. The chronic epigastric pain, weight loss and diarrhoea are suggestive of malabsorption and this is likely to be related to chronic pancreatitis and subsequent exocrine pancreatic insufficiency. Faecal elastase measurement is a direct indicator of the ability to produce pancreatic enzymes. Normal elastase values are usually considered to be greater than 200 mcg/g of stool, 100-200 may indicate mild exocrine insufficiency, less than 100 is indicative of pancreatic exocrine insufficiency.

Serum amylase is usually only usually elevated in episodes of acute pancreatitis, it isn't therefore useful here. Faecal calprotectin is a measure of GI tract inflammation, it's of value in evaluation of inflammatory bowel disease, not in chronic pancreatitis. D-xylose absorption testing is used to assess GI tract membrane integrity, and is normal in patients with chronic pancreatitis. Anti-tissue transglutaminase antibody testing is the screening test of choice for coeliac disease. Although this is a cause of malabsorption, it isn't usually a cause of epigastric pain.

Question 74 of 86

A 23-year-old woman has been referred to the gastroenterology clinic. She has been found to have positive IgA tissue transglutaminase following investigations for chronic diarrhoea and weight loss. She has no other past medical history and takes no regular medications. As the diagnosis of coeliac disease was suspected she has omitted gluten over the last month

Which further investigation is recommended in confirming coeliac disease?

A. No further investigations are needed
B. HLA DQ2 testing
C. HLA DQ8 testing
D. Endoscopic intestinal biopsy urgently
E. Endoscopic intestinal biopsy after re-introducing gluten for six weeks

E. Endoscopic intestinal biopsy after re-introducing gluten for six weeks

Patients must eat gluten for at least 6 weeks before they are tested

The correct answer is endoscopic intestinal biopsy after re-introducing gluten for six weeks. This is a patient which symptoms matching coeliac disease who has tested positive with immunological studies. However, a diagnosis of coeliac disease requires a biopsy. To increase the diagnostic yield of the biopsy gluten should be re-introduced prior to the biopsy. HLA testing is only recommended in cases where biopsy would not be possible, but if the patient refuses biopsy it can be considered an option.

Question 75 of 86

A 24 year-old man presents to his GP because he is worried that he may have bowel cancer. He does not currently have any clinical features that would suggest bowel cancer and is taking no medication. His father died of colon cancer at the age of 49 and his paternal uncle also had bowel cancer diagnosed at the age of 44. He was previously referred for a colonoscopy four months ago which was normal.

His abdominal examination is normal. You rearrange for the patient to have a colonoscopy in 2 years time.

What is the most appropriate next management step?

A. Check for a mutation in chromosome 5q21
B. Check for a DNA mismatch repair gene mutation
C. Perform a computed tomography (CT) with contrast scan of his abdomen
D. Check carcinoembryonic antigen (CEA) level
E. Check for a P53 mutation

B. Check for a DNA mismatch repair gene mutation

The most likely diagnosis in this instance is hereditary non-polyposis colorectal cancer, given his strong family history. This accounts for roughly 2% of all colorectal cancers and is due to a mutation in the DNA mismatch repair gene. Once a diagnosis has been established, the preferred treatment is a colonoscopy every 1-3 years.

Question 76 of 86

A 27-year-old man has been troubled with diarrhoea for over 2 months. He reports passing watery, unformed stool, with some urgency at least 3 times per day. He also complains of mild abdominal pain but his main concern is the diarrhoea.

He has a background of Crohn's disease and has had an ileal resection. He has no other past medical history and he only takes mesalazine as maintenance for his Crohn's disease.

Results of tests he has had are shown below:

Hb	137 g/L	(135-180)
Platelets	275 * 10 /L 9	(150 - 400)
WBC	5.2 * 10 /L 9	(4.0 - 11.0)
Na +	137 mmol/L	(135 - 145)
K +	4.1 mmol/L	(3.5 - 5.0)

Urea	4.9 mmol/L	(2.0 - 7.0)
Creatinine	85 µmol/L	(55 - 120)
CRP	9 mg/L	(< 5)
Faecal culture	negative	
Faecal elastase	210µg	(>200)
Faecal calprotectin	45µg	(<50)

Given the likely diagnosis, what is the first line treatment for this patient?

A. Pancreatin (Creon)
B. Cholestyramine
C. Loperamide
D. Hydroxocobalamin
E. Prednisolone

B. Cholestyramine

Bile-acid malabsorption may be treated with cholestyramine

This patient is suffering from bile acid malabsorption secondary to the ileal resection he had for Crohn's disease. The ileum is the site where over 95% of bile acids are reabsorbed, therefore, loss of the ileum or non-functioning ileum due to disease means bile acids remain in the gastrointestinal tract, producing watery diarrhoea. The treatment for bile acid malabsorption is with bile acid sequestrants such as cholestyramine.

Pancreatic insufficiency such as in pancreatitis can also cause diarrhoea. The description of diarrhoea in this instance may be of foul-smelling stool that is difficult to flush (steatorrhoea) due to the fat content in the stool. This is usually treated with pancreatin (more commonly known as Creon). In this case, faecal elastase, which is a measure of pancreatic exocrine function, is normal, making pancreatic insufficiency unlikely, and so pancreatin is incorrect.

Loperamide is an anti-diarrhoeal that can be used for many causes of diarrhoea. However, from the presentation, bile acid malabsorption is the most obvious cause of his symptoms therefore cholestyramine should be used 1st line rather than loperamide.

Vitamin B12 deficiency is also a consequence of ileal resection as this is the site where it is absorbed. However, vitamin B12 deficiency would not give the presenting symptoms and the patient is not anaemic therefore hydroxocobalamin is not indicated.

Prednisolone can be used for acute flares of Crohn's disease which can present with diarrhoea. However, the faecal calprotectin, CRP and platelets (which often rise in inflammation) are normal making an acute disease flare unlikely; therefore, prednisolone is not indicated.

Question 77 of 86

A 68-year-old female presents with a 4 month history of diarrhoea. She denies mucus, melaena or hematochezia. On examination her abdomen is soft and non-tender.

Investigation results are as follows:

Hb	135 g/l	Na 138 mmol/l Bilirubin 24 µmol/l+
Platelets	360 * 10 /l 9	K 4.2 mmol/l ALP 105 u/l+
WBC	9.6 * 10 /l 9	Urea 5.4 mmol/l ALT 32 u/l
Neuts	6.2 * 10 /l 9	Creatinine 66 µmol/l γGT 84 u/l
Lymphs	3.2 * 10 /l 9	Albumin 38 g/l
Eosino	0.1 * 10 /l9	
CRP	<4 mg/l	
SeHCAT test	7 day retention value = 7%	Normal > 15%
Faecal calprotectin	45 ug/g (normal range 0-110)	

What would be the next step in the management?

A. Loperamide
B. Prednisolone
C. Cholestyramine
D. Colonoscopy
E. CT colonography

C. Cholestyramine

SeHCAT is bile acid analogue which can be detected by a nuclear medicine scan. The SeHCAT test involves a baseline scan, and then a 7 day scan. A 7-day SeHCAT retention value of less than 15% is generally considered indicative of bile salt malabsorption

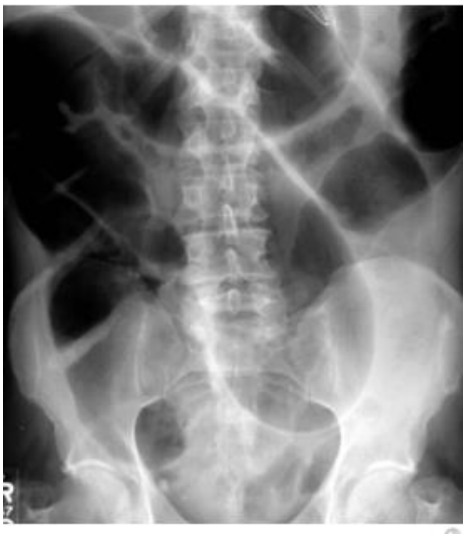
SeHCAT is bile acid analogue that can be detected by a nuclear medicine scan. The SeHCAT test involves a baseline scan, and then a 7-day scan. A 7-day SeHCAT retention value of less than 15% is generally considered indicative of bile salt malabsorption.

Treatment of bile salt malabsorption is with cholestyramine, a bile salt sequestrant.

Question 78 of 86

An 81-year-old man develops abdominal pain in the acute medical unit having not opened his bowels for four days. It is unclear if he has been passing flatus however he has not been vomiting. He was admitted following a fall and is currently receiving analgesia with paracetamol and codeine as needed and is awaiting nursing home placement. His past medical history includes Alzheimer's dementia, gout, and appendicectomy as a child.

On assessment, his respiratory rate is 19/min and oxygen saturations are 93% on air. His heart rate is 84/min and his blood pressure is 131/90mmHg. Examination of the abdomen reveals a distended, generally tender abdomen and a rectal examination reveals an empty rectum. His temperature is 37.1ºC and his Glasgow coma score is 14 due to confusion (which is his baseline).



What is the most likely diagnosis?

- A. Caecal volvulus
- B. Constipation
- C. Sigmoid volvulus
- D. Small bowel obstruction
- E. Toxic megacolon

C. Sigmoid volvulus

This man has a sigmoid volvulus as confirmed on his abdominal x-ray which shows the classic coffee bean sign. Note that a sigmoid volvulus will not retain haustra and typically originate in the left lower quadrant and extend to the right upper quadrant as is the case here.

A caecal volvulus will typically originate in the right lower quadrant and extend to the left upper quadrant. Additionally, caecal volvuli retain haustra. As both of these features are not present here, a sigmoid volvulus is a more likely diagnosis.

In constipation, large quantities of faeces can be seen throughout the bowel however the bowel loops, in this case, are air-filled. This, therefore, rules out a diagnosis of constipation which is incorrect.

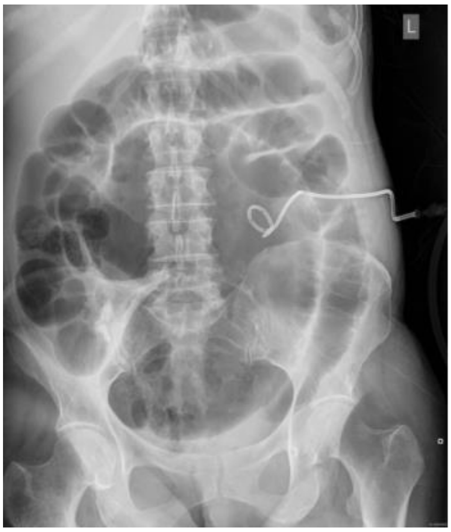
This x-ray displays a coffee bean appearance typical of volvuli rather than small bowel obstruction. In small bowel obstruction, valvulae conniventes are seen which are absent in the distended bowel loops here confirming the pathology is large bowel in origin.

Toxic megacolon is a complication of inflammatory bowel disease (IBD) and less commonly, infectious colitis. Both of these diagnoses are unlikely given the lack of any relevant history here. Additionally, the radiographic features are specific for a volvulus (coffee bean sign) rather than toxic megacolon which typically shows dilated large bowel possibly with thumb-printing.

Question 79 of 86

A 59-year-old woman is admitted to hospital after developing an infection of a non-functioning left kidney, Intravenous antibiotics are given and a nephrostomy tube is inserted. During her recovery, she starts to develop a distended abdomen and complains of nausea and abdominal pain.

An abdominal film is requested:



What is the cause of the abdominal distension?

- A. Bowel perforation
- B. Intussusception
- C. Bacterial peritonitis
- D. Caecal volvulus
- E. Subcutaneous emphysema

D. Caecal volvulus

Small bowel obstruction is visible on this film (note the valvulae conniventes, mucosal folds, that cross the full width of the bowel) secondary to caecal volvulus. Note the left nephrostomy tube insitu.

Question 80 of 86

A 17-year-old male presents with a 3-month history of PR bleeding, lower abdominal pain and left jaw pain. He reports an inability to gain weight and appears 'skinnier than his friends'. He has no other medical history. He knows his father and uncle have 'some problems with their bowels but they don't talk about it'. On examination, he has a body mass index of 14.7 kg/m² and abdomen is soft. PR is negative. Cardiovascular and respiratory examinations are normal. Limb neurological examination is unremarkable. However, when testing muscles of mastication, you note a raised bony mass in his left jaw that is tender in a non-cranial nerve distribution to palpation. Colonoscopy reveals hundreds of polyps in his small bowel.

What is the most likely diagnosis?

- A. Colorectal carcinoma
- B. Gardners syndrome
- C. Peutz-Jeghers syndrome
- D. Hereditary non-polyposis colorectal cancer
- E. Ulcerative colitis

B. Gardners syndrome

This is a difficult question requiring knowledge of a combination of colorectal polyps with extracolonic manifestations. The presence of classical osteomas with a familial polyposis syndrome, sometimes also with cutaneous, adrenal or nasal lesions, is diagnostic of

Gardners syndrome, a variant of familial adenomatous polyposis. Peutz-Jeghers is a reasonable differential but its polyps are more frequently associated with pigmented lesions instead of bony outgrowths. HNPCC increases the risk of colonic cancer without polyps, a diagnosis of carcinoma, despite at high risk, cannot be made at this stage without positive malignant histology.

Question 81 of 86

A 29 year-old adopted woman is referred by her GP with a two week history of blood in her stools. Furthermore, over the last three months her stools have become more loose and she has 2-3 bowels motions a day. Her past medical history includes an osteoma of her left tibia as well as one of her skull. The only relevant family history she is aware of is a brother who has ulcerative colitis. She currently smokes five cigarettes per day and drinks 10 units of alcohol per week. Her occupation is a lawyer.

What is her most likely diagnosis?

A. Crohn's disease
B. Ulcerative colitis
C. Gardener's syndrome
D. Peutz-Jeghers syndrome
E. Familial adenomatous polyposis

C. Gardener's syndrome

Gardener's syndrome is a condition that is inherited in an autosomal dominant fashion. It's main clinical features include adenomatous intestinal polyposis, which can present with changes in bowel habit and rectal bleeding, osteomas and fibromas. 50% of patients with Gardener's syndrome have colon cancer by the age of 39.

Question 82 of 86

A 38 year-old nulliparous woman, who is at 35 weeks gestation, presents with a 10-hour history of fatigues, nausea and vomiting. She has not eaten for the past 24 hours and says that she feels a little dizzy. She describes no blood in her vomit and has had no recent changes in her bowel habit. She has no past medical history of note and she does not currently take any regular medication. She has no family history of note and does not smoke or drink any alcohol.

On examination she appears to be very anxious and abdominal exam reveals right upper quadrant and epigastric pain on deep palpation. There is no organomegaly and bowel sounds are present. The baby is moving normal.

She has a temperature of 37.5oC, heart rate of 85 beats per minute, blood pressure of 135/80mmHg, oxygen saturation of 99% and a respiratory rate of 18 breaths per minute.

Blood tests reveal:

Hb	10.5 g/dL
Platelets	82 * 10 /l9
WBC	12.9 * 10 /l9
Na +	134 mmol/l
K +	3.9 mmol/l
Urea	7.2 mmol/l
Creatinine	95 µmol/l
CRP	20 mg/l
ESR	8 mm/hr
Bilirubin	90 µmol/l
ALP	140 u/l
ALT	431 u/l
γGT	45 u/l
Albumin	30 g/l
Prothrombin time (PT)	23 seconds

What is the most likely diagnosis?

A. Ascending cholangitis
B. Intrahepatic cholestasis of pregnancy
C. Fatty liver of pregnancy
D. Infectious gastroenteritis
E. Hyperemesis gravidarum

C. Fatty liver of pregnancy

The most likely diagnosis in this case is fatty liver of pregnancy. This usually presents with nausea and vomiting in combination with pain in the epigastrium and right upper quadrant of the abdomen. The normal gestational range it tends to affect is 28-42 weeks, as opposed to hyperemesis gravidarum, which affects early pregnancy.

Disseminated intravascular coagulation (DIC) is a risk of developing this condition and this patient is displaying some of the signs (low platelets and raised prothrombin time) of starting to develop DIC. Intrahepatic cholestasis of pregnancy tends to present with fatigue and itching as the two main symptoms and ascending cholangitis tends to be a triad of fever, right upper quadrant pain and jaundice. Gastroenteritis does not tend to raise liver function tests to the values displayed above.

Question 83 of 86

A 30-week pregnant woman presents with itchy palms and soles of the feet particularly at night but no rash. She is not jaundiced and has not travelled recently. Liver enzymes and serum bile acids are raised (see table). Prothrombin time is normal. Urine dipstick reveals 1+ of protein and her blood pressure is 132/80.

ALT	65 mmol/l
ALP	171 mmol/l
Bilirubin	40 mmol/l
Bile acids	80 mmol/l
Pro-thrombin time	Normal
Anti-nuclear Antibodies (ANA)	Negative
Anti-Mitochondrial antibodies	Negative
Urine Protein-Creatinine Ratio	14 mg/mmol/l

What is the most likely diagnosis?

A. Acute fatty liver of pregnancy
B. Intrahepatic cholestasis of pregnancy
C. Pre-eclampsia
D. Primary biliary cirrhosis
E. Gallstones

B. Intrahepatic cholestasis of pregnancy

This is a typical presentation for intrahepatic cholestasis of pregnancy. Pruritis affects the limbs and trunk particularly the palms and soles, developing in the second half of pregnancy. Rash and jaundice are rare features of this condition. There is a moderate elevation (less than three-fold) in transaminases and primary bile acids rise (10-100 fold). However these are not specific and other causes of deranged liver function tests in pregnancy should be excluded. There is a risk of fetal death with raising bile acids. Complete recovery occurs within 48 hours of delivery. Management is with ursodeoxycholic acid (UDCA). It has a high recurrence in future pregnancies.

Acute fatty liver of pregnancy is rare but potentially lethal for both mother and fetus. It often presents near term with severe vomiting and abdominal pain. The patient is usually jaundiced.

Pre-eclampsia is a condition associated with hypertension and proteinuria in pregnancy. There is 1+ protein on urine dipstick so a urinary protein-creatinine ratio has been requested which is normal (>30 mg/mmol is pathological). Pre-eclampsia symptoms such as headache, blurred vision, abdominal pain and oedema are not present.

Primary biliary cirrhosis is an autoimmune liver disease associated with destruction of the intralobular ducts. Anti-Mitochondrial antibodies are highly specific to the condition (negative in this patient).

The patient does not complain of abdominal pain, which would suggest an obstructing gallstone. Note: Pregnancy-specific reference ranges for liver function tests should be used (~20% lower than non-pregnant ranges). Also remember that ALP is produced by the placenta and therefore not useful in pregnant women.

Question 84 of 86

A 55-year-old lady if referred to the gastroenterology clinic. She has a 3-year history of iron deficient anaemia despite iron therapy. When you see her she describes feeling tired all of the time with no energy. She denies any haematemesis, weight loss, melena or fresh red blood PR. She also denies any haemoptysis, cough or urinary symptoms. She is post-menopausal and has not been on any HRT. She is a non-smoker and drinks alcohol socially. She is otherwise well and only on Ferrous sulphate 200mg BD. On examination, she has pale conjunctiva but an examination is otherwise unremarkable. She has been admitted twice over the last 6 months for blood transfusions and has subsequently dropped her Hb after being discharged.

She has been extensively investigated by her GP who has attached her test results with the referral letter.

Hb	81 g/l
MCV	69 fl
Platelets	299 * 10 /l9
WBC	6.2 * 10 /l9
Na +	141 mmol/l
K +	4.1 mmol/l
Urea	7.1 mmol/l
Creatinine	101 µmol/l
CRP	11 mg/l
Bilirubin	10 µmol/l
ALP	56 u/l
ALT	21 u/l
Albumin	38 g/l
Serum electrophoresis	no paraprotein
Immunoglobulins	within normal limits
Ferritin	4 ng/mL (10-150)

USS abdomen: NAD

Transvaginal USS: NAD CT chest, abdomen and pelvis: NAD

OGD: NAD

Colonoscopy: NAD

Faecal occult blood: positive

The Gastroenterologist that sees her suspects an occult GI bleed.

What is the next most appropriate investigation for this lady?

A. Endoscopic ultrasound
B. CT angiography
C. Repeat colonoscopy
D. Capsule endoscopy
E. Repeat CT chest abdo and pelvis

D. Capsule endoscopy

This lady has an occult GI bleed. The SIGN guidelines for occult bleeding recommend OGD and colonoscopy. If they are both normal they recommend either repeat OGD or capsule endoscopy. If the capsule is negative then either a second capsule or enteroscopy is indicated. CT angiography would be used for acute occult bleeding where the patient is compromised and embolisation of the bleeding vessel is needed.

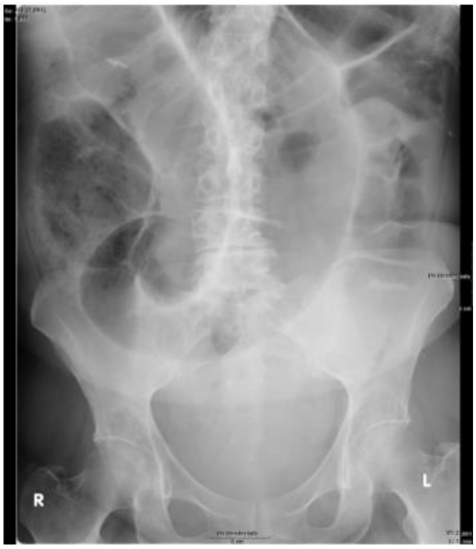
Question 85 of 86

A 75-year-old woman presents to the Emergency Department with abdominal pain, bloating and constipation. The pain is described as being in the centre of his abdomen. She last opened her bowels around three days ago.

Her past medical history includes constipation and Parkinson's disease.

On examination she is diffusely tender in the left lower quadrant of the abdomen but there is no guarding or rigidity. Her blood pressure is 130/82 mmHg and heart rate is 90/min.

The abdominal film is shown below:



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What is the cause of this patient's symptoms?

A. Ulcerative colitis with toxic megacolon
B. Diverticulitis
C. Faecal loading
D. Sigmoid volvulus
E. Colorectal cancer

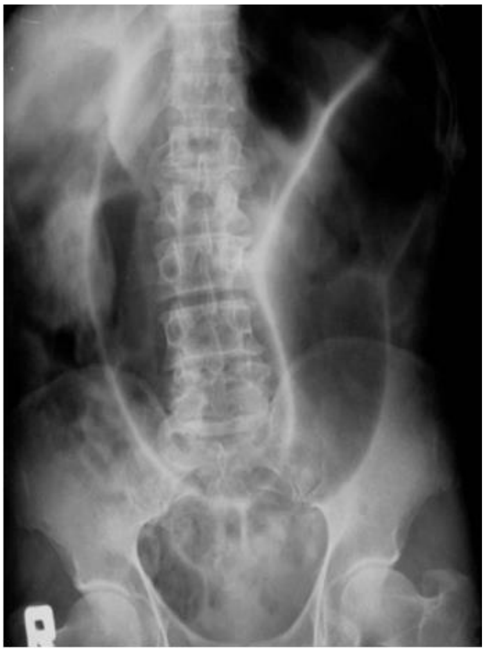
D. Sigmoid volvulus

The abdominal film is consistent with sigmoid volvulus. Note the signs of large bowel obstruction alongside the coffee bean sign.

Question 86 of 86

A 62-year-old biology professor is seen after returning from a trip to South America. For the past week he has been investigated for fever, lymphadenopathy, periorbital oedema and headaches. A diagnosis of Chagas' disease is suspected.

During his admission he develops abdominal pain, constipation and distension. An abdominal film is requested:

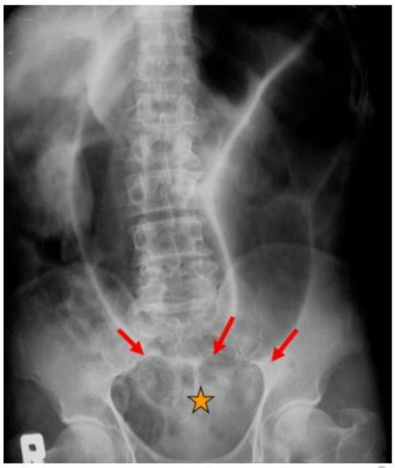


What complication has developed?

A. Sigmoid volvulus
B. Colon cancer
C. Intussusception
D. Hydronephrosis
E. Ischaemic colitis

A. Sigmoid volvulus

You can see the typical 'coffee bean' appearance with three dense lines converging towards the site of obstruction (Frimann Dahl's sign) in keeping with sigmoid volvulus.



Alrajhi Jad

PASSMEDICINE
NEPHROLOGY

Internal medicine

EDITED BY
DR. JAMIL ALRAJHI

2024

773-555-493
jameelalrajhi@gmail.com

PART 2

ENDOCRINE

2024

NEPHROLOY

PART 2

2024

Question 1 of 183

A 53-year-old male presents acutely as a primary percutaneous coronary intervention (PPCI) call after sudden onset chest pain of onset 3 hours ago, associated with nausea, vomiting and profuse sweating.

His ECG in the ambulance demonstrates ST elevation noted on V2-V5. His past medical history includes include hypertension, type 2 diabetes mellitus, chronic kidney disease (creatinine baseline = 170 µmol/l at the renal clinic 2 months ago). He is taken straight to the cardiac catheter laboratory, where he undergoes angioplasty and a single drug-eluting stent to his left anterior descending artery. On examination, he is pain-free, warm peripherally without respiratory distress. Capillary refill time is less than 2s and jugular venous pulse at 3 cm above the angle of Louis. His heart sounds and chest are unremarkable. A post-procedure transthoracic echocardiogram demonstrates moderate left ventricular impairment with no valve abnormalities.

At 24 hours after admission, his blood tests are repeated and are as follows:

Hb	92 g/l
WCC	13.5 * 10 /l9
Plt	198* 10 /l9
Na	138 mmol/l
K	4.2 mmol/l
Urea	12.9 mmol/l
Creat	254 µmol/l
Trop	0.87 (normal < 0.03)

What is the most appropriate next management step in addition to optimisation of hydration status?

A. Monitor with daily U+Es
B. Haemofiltration
C. Organise urgent outpatient haemodialysis
D. Intravenous n-acetylcysteine
E. Intravenous mannitol

A. **Monitor with daily U+Es 75%**

Acute kidney injury following a percutaneous coronary intervention→ ?contrast-induced nephropathy

The patient has no indications for acute renal replacement therapy. There is little to suggest the patient has a significant chronic deterioration of renal function since his last renal appointment. The main consideration here is that he has just received a significant amount of contrast during his PPCI and hence explains the increase in creatinine. In studies of post-contrast nephropathy, creatinine increases of around 25% were common . While pre and post contrast hydration reduces the risk of contrast nephropathy, there is no role for mannitol, which produces a greater incidence of adverse events .

The evidence for intravenous n-acetylcysteine as a preventative measure is equivocal, with conflicting results regarding the efficacy of prevention. However, given its cheap cost and ready availability, it is recommended in the 2012 KDIGO guidelines for prevention of contrast nephropathy in high-risk patients. It is not, however, routinely used as a treatment after contrast nephropathy. The next most appropriate step in this patient is monitoring of renal dysfunction, which should spontaneously reverse in most patients.

Nephrotoxicity due to contrast media

Contrast media nephrotoxicity may be defined as a 25% increase in creatinine occurring within 3 days of the intravascular administration of contrast media. Contrast-induced nephropathy occurs 2 -5 days after administration.

Risk factors include

- known renal impairment (especially diabetic nephropathy)
- age > 70 years
- dehydration
- cardiac failure
- the use of nephrotoxic drugs such as NSAIDs

Examples of procedures that may cause contrast-induced nephropathy includes:

- CT with contrast
- coronary angiography/percutaneous coronary intervention (PCI)
    - around 5% of patients who've undergone PCI develop a transient rise in the plasma creatinine concentration of more than 88 µmol/L

Prevention

- the evidence base currently supports the use of intravenous 0.9% sodium chloride at a rate of 1 mL/kg/hour for 12 hours pre- and post- procedure. There is also evidence to support the use of isotonic sodium bicarbonate
- N-acetylcysteine has been given in the past but recent evidence suggests it is not effective*

Patients who are high-risk for contrast-induced nephropathy should have metformin withheld for a minimum of 48 hours and until the renal function has been shown to be normal. This is due to the risk of lactic acidosis.

Question 2 of 183

A 43-year-old man with alcoholic liver disease and portal hypertension was admitted with confusion. Abdomen was distended but non-tender. He was taking propranolol, spironolactone, vitamin B12 , and thiamine; these medications were continued over the next 3 days. He was initially treated with intravenous fluids and lactulose. Renal scan done was normal. A ward registrar reviewed the patient with blood results below on the 4th day of admission and observed he had an acute kidney injury.

Investigations on admission:

Urea	4.0 mmol/l (2.5-7.0)
Creatinine	70 µmol/l (60-110)

Investigations 3 days later

Urea	8.0 mmol/l (2.5-7.0)
Creatinine	150 µmol/l (60-110)
Urine sodium	60meq/l
Urine protein	250mg/24hours
Urine osmolality	800mOsm/kg
Urine microscopy	Granular casts and renal epithelial cells seen

Which of the following is the most likely cause of his acute kidney injury?

A. Pre-renal azotaemia
B. Acute tubular necrosis
C. Hepatorenal syndrome
D. Nephrotic syndrome
E. Obstructive uropathy

B. Acute tubular necrosis

Granular casts can be found in both acute tubular necrosis (ATN) and hepatorenal syndrome (HRS), but renal epithelial cells are more suggestive of ATN

Granular casts can be found in both acute tubular necrosis (ATN) and hepatorenal syndrome (HRS), but renal epithelial cells are more suggestive of ATN. A normal USS is useful for excluding an obstructed urinary system and parenchymal disease, but could be seen in both ATN and HRS. A low urinary sodium concentration and high urinary osmolarity are consistent with pre-renal azotaemia and HRS, as renal tubular integrity is usually maintained, in contrast to ATN, although these findings are not a criterion for the diagnosis of HRS. Importantly, this patient remained on diuretics and therefore the urinary electrolyte measurements are uninterpretable. Diuretics should be stopped for at least 2 days before a diagnosis of HRS can be made.

Acute kidney injury: NICE guidelines and staging

This guideline discuss what increases the risk of AKI:

- Emergency surgery, ie, risk of sepsis or hypovolaemia
- Intraperitoneal surgery
- CKD, ie if eGFR < 60
- Diabetes Heart failure Age >65 years Liver disease
- Use of nephrotoxic drugs NSAIDs aminoglycosides
- ACE inhibitors/angiotensin II receptor antagonists diuretics

Diagnostic criteria

It also defines the criteria for diagnosing AKI

Rise in creatinine of 26µmol/L or more in 48 hours OR >= 50% rise in creatinine over 7 days OR

Fall in urine output to < 0.5ml/kg/hour for more than 6 hours in adults (8 hours in children) OR

>= 25% fall in eGFR in children / young adults in 7 days.

Staging criteria

The Kidney Disease: Improving Global Outcomes (KDIGO) criteria are widely used to stage the AKI:

Stage 1	Increase in creatinine to 1.5-1.9 times baseline, or Increase in creatinine by ≥26.5 µmol/L, orReduction in urine output to <0.5 mL/kg/hour for ≥ 6 hours
Stage 2	Increase in creatinine to 2.0 to 2.9 times baseline, or Reduction in urine output to <0.5 mL/kg/hour for ≥12 hours
Stage 3	Increase in creatinine to ≥ 3.0 times baseline, orIncrease in creatinine to ≥353.6 µmol/L orReduction in urine output to <0.3 mL/kg/hour for ≥24 hours, or The initiation of kidney replacement therapy, or,In patients <18 years, decrease in eGFR to <35 mL/min/1.73 m2

Referral criteria

Refer to a nephrologist if any of the following apply:

- Renal tranplant
- ITU patient with unknown cause of AKI
- Vasculitis/ glomerulonephritis/ tubulointerstitial nephritis/ myeloma
- AKI with no known cause
- Inadequate response to treatment
- Complications of AKI
- Stage 3 AKI (see guideline for details)
- CKD stage 4 or 5
- Qualify for renal replacement hyperkalaemia / metabolic acidosis/ complications of uraemia/ fluid overload (pulmonary oedema)

Question 3 of 183

An 11-year-old girl presents with a gradual onset of short-sightedness. She has no prior medical history and is developmentally normal.

Examination reveals reduced visual acuity and lens protrusion. Her blood pressure is 150/92 mmHg.

A urinalysis shows moderate levels of blood and protein.

Na +	138 mmol/L	(135 - 145)
K +	5.6 mmol/L	(3.5 - 5.0)
Bicarbonate	20 mmol/L	(22 - 29)
Urea	8.7 mmol/L	(2.0 - 7.0)
Creatinine	179 µmol/L	(55 - 120)

What is the most likely diagnosis?

A. Alport syndrome
B. Fabry disease
C. Hyperoxaluria
D. Nail-patella syndrome
E. Thin basement membrane disease

A. Alport syndrome

Think of Alport syndrome when renal failure, sensorineural hearing loss and ocular abnormalities develop in a child

This patient presents with ocular disturbances, and renal impairment consistent with **Alport syndrome**. If tested, we would expect audiometry to reveal bilateral sensorineural hearing impairment. Although Alport syndrome typically causes severe defects in males due to its X-linked inheritance pattern, approximately 15% of cases are inherited in an autosomal recessive fashion and affect females equally. There is no specific treatment for Alport syndrome. However, as with any progressive glomerular disease, management of blood pressure and proteinuria should be optimised with ACE inhibitors, angiotensin receptor blockers, and sodium-glucose cotransporter 2 inhibitors.

Fabry disease is an X-linked multisystem disorder resulting from a deficiency of the enzyme α- galactosidase. It typically presents in adolescence with skin, cardiac, and neurological involvement. Although renal impairment does occur, it usually develops later (typically between ages 30 and 50). The ocular manifestations of Fabry disease include corneal opacities and dystrophy, and sensorineural hearing loss can occur but at later stages. The lens protrusion and early onset of kidney impairment are more consistent with Alport syndrome.

Hyperoxaluria is an autosomal recessive condition caused by defective enzyme pathways that lead to an accumulation of oxalate. Clinically, it presents in childhood with calcium oxalate calculi and nephrocalcinosis. There is also significant oxalate deposition in the heart, vasculature, and joints, leading to morbidity. It doesn't typically cause eye problems, as seen in this case. The treatment of choice is a combined liver and kidney transplant.

Nail-patella syndrome is a rare autosomal dominant condition, with renal involvement occurring in 50% of patients. It is caused by a gene mutation in the LMX1B gene on chromosome 9, which codes for a transcription factor essential for maintaining the glomerular basement membrane. Nails may be absent, dysplastic, discoloured, misshapen, or have ridges. Patellae may be absent, leading to early-onset osteoarthritis. Renal features typically present in adolescence, including microscopic haematuria, proteinuria, hypertension, and chronic kidney disease. However, it does not cause the ocular features presenting in this case.

Thin basement membrane disease is a common autosomal dominant condition caused by mutations in the COL4A3 and COL4A4 genes. This results in partial failure of the glomerular basement membrane, and some clinicians consider it a milder form of Alport syndrome. It presents with microscopic haematuria, but renal function is generally normal.

Alport's syndrome

Alport's syndrome is usually inherited in an X-linked dominant pattern*. It is due to a defect in the gene which codes for type IV collagen resulting in an abnormal glomerular-basement membrane (GBM). The disease is more severe in males with females rarely developing renal failure.

A favourite question is an Alport's patient with a failing renal transplant. This may be caused by the presence of anti-GBM antibodies leading to a Goodpasture's syndrome like picture.

Alport's syndrome usually presents in childhood. The following features may be seen:

- microscopic haematuria
- progressive renal failure
- bilateral sensorineural deafness
- lenticonus: protrusion of the lens surface into the anterior chamber
- retinitis pigmentosa
- renal biopsy: splitting of lamina densa seen on electron microscopy

Diagnosis

- molecular genetic testing
- renal biopsy electron microscopy: characteristic finding is of the longitudinal splitting of the lamina
- densa of the glomerular basement membrane, resulting in a 'basket-weave' appearance

*in around 85% of cases - 10-15% of cases are inherited in an autosomal recessive fashion with rare autosomal dominant variants existing

Question 4 of 183

A 57-year-old woman with a background of systemic lupus erythematosus (SLE) has been transferred to the renal ward with worsening renal function, as shown below.

Na +	137 mmol/L	(135 - 145)
K +	5.0 mmol/L	(3.5 - 5.0)
Bicarbonate	16 mmol/L	(22 - 29)
Urea	19.2 mmol/L	(2.0 - 7.0)
Creatinine	429 µmol/L	(55 - 120)

The renal team has performed a renal biopsy.

Renal biopsy	'Wire-loop' appearance with endothelial and mesangial proliferation in keeping with diffuse proliferative glomerulonephritis.

She has been commenced on high-dose methylprednisolone with a good response in her renal function.

Which of the following is the most appropriate maintenance treatment?

A. Azathioprine
B. Cyclophosphamide
C. Mycophenolate
D. Steroids
E. Tacrolimus

C. Mycophenolate

Mycophenolate is the preferred follow-on agent in lupus nephritis once there has been a response to the initial therapy

This case describes a woman with known SLE developing significant kidney dysfunction, most likely secondary to lupus nephritis. Her renal biopsy has shown diffuse proliferative glomerulonephritis which is class IV lupus nephritis on the WHO classification.

Lupus nephritis is initially managed with high-dose steroids alongside either mycophenolate or cyclophosphamide. The preferred treatment for maintenance is mycophenolate.

Azathioprine is a recognised maintenance treatment, however has a higher risk of development of end-stage renal disease and is, therefore, less preferred.

Cyclophosphamide can be used alongside steroids to induce remission, however is not used for maintenance.

Steroids, as with cyclophosphamide, are only used in the initial treatment and are not used in long-term management.

Tacrolimus is also a recognised treatment however mycophenolate is preferred.

Question 5 of 183

A 19-year-old university student presents to the Emergency Department with a decreased consciousness level. His flatmate says that he has been struggling with living away from home recently and has been isolating himself from his friends. Numerous friends have recently had a self-limiting episode of vomiting and diarrhoea. On admission, the patient is tachycardia, tachypnoeic, hypotensive and hyperglycaemic.

His initial venous blood gas:

pH	7.08	(7.35-7.45)
pO2	5.6 kPa	(10-14)
pCO2	6.2 kPa	(4.0 - 6.0)
HCO3	8 mmol/L	(22-29)
Lactate	5 mmol/L	(0-2)
Na	122 mmol/L	(135-145)
K	3.0 mmol/L	(3.5-5)
Cl	98 mmol/L	(96-106)

After aggressive fluid management and potassium replacement, an insulin infusion was started. Four hours later the blood gas shows:

pH	7.31	(7.35-7.45)
pO2	5.3 kPa	(10-14)
pCO2	5.2 kPa	(4.0 - 6.0)
HCO3	18 mmol/L	(22-29)
Lactate	1.3 mmol/L	(0-2)
Na	132 mmol/L	(135-145)
K	3.5 mmol/L	(3.5-5)
Cl	118 mmol/L	(95-109)

What is the cause of the hyperchloraemic metabolic acidosis in this patient?

A. 0.9% saline infusion
B. Diarrhoea
C. Ethylene glycol ingestion
D. Renal failure
E. Vomiting

A. 0.9% saline infusion

Aggressive treatment with 0.9% saline in DKA commonly causes a hyperchloraemic metabolic acidosis

0.9% saline infusion is correct. The patient has presented in DKA which requires rapid fluid resuscitation before and after starting an insulin infusion, as per local trust guidelines. 0.9% saline contains 154mEq of chloride ions, this leads to an increase in the extracellular concentration of chloride ions. The increase in extracellular chloride ions causes an intracellular influx of bicarbonate ions resulting in metabolic acidosis. This can also lead to a rise in the extracellular potassium levels.

Diarrhoea is incorrect because this is unlikely in this scenario. Although diarrhoea can cause hyperchloraemic metabolic acidosis due to loss of bicarbonate this is not the likely cause in this case. If diarrhoea from the primary infection was the cause then this would be present in their initial investigations. Hyperchloraemic metabolic acidosis has developed since the initiation of treatment, making this less likely to be the cause.

Ethylene glycol ingestion is incorrect because this is unlikely in this scenario. Ethylene glycol as a parent molecule is not considered toxic. However, its metabolites created by the metabolism of the compound by alcohol dehydrogenase are extremely toxic. By blocking alcohol dehydrogenase, either with ethanol or fomepizole, the non-toxic ethylene glycol can be excreted by the kidneys. Initially, the patient will have a high osmolar gap but as their condition deteriorates then they will develop a metabolic acidosis with raised anion gap. If there is any suspicion of ethylene glycol ingestion then an urgent consultation with a toxicologist is required.

A pneumonic to remember causes of a raised anion gap metabolic acidosis, MUDPILES:

- Methanol
- Uraemia
- Diabetic ketoacidosis
- Phenformin, paraldehyde
- Iron
- Lactate
- Ethanol
- Salicylates

Renal failure is incorrect because this is less likely the cause of hyperchloraemic metabolic acidosis in this case. Acute renal failure can cause hyperchloraemia metabolic acidosis but in this case, it has developed after resuscitation with 0.9% saline and the rest of the blood results are improving. In acute renal failure you would expect hyperkalaemia rather than hypokalaemia, but this is not always the case.

Vomiting is incorrect because vomiting causes an increase in chloride ion loss. In severe cases, such as pyloric stenosis in neonates and hyperemesis cannabinoid syndrome the patient may develop hypochloraemic hypokalaemic metabolic alkalosis.

Metabolic acidosis

Metabolic acidosis is commonly classified according to the anion gap. This can be calculated by: (Na + K) - (Cl + HCO). If a question supplies the chloride level then this is often a clue that the anion gap should be calculated. The normal range = 10-18 mmol/L

Normal anion gap (= hyperchloraemic metabolic acidosis)

- gastrointestinal bicarbonate loss:
  - prolonged diarrhoea: may also result in hypokalaemia
  - ureterosigmoidostomy
  - fistula
- renal tubular acidosis drugs: e.g. acetazolamide
- ammonium chloride injection
- Addison's disease

Raised anion gap

- lactate:
    - shock
    - sepsis
    - hypoxia
- ketones:
    - diabetic ketoacidosis
    - alcohol
- urate: renal failure
- acid poisoning: salicylates, methanol

Metabolic acidosis secondary to high lactate levels may be subdivided into two types:

- lactic acidosis type A: sepsis, shock, hypoxia, burns
- lactic acidosis type B: metformin

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A 72-year-old man is admitted with sudden onset shortness of breath and haemoptysis, with oxygen saturations of 92% on room air. Two days prior to admission, his general practitioner had prescribed furosemide 40mg once a day for progressive lower limb swelling and fatigue. He was due to see his general practitioner again tomorrow to review the initial blood tests. His past medical history includes systemic hypertension and stage 2 colon cancer treated with surgical resection and adjuvant chemotherapy (15 years ago). He is a current smoker.

An arterial blood gas shows a type 1 respiratory failure, with a pH of 7.31 and a pO2 of 9.7 kPa. Oxygen saturations of >96% are achieved with four litres of oxygen. A chest x-ray shows alveolar infiltrates within the right lower lobe. Urgent spirometry reveals an increased transfer factor.

Urine dip demonstrates a loss of protein, but no blood, leucocytes or nitrites. A full panel of bloods, including a full vasculitic screen is requested (see below).

White cells	12.0 Ã— 10^9/l
Haemoglobin	11.7 g/dl
Sodium	144 mmol/l
Potassium	5.6 mmol/l
Urea	18 mmol/l
Creatinine	240 mol/l
Albumin	19 g/l
ANA	negative
ANCA	negative
Anti-GBM	positive

What is the next management step?

A. Thrombolysis
B. Intravenous antibiotics
C. Haemodialysis
D. Continuous positive airway pressure (CPAP)
E. Plasmapheresis

A. Plasmapheresis

Anti-GBM disease is typically managed with a combination of corticosteroids, cyclophosphamide and plasmapheresis

This gentleman has Goodpasture's disease, an autoimmune disease characterised by antibodies directed towards the basement membranes of the glomeruli and alveoli. He has subsequently developed a pulmonary haemorrhage as evidenced by the hypoxia, chest x-ray changes and increased transfer factor (KCO). This gentleman should be started on intravenous corticosteroids, cyclophosphamide and plasmapheresis.

There is no role for thrombolysis given that he has had a pulmonary haemorrhage.

There is no current evidence of infection to warrant intravenous antibiotics. However, this is commonly a precipitant for a pulmonary haemorrhage.

This gentleman may require haemodialysis however at present he does not require acute haemodialysis given his pH and potassium.

CPAP may be used in patients with pulmonary haemorrhage who have a type 1 respiratory failure. However, his oxygen demand has been managed successfully with supplementary oxygen and what he requires is treatment of the underlying condition.

Anti-glomerular basement membrane (GBM) disease (Goodpasture's syndrome)

Anti-glomerular basement membrane (GBM) disease (previously known as Goodpasture's syndrome) is a rare type of small-vessel vasculitis associated with both pulmonary haemorrhage and rapidly progressive glomerulonephritis. It is caused by anti-glomerular basement membrane (anti-GBM) antibodies against type IV collagen. Goodpasture's syndrome is more common in men (sex ratio 2:1) and has a bimodal age distribution (peaks in 20-30 and 60-70 age bracket). It is associated with HLA DR2.

Features

- pulmonary haemorrhage
- rapidly progressive glomerulonephritis
    - this typically results in a rapid onset acute kidney injury
    - nephritis → proteinuria + haematuria

Investigations

- renal biopsy: linear IgG deposits along the basement membrane
- raised transfer factor secondary to pulmonary haemorrhages

Management

- plasma exchange (plasmapheresis)
- steroids
- cyclophosphamide

One of the main complications is pulmonary haemorrhage. Factors that increase the likelihood of this include:

- smoking
- lower respiratory tract infection
- pulmonary oedema
- inhalation of hydrocarbons
- young males

Question 7 of 183

A 30-year-old man with bipolar disorder is admitted with malaise and lethargy. He takes lithium regularly and complains of increased thirst and weakness.

His urine output is 4.5L in 24 hours.

Na +	154 mmol/l
K +	4.0 mmol/l
Urea	6.1 mmol/l
Creatinine	72 µmol/l
Calcium	2.47 mmol/l
Glucose	7.2 mmol/l
Urine Osmolarity	254 osmol/l (NR 500-800)

What is the most appropriate next step in management?

A. 5% Dextrose
B. 0.45% Saline
C. Desmopressin
D. Fluid restriction
E. Thiazide diuretic

E. Thiazide diuretic

Diabetes insipidus is characterised by a **high plasma** osmolality and a **low urine** osmolality

This patient has hypernatraemia secondary to nephrogenic diabetes insipidus caused by lithium therapy. This is indicated by polyuria, hypernatraemia, lithium use. The urine is inappropriately dilute for his serum sodium.

Treatment is with thiazide diuretics or NSAIDs. Diuretics act to induce hypovolaemia leading to a subsequent increase in proximal sodium and water reabsorption, thereby diminishing the water delivery to the ADH sensitive sites in the collecting tubules, and reducing the urine output.

Hypernatraemia is usually caused by:

- excess of hypertonic fluids (IV saline, enteral or parenteral nutrition);
- excessive free water loss - renal (diabetes insipidus, diuretics, osmotic diuresis as with hyperglycaemia), GI (diarrhoea, vomiting), skin (sweating, burns)
- reduced thirst - seen in very old and very young patients.

Treatment is aimed at the underlying cause. An acute hypernatraemia can be corrected quickly but if chronic (>24hours) then it should be corrected at <0.5mmol/L/hr. Fluid resuscitation should involve oral water, 0.45% saline or 5% dextrose IV.

Diabetes insipidus

Diabetes insipidus (DI) is a condition characterised by either a decreased secretion of antidiuretic hormone (ADH) from the pituitary (cranial DI) or an insensitivity to antidiuretic hormone (nephrogenic DI).

Causes of cranial DI

- Idiopathic
- post head injury
- pituitary surgery
- craniopharyngiomas
- infiltrative
    - histiocytosis X
    - sarcoidosis
- DIDMOAD is the association of cranial Diabetes Insipidus, Diabetes Mellitus, Optic Atrophy and Deafness (also known as Wolfram's syndrome)
- haemochromatosis

Causes of nephrogenic DI

- **genetic:**
    - more common form affects the vasopression (ADH) receptor
    - less common form results from a mutation in the gene that encodes the aquaporin 2
    - channel
- **electrolytes**
    - **hypercalcaemia**
    - **hypokalaemia**
- lithium
    - lithium desensitizes the kidney's ability to respond to ADH in the collecting ducts
- demeclocycline
- tubulo-interstitial disease: obstruction, sickle-cell, pyelonephritis

Features

- polyuria
- polydipsia

Investigation

- high plasma osmolality, low urine osmolality
- a urine osmolality of >700 mOsm/kg excludes diabetes insipidus
- water deprivation test

Management

- nephrogenic diabetes insipidus thiazides low salt/protein diet
- central diabetes insipidus can be treated with desmopressin

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A 37-year-old man presents with bilateral flank pain and frank haematuria, which have occurred several times over the past three years and always resolved spontaneously. In addition, he has experienced increasing fatigue and nausea over the past few months. His mother has hypertension, but there is no other relevant family history, particularly no history of renal disease. He does not take any regular medications. Examination reveals a blood pressure of 189/97 mmHg, 4 cm hepatomegaly, and bilateral ballotable kidneys.

Na +	138 mmol/L	(135 - 145)
K +	4.9 mmol/L	(3.5 - 5.0)
Bicarbonate	23 mmol/L	(22 - 29)
Urea	10.2 mmol/L	(2.0 - 7.0)
Creatinine	259 µmol/L	(55 - 120)

Given the likely diagnosis, what chromosome will carry a pathological mutation?

A. Chromosome 16
B. Chromosome 17
C. Chromosome 4
D. Chromosome 9
E. X chromosome

A. Chromosome 16

Renal impairment, flank masses, hypertension → ?ADPKD

This patient presents with a history consistent with autosomal dominant polycystic kidney disease (ADPKD). The combination of renal impairment, flank masses (bilateral ballotable kidneys), haematuria, and hypertension should prompt consideration of this diagnosis. Although there is no reported family history, up to 25% of patients with ADPKD have no family history. Given the patient's advanced kidney disease at a young age, a mutation in the PKD-1 gene, located on **chromosome 16**, is most likely. PKD1 mutations account for about 85% of ADPKD cases and are associated with earlier onset and more rapid progression of kidney disease compared to PKD2 mutations.

Mutations in the CTNS gene on **chromosome 17** are responsible for cystinosis, inherited in an autosomal recessive pattern. This condition impairs the export of cystine from lysosomes, causing cystine accumulation in target organs. Patients typically present with growth failure, Fanconi's syndrome, and progressive renal impairment during childhood.

Mutations in the PKD-2 gene on **chromosome 4** also cause ADPKD. PKD-2 mutations are less common than PKD-1 mutations and result in a milder form of the disease, with end-stage renal failure occurring approximately 15 years later than in PKD-1 mutations. Given the patient's advanced kidney disease at a young age, a PKD-1 mutation is more likely.

Tuberous sclerosis is a multisystem disorder characterised by hamartomatous lesions. Mutations in the TSC1 gene on **chromosome 9** and the TSC2 gene on chromosome 16 are pathogenic. The condition typically presents with neurological manifestations, such as developmental delay and seizures, as well as skin disease. Renal manifestations include angiomyolipomas, cystic disease, and renal cell carcinoma.

Several renal disorders are caused by mutations on the **X chromosome**, leading to diseases predominantly affecting men. Examples include Alport syndrome, characterised by a triad of progressive nephropathy, sensorineural hearing loss, and ocular abnormalities, and Fabry's disease, which involves skin, cardiac, neurological, ocular, pulmonary, and renal symptoms.

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A 48-year-old gentleman with chronic kidney disease secondary to adult polycystic kidney disease presents with a three-month history of increasing lethargy and fatigue. He reports feeling 'tired all the time' and some mild shortness of breath on climbing stairs. He denies any feelings of light headedness and has walked from the car park to the clinic without any difficulty. On examination he is pale. He has bilateral ballotable kidneys and fullness in the flanks. In his left arm, he has a maturing arterio-venous fistula that has not yet been used.

Bloods show:

Hb	80 g/l
Platelets	125 * 10 /l9
WBC	4.4 * 10 /l9
Na +	135 mmol/l
K +	4.8 mmol/l
Urea	12.2 mmol/l
Creatinine	265 µmol/l
eGFR	15ml/min
Ferritin	102ng/ml
Transferrin saturation	23%

What is the best strategy for managing this patients' anaemia?

A. Transfusion of two units of packed red blood cells immediately.
B. Measurement of serum erythropoietin levels before any treatment.
C. Commencement of erythropoiesis stimulating agents (ESA) aiming for a haemoglobin of100-120 g/dL
D. Commencement of erythropoiesis stimulating agents with a target haemoglobin of 120-140 g/dL
E. Trial of oral steroids

C. Commencement of erythropoiesis stimulating agents (ESA) aiming for a haemoglobin of100-120 g/dL

Anaemia in CKD - aim for 10-12 g/dl

Anaemia is commonly seen in chronic kidney disease (CKD). It is well known that a reduction in haemoglobin is seen with reducing glomerular filtration rates. It is thought to be secondary to reduced production of erythropoietin (EPO) by the kidney. Whilst measurements of EPO levels in CKD are usually normal or slightly reduced they are considered inappropriately low considering the degree of anaemia. Anaemia is associated with an increased morbidity and mortality in patients with CKD.

The introduction of human recombinant EPO has now prevented patients from becoming iron overloaded as a result of multiple transfusions.

The UK renal association recommends that haemoglobin levels should be tested at least annually in CKD patients to identify anaemia. CKD should be considered as a possible cause of anaemia when the glomerular filtration rate is <60ml/min/1.73m . Other causes of anaemia such as blood loss, folic acid and vitamin B12 deficiency should be excluded before the anaemia is attributed to the CKD. The measurement of EPO levels is not necessary for the diagnosis or management of anaemia in CKD patients.

The renal association recommends that treatment with erythropoiesis-stimulating agents (ESAs) should be offered to patients with anaemia of CKD who may benefit in terms of symptoms, quality of life and in order to avoid transfusion in patients considered suitable for future transplantation.

Patients with CKD on ESA therapy should achieve a haemoglobin of between 10-12 g/dL. Adjustments to the patients therapy should be based on the haemoglobin level. In order for ESA to have their intended benefit, patients need to have adequate iron stores for effective erythropoiesis. Less than a third of patients with CKD and anaemia have a satisfactory iron status (ferritin >100ng/ml, transferrin saturation >20%). Patients who are being assessed for ESA therapy need to have their iron status assessed and unless contraindicated are commenced on a regime of parenteral iron therapy in addition to their ESA.

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A 20-year-old man presents to his GP following a pre-employment health check where he was found to have microscopic haematuria. He is not experiencing dysuria or urinary frequency. He is systemically well and has no relevant past medical history.

His blood pressure is 125/74 mmHg, and his cardiovascular, respiratory, and gastrointestinal examinations are unremarkable.

Urinalysis shows 3+ blood and 1+ protein.

Hb	142 g/L	(135-180)
Platelets	272 * 10 /L 9	(150 - 400)
WBC	8.2 * 10 /L 9	(4.0 - 11.0)
Na +	140 mmol/L	(135 - 145)
K +	3.8 mmol/L	(3.5 - 5.0)
Urea	8.1 mmol/L	(2.0 - 7.0)
Creatinine	152 µmol/L	(55 - 120)
CRP	2 mg/L	(< 5)
Urine PCR	180 mg/mmol	

Given the likely diagnosis, what investigation is most likely to be diagnostic?

A. CT kidneys/ureters/bladder
B. Dimercaptosuccinic acid (DMSA) scan
C. Flexible cystoscopy
D. Kidney biopsy
E. Micturating cystogram (MCUG)

D. Kidney biopsy

Routine renal review may be more appropriate than urgent urological review in younger patients with haematuria

In this scenario, the patient presents with microscopic haematuria, sub-nephrotic range proteinuria, and deranged renal function. The most likely diagnosis is IgA nephropathy (though another type of glomerulonephritis is also possible), and therefore, a **kidney biopsy** is most likely to be diagnostic. Any patient under 60 years old presenting with haematuria (provided there are no obvious risk factors for urological malignancy) should be referred to renal services for further investigation and management, especially if there is co-existing proteinuria and reduced kidney function.

A **CT kidneys/ureters/bladder** would be the investigation of choice for potential renal calculi and may also be useful for investigating urothelial malignancy/renal cell carcinoma. Although renal calculi can present with visible haematuria, they are usually also associated with loin-groin pain, which is not present in this case.

A **dimercaptosuccinic acid (DMSA) scan** uses radioisotopes to assess kidney functionality, location, and any evidence of scarring. A DMSA is a useful test in children, especially for detecting scarring due to vesicoureteric reflux (VUR), but it has no role in investigating glomerulonephritis.

In patients presenting with haematuria, urological malignancy must be considered. For older patients (over 60 years old) or younger patients (under 45 years old) at high risk of urological malignancy or whose symptoms have persisted despite treatment of urinary tract infection, **flexible cystoscopy** would be an appropriate investigation. However, in this case, the patient is young and has no reported risk factors for urological malignancy, making a referral to renal and a kidney biopsy more appropriate.

A **micturating cystogram (MCUG)** is used to investigate VUR in the context of recurrent urinary tract infections (UTI), primarily in children, with limited use in adults. In the context of recurrent UTIs, it would demonstrate the backflow of urine into the ureters. Since there is no history of recurrent UTIs in this patient, an MCUG is not indicated.

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A 69-year-old man undergoing continuous ambulatory peritoneal dialysis, with four manual exchanges daily, presents with cloudy peritoneal fluid. There is no history of abdominal pain or systemic symptoms.

On examination, there is minor erythema at the peritoneal dialysis (PD) catheter site. His abdomen is soft and non-tender, with active bowel sounds.

Na +	142 mmol/L	(135 - 145)
K +	5.2 mmol/L	(3.5 - 5.0)
Urea	9.2 mmol/L	(2.0 - 7.0)
Creatinine	350 µmol/L	(55 - 120)
CRP	65 mg/L	(< 5)
Peritoneal fluid microscopy		
WCC	210 WBC/mm (80% polymorphs) 3	(< 100)

Which treatment should be initiated?

A. Intraperitoneal ceftazidime and gentamicin
B. Intraperitoneal vancomycin and oral ciprofloxacin
C. Intravenous vancomycin and aztreonam
D. Oral flucloxacillin and intraperitoneal gentamicin
E. Oral rifampicin and Intraperitoneal ceftazidime

B. Intraperitoneal vancomycin and oral ciprofloxacin

Coagulase-negative *Staphylococcus* is the most common cause of peritonitis secondary to peritoneal dialysis

The patient presents with cloudy peritoneal dialysis fluid, which is highly suggestive of PD peritonitis. A PD fluid count of > 100 WBC/mm , with > 50% polymorphs, confirms the diagnosis. Patients can present with minimal symptoms, and raised CRP is consistent with PD peritonitis. Coagulase-negative *Staphylococcus* is the most common causative organism. Initial treatment is with broad-spectrum antibiotics. Therefore, **intraperitoneal vancomycin and oral ciprofloxacin** is the correct treatment as this provides cover for gram-positive and gram-negative organisms. In some centres, intraperitoneal ceftazidime may be used instead of oral ciprofloxacin.

Intraperitoneal ceftazidime and gentamicin are used in treating PD peritonitis. However, this regimen provides minimal cover against gram-positive organisms. As the most common cause of PD peritonitis is coagulase negative *Staphylococcus*, empirical antibiotic regimens must provide gram-positive cover. Ceftazidime or gentamicin are frequently combined with intraperitoneal vancomycin as an empirical treatment for PD peritonitis, as this will provide broad spectrum coverage.

Although **intravenous vancomycin and aztreonam** would provide broad-spectrum antibiotic coverage, it is less effective than appropriate intraperitoneal antibiotics for PD peritonitis. Therefore, intravenous vancomycin and aztreonam is an incorrect option. Broad-spectrum IV therapy would be a reasonable interim measure if no staff were trained and available to deliver intraperitoneal antibiotics, but it should only be a bridge to appropriate intraperitoneal therapy.

Oral flucloxacillin and intraperitoneal gentamicin would be inappropriate as an initial therapy. Although oral flucloxacillin provides coverage against *Staphylococci*, it does not cover methicillinresistant *Staphylococcus* aureus (MRSA), which is a concern of PD peritonitis. For this reason, this regimen is incorrect.

Oral rifampicin and intraperitoneal ceftazidime is an incorrect answer as this will not provide adequate coverage for gram-positive organisms. Whilst intraperitoneal ceftazidime is commonly used in PD peritonitis, it should be accompanied by intraperitoneal vancomycin otherwise there is insufficient coverage against gram-positive organisms. If microbiological investigations identified *Staphylococcus aureus*, oral rifampicin could be added to intraperitoneal vancomycin, as it may help to disrupt biofilms formed within the PD catheter.

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An 18-year-old man presents to the Emergency Department with a 3-hour history of severe left flank pain radiating to the groin. The pain is constant but waxes and wanes. He tells you he has had this pain before and that it is due to 'kidney stones'.

His electronic hospital record documents three previous admissions with ureteric calculi; the most recent episode requiring intervention with ureteric stents and lithotripsy. He takes no regular medications but consumes 3 litres of water per day on the advice of the urologists.

On examination, he is restless and pacing. He is afebrile, his pulse is 93bpm and his blood pressure is 148/79mmHg. His abdomen is soft but there is percussion tenderness over the left costovertebral angle. A plain abdominal film shows a semi-opaque, left proximal ureteric stone with a 'ground glass' appearance.

His 24-hour urine results are as follows:

Calcium	3.1 mmol/day (2.5 - 7.5)
Oxalate	0.28 mmol/day (0.11 - 0.46)
Phosphate	17.4mmol/day (15 - 20)
Urate	1.8mmol/day (1.5 - 4.5)
Cystine	1.9mmol/day (<0.13)
pH	7.5

Given the likely diagnosis, what is the most appropriate treatment?

A. Allopurinol
B. Potassium citrate
C. Sodium bicarbonate
D. D-penicillamine
E. Bendroflumethiazide

D. D-penicillamine

Recurrent renal stones presenting during the first two decades of life is highly abnormal and should alert you to the possibility of cystinuria; confirmed in this case by the significantly elevated urinary cystine concentration.

The cornerstones of cystinuric stone prevention are hydration and urinary alkalinisation. Sodium bicarbonate was traditionally the agent of choice, however, there is concern about the development of hypertension in patients on long-term treatment. As a result, potassium citrate has become the alkalinising agent of choice. This patient is already drinking 3 litres per day and his pH is already 7.5, so neither of these options is correct.

If hydration and alkalinisation fail, the chelating agent D-penicillamine can be used. It combines with cysteine and renders it 50 times more soluble than cystine. Adverse reactions are common, and the newer agent alpha-mercaptopropionylglycine is better tolerated. Captopril is used in patients with concomitant hypertension.

Bendroflumethiazide is used to lower the risk of calcium stone formation, whilst allopurinol is used in those with uric acid stones.

Question 13 of 183

A 62-year-old woman presents with a 1-week history of a cough productive of sputum with fresh streaks of blood. She is a current smoker but otherwise has no relevant past medical history.

Examination reveals a SpO of 92% on a 28% Venturi mask. Her blood pressure is 160/92 mmHg, and her heart rate is 109 beats per minute. Widespread coarse crackles are noted on auscultation.

Urinalysis shows 2+ protein and 3+ blood.

Hb	103 g/L	(115 - 160)
Platelets	420 * 10 /L 9	(150 - 400)
WBC	7.2 * 10 /L 9	(4.0 - 11.0)
Na +	140 mmol/L	(135 - 145)
K +	5.8 mmol/L	(3.5 - 5.0)
Urea	13.2 mmol/L	(2.0 - 7.0)
Creatinine	689 µmol/L	(55 - 120)
CRP	54 mg/L	(< 5)
Anti-PR3	1.2 IU/ml	(< 2)
Anti-MPO	2 IU/ml	(< 3.5)
Anti-GBM	92 iu/ml	(0.3-7)
C3	0.84 g/L	(0.75-1.65)
C4	0.21 g/L	(0.14-0.54)
ANA	1/40	(< 1/80)

Given the likely diagnosis, what is the most likely finding on kidney biopsy?

A. Crescentic glomerulonephritis with linear capillary wall IgG and C3
B. Crescentic glomerulonephritis without immune staining
C. Diffuse GBM thickening with spikes and foot process effacement
D. Mesangial expansion and glomerular and tubular basement membrane thickening
E. Mesangial proliferation with IgA and C3 deposition

A. Crescentic glomerulonephritis with linear capillary wall IgG and C3

Anti-GBM disease typically presents with haemoptysis + AKI/proteinuria/haematuria

This patient presents with a pulmonary-renal syndrome, characterised by haemoptysis and rapidly progressive glomerulonephritis (RPGN). The widespread coarse crackles heard on auscultation likely indicate alveolar haemorrhage. The primary differential diagnoses are anti-glomerular basement membrane (anti-GBM) disease and ANCA-associated small vessel vasculitis. The rapid onset of symptoms and the patient's age make anti-GBM disease the more likely diagnosis, which is confirmed by a positive anti-GBM titre. A kidney biopsy in this condition would reveal **crescentic glomerulonephritis with linear capillary wall IgG and C3**.

ANCA-associated vasculitis is a key differential in this case; however, it typically presents with a less acute clinical course and is associated with serum ANCA positivity. The presence of a positive anti-GBM titre strongly supports anti-GBM disease. Classically, ANCA-associated vasculitis leads to pauci-immune glomerulonephritis, characterised by the absence of immune deposits on immunofluorescence. Therefore, **crescentic glomerulonephritis without immune staining** would be more indicative of ANCA-associated vasculitis, making it an incorrect answer.

Diffuse GBM thickening with spikes and foot process effacement is the classical histological feature of advanced membranous nephropathy. Membranous nephropathy primarily presents with nephrotic syndrome (in around 80% of cases) rather than RPGN. Additionally, a positive anti-GBM titre is inconsistent with membranous nephropathy, which is typically associated with a positive anti-PLA2R antibody.

Mesangial expansion and glomerular and tubular basement membrane thickening is characteristic of diabetic nephropathy. Diabetic nephropathy typically has a gradual onset, with a slow decline in kidney function and progressively increasing proteinuria, contrasting with the acute RPGN presentation described here which is consistent with anti-GBM disease.

IgA nephropathy, the most common primary glomerulonephritis worldwide, often presents with asymptomatic microscopic urinary abnormalities. The typical biopsy finding in IgA nephropathy is **mesangial proliferation with IgA and C3 deposition**. While IgA nephropathy can occasionally present with an RPGN-like picture, this is uncommon, and the current clinical scenario is more consistent with anti-GBM disease.

Anti-glomerular basement membrane (GBM) disease (Goodpasture's syndrome)

Anti-glomerular basement membrane (GBM) disease (previously known as Goodpasture's syndrome) is a rare type of small-vessel vasculitis associated with both pulmonary haemorrhage and rapidly progressive glomerulonephritis. It is caused by anti-glomerular basement membrane (anti-GBM) antibodies against type IV collagen. Goodpasture's syndrome is more common in men (sex ratio 2:1) and has a bimodal age distribution (peaks in 20-30 and 60-70 age bracket). It is associated with HLA DR2.

Features

- pulmonary haemorrhage
- rapidly progressive glomerulonephritis
  - this typically results in a rapid onset acute kidney injury
  - nephritis → proteinuria + haematuria

Investigations

- renal biopsy: linear IgG deposits along the basement membrane
- raised transfer factor secondary to pulmonary haemorrhages

Management

- plasma exchange (plasmapheresis)
- steroids
- cyclophosphamide

One of the main complications is pulmonary haemorrhage. Factors that increase the likelihood of this include:

- smoking
- lower respiratory tract infection
- pulmonary oedema
- inhalation of hydrocarbons
- young males

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A 44-year-old female attends with worsening widespread joint pain and lethargy. She also has a history of recurrent oral ulcers.

Blood results are as follows:

Hb	110 g/L	Male: (135-180)Female: (115 - 160)
Platelets	124 * 10 /L 9	(150 - 400)
WBC	14.2 * 10 /L 9	(4.0 - 11.0)
Na +	135 mmol/L	(135 - 145)
K +	4.8 mmol/L	(3.5 - 5.0)
Urea	14.2 mmol/L	(2.0 - 7.0)
Creatinine	286 µmol/L	(55 - 120)
CRP	2 mg/L	(< 5)
ANA	1:640	(<1:40)
Anti dsDNA	82 IU/mL	(<10)

A kidney biopsy is arranged:

Kidney biopsy	Diffuse glomerulonephritis involving 60% of glomeruli

You start the patient on high-dose steroids and mycophenolate with a subsequent marked improvement in kidney function.

What maintenance therapy is indicated?

A. Azathioprine
B. Cyclophosphamide
C. Etanercept
D. Mycophenolate
E. Steroids

D. Mycophenolate

Mycophenolate is the preferred follow-on agent in lupus nephritis once there has been a response to the initial therapy

The clinical features and strongly positive ANA and anti-dsDNA confirm a diagnosis of systemic lupus erythematosus. The kidney biopsy results confirm class IV diffuse lupus nephritis.

Mycophenolate is correct. This drug has the best evidence for preventing the development of end-stage renal disease.

Azathioprine is incorrect. Although azathioprine can be used as maintenance, new evidence shows that mycophenolate has better efficacy in preventing end-stage renal disease.

Cyclophosphamide is incorrect. Cyclophosphamide can be used in the acute phase to induce remission, however, it is not used as maintenance.

Etanercept is incorrect. This drug is not used to treat lupus nephritis. There are a growing number of cases of drug-induced lupus as a consequence of anti-tumour necrosis factor (TNF) alpha drugs.

Steroids is incorrect. Steroids are used in the acute phase to induce remission, however, they are not used as maintenance.

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A 75-year-old gentleman is admitted to hospital with worsening productive cough, pyrexia and shortness of breath. On assessment, he appears confused and dehydrated, complaining of increasing muscle pain. He has a temperature of 37.7ºC, a heart rate of 102/min and a blood pressure of 95/65 mmHg. Current medications are: bisoprolol 2.5 mg OD, aspirin 75 mg OD, simvastatin 40 mg OD, ramipril 3.75 mg OD, amlodipine 5 mg OD and allopurinol 300 mg OD. His wife also informs you that his GP recently prescribed a new medication for his productive cough.

His blood picture is the following:

Hb	104 g/l	Na +	131 mmol/l
Platelets	525 * 10 /l 9	K +	6.2 mmol/l
WBC	16.5 * 10 /l 9	Urea	21.1 mmol/l
Neuts	10.0 * 10 /l 9	Creatinine	365 µmol/l
Lymphs	3.2 * 10 /l 9	CRP	79 mg/l
Eosin	0.2 * 10 /l 9	Creatinine Kinase	1325 u/l

Which medication recently prescribed could explain the blood picture above?

A. Co-amoxiclav
B. Clarithromycin
C. Doxycycline
D. Amoxicillin
E. Cefalexin

B. Clarithromycin

Rhabdomyolysis can result from co-prescription of clarithromycin and statins

Statins are metabolised via the CYP3A4 pathway. Drugs that inhibit its action and lead to excess statin toxicity include macrolide antibiotics such as clarithromycin. This combination leads to increase in risk of myopathy and rhabdomyolysis as exhibited by this patient. It is important to note that atorvastatin (as a more hydrophilic agent) is less metabolised by CYP3A4 and hence the side effects of this combination are less profound.

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A 50 year-old man presents with right flank pain. The pain is 9/10 on the pain scale, and does not radiate anywhere. He has also suffered from thirst, polyuria and fatigue, which he has put down to stress from being off work due to a history of chronic lumbar back pain. He has a smoking history of 10 pack years and drinks on average a couple of pints of beer at the weekends. He has no relevant family history. On examination, he has tenderness in the right flank and lower back and his blood pressure is 149/92 mmHg.

Blood tests reveal:

Hb	9.9 g/dL
Platelets	284 * 10 /l9
WBC	10.5 * 10 /l9
Mean corpuscular volume	86 fl
Na +	129 mmol/l
K +	3.9 mmol/l
Urea	16.3 mmol/l
Creatinine	410 µmol/l
Calcium	2.19 mmol/l
Phosphate	1.79 mmol/l
Uric acid	0.40 mmol/l

Urinalysis reveals protein ++, leucocytes +. Microscopy reveals renal papillary cells and casts. No organisms are identified.

A plain abdominal x-ray is negative for calculi and calcification. Intravenous urogram reveals clubbed calyces and ring signs.

What is the most likely diagnosis?

A. Renal calculi
B. Acute interstitial nephritis
C. Pyelonephritis
D. Analgesic nephropathy

D. Analgesic nephropathy

Chronic analgesic use (in this case for the patient's chronic lower back pain) causes tubulointerstitial nephritis and papillary necrosis. The sloughing of the papillae can result in urinary tract obstruction - leading to an acute kidney injury on chronic renal failure. The low sodium level is caused by a salt-losing nephropathy, which is sometimes seen in this condition.

Question 17 of 183

A 35-year-old man of African descent attended for routine review in the HIV clinic. He had been taking highly-active retroviral therapy (HAART) for 5 years but admitted to frequently forgetting to take his tablets.

Examination revealed pitting oedema of both ankles. Urinalysis performed in clinic showed protein 4+ and blood 1+.

Blood tests showed his CD4 count was 140 cells/mm3 (500-1500). Serum complement was within the normal range. Renal biopsy revealed collapsing focal segmental glomerulosclerosis (FSGS).

What is the most likely diagnosis?

A. Tenofovir nephrotoxicity
B. HIV-associated nephropathy (HIVAN)
C. Lupus nephritis
D. HIV-associated immune complex kidney disease (HIVICK)
E. Hepatitis C infection

B. HIV-associated nephropathy (HIVAN)

HIV-associated nephropathy (HIVAN) causes collapsing FSGS and usually presents as nephrotic syndrome

HIV-associated nephropathy (HIVAN) typically presents with nephrotic syndrome and causes a collapsing FSGS on renal biopsy. It is more likely if CD4 count is below 200, as in this case.

HIV-associated immune complex kidney disease (HIVICK) describes immune complex deposition in the kidney which causes a membranoproliferative glomerulonephritis. Typically this would present with nephritic syndrome and low serum complement.

Tenofovir toxicity can cause acute tubular necrosis (ATN) or Fanconi syndrome.

Lupus nephritis and hepatitis C can both present with nephrotic syndrome but a renal biopsy would show membranous or membranoproliferative patterns rather than FSGS.

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A 72-year-old man presents to the Emergency Department with worsening shortness of breath over the last three days. He denies any history of angina or orthopnoea and denies any recent fever. He has a slight cough over the last week and yesterday noted a small amount of blood in his otherwise clear sputum. His past medical history includes type two diabetes mellitus for which he takes metformin and gliclazide. On a more detailed systems review, he admits to feeling generally run down over the last week and has noticed he hasn't been passing much urine in the last two days.

On examination, he is breathless at rest with a respiratory rate of 26 breaths/min, oxygen saturations of 88% on air and a few scattered crepitations on auscultation. His heart rate is 95 beats/min, his blood pressure 169/102 mmHg and his capillary refill time is less than three seconds. His heart sounds are normal, there is no significant peripheral oedema and his mucous membranes are moist. His abdomen is soft to palpation and there is no organomegaly. After urinary catheterization only a small amount of urine is passed and the nurse reports it only showed protein and blood. A chest x-ray reveals widespread patchy infiltrates.

Hb	105 g/l
Platelets	490 * 10 /l9
WBC	8.5 * 10 /l9
Na +	139 mmol/l
K +	5.1 mmol/l
Urea	31.5 mmol/l
Creatinine	894 µmol/l

What is the most appropriate treatment?

A. Plasmapheresis
B. Crystalloid infusion
C. Haemofiltration
D. High dose steroids
E. Dialysis

A. Plasmapheresis

Anti-GBM disease is typically managed with a combination of corticosteroids, cyclophosphamide and plasmapheresis

This gentleman clearly has an acute kidney injury. There is nothing in the history to suggest prerenal failure and although bilateral ureteric obstruction is a possibility that needs to be excluded it is probably less likely than 'intrinsic' renal failure. Given the speed of onset of symptoms and the grossly elevated creatinine a diagnosis of acute glomerulonephritis is likely.

Goodpasture's syndrome is an autoimmune condition where antibodies attack the basement membrane found in the glomerulus and the lung. This results in a glomerulonephritis with acute kidney injury and a nephritic syndrome, and also pulmonary haemorrhage. Pulmonary haemorrhage can present with a variety of symptoms such as breathlessness and chest pain; notably, haemoptysis is by no means universally present. The chest x-ray findings can help differentiate pulmonary haemorrhage from pulmonary oedema that might be found in someone with acute kidney injury with volume overload. The most important treatment is the removal of the antibodies using plasmapheresis. High dose steroids can have a role as part of immunosuppression after initial control and would be used first line if the aetiology was a small vessel vasculitis such as Wegener's granulomatosis. Haemofiltration or dialysis may also be necessary in the interim but there is no indication for immediate use of these services in this case (refractory acidosis, hyperkalaemia, pulmonary oedema). Although treatment with IV fluids may be appropriate to ensure there is no 'pre-renal component' to this acute kidney injury, it is unlikely to be of much help, and given the already raised blood pressure may make pulmonary oedema more likely.

Anti-glomerular basement membrane (GBM) disease (Goodpasture's syndrome)

Anti-glomerular basement membrane (GBM) disease (previously known as Goodpasture's syndrome) is a rare type of small-vessel vasculitis associated with both pulmonary haemorrhage and rapidly progressive glomerulonephritis. It is caused by anti-glomerular basement membrane (anti-GBM) antibodies against type IV collagen. Goodpasture's syndrome is more common in men (sex ratio 2:1) and has a bimodal age distribution (peaks in 20-30 and 60-70 age bracket). It is associated with HLA DR2.

Features

- pulmonary haemorrhage
- rapidly progressive glomerulonephritis
    - this typically results in a rapid onset acute kidney injury
    - nephritis → proteinuria + haematuria

Investigations

- renal biopsy: linear IgG deposits along the basement membrane
- raised transfer factor secondary to pulmonary haemorrhages

Management

- plasma exchange (plasmapheresis)
- steroids
- cyclophosphamide

One of the main complications is pulmonary haemorrhage. Factors that increase the likelihood of this include:

- smoking
- lower respiratory tract infection
- pulmonary oedema
- inhalation of hydrocarbons
- young males

Question 19 of 183

A 65-year-old man presents to the Emergency Department with severe left-sided chest pain. His pulse was 88 and regular and his BP was 114/67 mmHg. His ECG showed sinus rhythm with ST elevation in V1-4. Bloods were taken which showed:

Na +	138 mmol/L	(135 - 145)
K +	4.3 mmol/L	(3.5 - 5.0)
Bicarbonate	25 mmol/L	(22 - 29)
Urea	7.8 mmol/L	(2.0 - 7.0)
Creatinine	136 µmol/L	(55 - 120)
Troponin T	43	(<14ng/L)

The patient was taken for primary percutaneous coronary intervention (PCI) and had 2 stents inserted into the left anterior descending artery. Post-procedure he recovered well and was commenced on dual antiplatelet therapy, ramipril, bisoprolol and high-dose atorvastatin.

His blood tests two days post-procedure show:

Na +	138 mmol/L	(135 - 145)
K +	4.8 mmol/L	(3.5 - 5.0)
Bicarbonate	27 mmol/L	(22 - 29)
Urea	11.9 mmol/L	(2.0 - 7.0)
Creatinine	196 µmol/L	(55 - 120)

Brown casts were seen on urine microscopy. What is the likely cause of his deranged blood tests?

A. Acute interstitial nephritis
B. Cardio-renal syndrome
C. Contrast nephropathy
D. Intrarenal vasoconstriction secondary to ACE inhibitor
E. Prolonged hypotension

C. Contrast nephropathy

known renal impairment (especially diabetic nephropathy)

Acute kidney injury following a percutaneous coronary intervention→ ?contrast-induced nephropathy

Muddy brown casts are indicative of acute tubular necrosis (ATN). ATN occurs due to damage to renal epithelial cells. This may be ischaemic or toxic in its aetiology. Ischaemic causes are prolonged hypotension such as during surgery or sepsis. Toxic causes include myoglobin from rhabdomyolysis/massive haemolysis, IV contrast, hyperuricaemia (tumour lysis syndrome), poisons (ethylene glycol, mercury) or drugs (aminoglycosides, NSAIDs, chemotherapy agents etc.). Given the presence of muddy brown casts and the timeline of events, the likely diagnosis is acute tubular necrosis and the only cause of this listed is **contrast nephropathy** which can occur following the use of IV contrast in percutaneous coronary intervention (PCI).

Intrarenal vasoconstriction secondary to ACE inhibitor is incorrect as, although ACE inhibitors can cause AKI, the mechanism is not via ATN and therefore not correct in this instance.

Acute interstitial nephritis is not characterised by muddy brown casts and there is no causative agent listed in this history and therefore not correct. Common causes of acute interstitial nephritis include antibiotics, NSAIDs, aciclovir, omeprazole and infections - though there are many more.

Cardio-renal syndrome is characterised by decreased kidney function, diuretic-resistant heart failure with congestion, and worsening kidney function during treatment for heart failure. As there is no history of heart failure for this patient this is incorrect.

Though **prolonged hypotension** is a cause of ATN, there is no significant history of prolonged hypotension in this case and therefore, the most likely cause of the ATN in this instance is contrast nephropathy following PCI.

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A 29-year-old woman with a background of type 1 diabetes was admitted to the hospital with acute onset abdominal pain and vomiting. On examination, she was tachycardic and hypotensive with generalised abdominal tenderness.

Her initial blood tests showed the following:

pH	7.25	(7.35 - 7.45)
pO2	12.1 kPa	(11 - 14.4)
pCO2	5.7 kPa	(4.6 - 6.4)
Sodium	138 mmol/L	(135 - 145)
Potassium	4.5 mmol/L	(3.5 - 5.5)
Chloride	99 mmol/L	(95 - 108)
Bicarbonate	13 mmol/L	(22 - 29)
Glucose	26.9 mmol/L	(4 - 7)
Lactate	3.9 mmol/L	(0.5 -2.2)
Ketones	5.1 mmol/L	(< 0.6)

She was admitted to the endocrinology ward and started on a fixed-rate insulin scale. After 2 days of treatment, she still reports being unwell.

A repeat arterial blood gas is taken:

pH	7.28	(7.35 - 7.45)
pO2	11.3 kPa	(11 - 14.4)
pCO2	4.7 kPa	(4.6 - 6.4)
Sodium	150 mmol/L	(135 - 145)
Potassium	3.0 mmol/L	(3.5 - 5.5)
Chloride	114 mmol/L	(95 - 108)
Bicarbonate	23 mmol/L	(22 - 29)
Glucose	6.9 mmol/L	(4 - 7)
Lactate	1.9 mmol/L	(0.5 -2.2)
Ketones	0.5 mmol/L	(< 0.6)

What is the most likely cause of this patient's presentation?

A. Renal tubular acidosis type 4
B. Sodium chloride administration
C. Spironolactone use
D. Starvation ketoacidosis
E. Unresolved diabetic ketoacidosis

B. Sodium chloride administration

Aggressive treatment with 0.9% saline in DKA commonly causes a hyperchloraemic metabolic acidosis

This patient has a likely diagnosis of diabetic ketoacidosis (DKA) given the raised glucose, ketonaemia, low bicarbonate and low pH on laboratory testing. The most initial management option is to deliver rapid fluid resuscitation with 0.9% sodium chloride. However, 0.9% sodium chloride contains 154 mmol/L of sodium and 154 mmol/L of chloride. In contrast, the plasma concentration of sodium and chloride are 135-145 mmol/L and 95-108 mmol/L, respectively. The repeat blood gas shows raised plasma sodium and chloride concentrations which is likely to reflect the increased concentrations delivered during initial fluid resuscitation.

It is important to assess the anion gap when assessing a patient with metabolic acidosis. The anion gap on the second blood gas is normal for this patient:

[Na (150) + K (3)] - [Cl (114) + HCO3 (23)] = 16 mmol/L (10 - 18).

Therefore, **sodium chloride administration** is the most likely cause of this patient's deranged blood results.

Renal tubular acidosis type 4 causes a normal anion gap metabolic acidosis. The clinical features are associated with reduced levels of aldosterone that, in turn, causes reduced ammonium excretion and hyperkalaemia. Although it is associated with other underlying conditions such as diabetes, it is a less likely cause of this patient's presentation given the hypokalaemia and raised sodium and chloride levels post-fluid resuscitation.

Spironolactone use is incorrect. Spironolactone is an aldosterone antagonist used in the treatment of heart failure and liver cirrhosis. Although its use can be linked to a normal anion gap metabolic acidosis, there is nothing to suggest in the clinical history that this patient would be taking spironolactone. It also does not explain the patient's hypokalaemia.

Starvation ketoacidosis is incorrect. It can take some time for patients with DKA to resume eating and drinking and glucose levels can be normal in a patient with starvation ketoacidosis. However, the absence of significantly raised proteins excludes this as a likely diagnosis.

Unresolved diabetic ketoacidosis is incorrect as the blood sugars and ketone levels have improved. Although the bicarbonate is not completely back to normal, it has made a significant improvement and may still be reduced secondary to other ongoing complications.

Metabolic acidosis

Metabolic acidosis is commonly classified according to the anion gap. This can be calculated by: (Na + K) - (Cl + HCO). If a question supplies the chloride level then this is often a clue that the anion gap should be calculated. The normal range = 10-18 mmol/L

Normal anion gap (= hyperchloraemic metabolic acidosis)

- gastrointestinal bicarbonate loss:
  - prolonged diarrhoea: may also result in hypokalaemia
  - ureterosigmoidostomy
  - fistula
- renal tubular acidosis drugs: e.g. acetazolamide
- ammonium chloride injection
- Addison's disease

Raised anion gap

- lactate:
  - shock
  - sepsis
  - hypoxia
- ketones:
  - diabetic ketoacidosis
  - alcohol
- urate: renal failure
- acid poisoning: salicylates, methanol

Metabolic acidosis secondary to high lactate levels may be subdivided into two types:

- lactic acidosis type A: sepsis, shock, hypoxia, burns
- lactic acidosis type B: metformin

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An 18-year-old male presented to the Emergency Department complaining of blood in his urine for the last day. He described it initially as few drops of dark blood at the end of micturition but has gradually worsened such that at the time of admission he was passing large amounts of cola coloured urine. He also complained of swollen ankles and puffy eyes developing over the last few hours and had been feeling increasing lethargic and unwell over the last two days. When questioned specifically he denied the presence of shortness of breath, chest pain, haemoptysis, previous haematuria or change in urine volume and had otherwise been well in himself. He had no previous renal problems, though he recalled that his brother had been prescribed a course of steroids when he was aged nine years old for leaky kidneys. Other than a one week course of phenoxymethylpenicillin 500mg QDS prescribed by his GP two weeks ago for tonsillitis and a one week course of amoxicillin 500mg TDS prescribed 6 months ago for acute sinusitis he had no drug history and no past medical history of note.

Examination revealed the presence of a young athletic male with a blood pressure of 162/84 mmHg, heart rate of 96bpm, respiratory rate of 18/min, oxygen saturations of 95% on air and temperature of 37.1 Celsius. Examination of the cardiovascular system revealed the presence of normal heart sounds, a JVP of 3cm and the presence of bilateral pitting oedema of his ankles. Examination of his respiratory system was unremarkable with no signs of respiratory distress. Examination of his gastrointestinal system was unremarkable. Examination of his face revealed the presence of bilateral periorbital oedema; examination of his ENT and neurological systems were both unremarkable.

Initial investigation revealed the following results:

Hb	132 g/l
Platelets	428* 10 /l9
WBC	14.2 * 10 /l9
ESR	26 mm/hr
Na +	138 mmol/l
K +	5.2 mmol/l
Urea	6.4 mmol/l
Creatinine	77 µmol/l
CRP	18 mg/l
Bilirubin	18 µmol/l
ALP	82 u/l
ALT	21 u/l
Protein	78 g/l
Albumin	39 g/l

Chest x-ray: normal appearance of heart and lung fields ECG: sinus tachycardia 108bpm

Urinalysis: blood ++++ protein ++++, ketones +, negative all other parameters Urine MCS: awaiting result

Blood MCS: awaiting result

What is the most likely diagnosis?

- A. Rapidly progressive glomerulonephritis
- B. Berger's nephropathy
- C. Granulomatosis with polyangiitis
- D. Post streptococcal glomerulonephritis
- E. Membraneous nephropathy

D. Post streptococcal glomerulonephritis

This gentleman has developed post-streptococcal glomerulonephritis (PSGN), an immunemediated reaction in response to the presence of specific strands of Streptococcus spp. The latent period is classically one to two weeks post infection and presents with an acute glomerulonephritis characterised by haematuria (with dark urine owing to the haemolysis of red blood cells that have penetrated the glomerular basement membrane), proteinuria, oedema with or without oliguria. A key differential diagnosis is Berger's nephropathy, the most common form of adult glomerulonephritis. This tends to present one to two days post a non-specific upper respiratory tract infection and is characterised predominantly by haematuria, with an usually excellent prognosis. Rapidly progressive glomerulonephritis is associated with a rapid decline of renal function but may present with similar features to PSGN, whilst membraneous nephropathy is characterised by a nephrotic syndrome.

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A 52-year-old female with a history of systemic lupus erythematosus presents to the emergency department with 4 days history of high-grade fever and productive cough. She recently travelled to France with her husband and has been feeling unwell ever since she got back home.

Her medication history included prednisolone 10mg daily, and hydroxychloroquine 200mg twice daily which she has been taking for the last 12 years. She is additionally on once-yearly zoledronic acid and occasional naproxen.

On examination, she has a temperature of 38.5°C and a pulse of 130 bpm which is regular and low volume. Her blood pressure in 80/50 mmHg and she has cold peripheries. She has a confluent rash on her cheeks with nasolabial sparing. Examination of her respiratory system reveals a patch of bronchial breathing in the left middle of the chest. The remaining physical examination is essentially unremarkable.

Laboratory investigations reveal:

Hb	110 g/dl
MCV	85 fl
MCH	24 pg (27 - 32 pg)
WBC	17 * 10 /l9
Plt	450 * 10 /l9
Urea	14.9 mmol/l
Creatinine	298µmol/l
Na+	128 mmol/l
K+	3.9 mmol/l
Albumin	32g/l

Urine dipstick shows protein 1+ Urinary electrolytes reveal:

Urinary specific gravity	1.031 (1.010 - 1.020)
Urinary osmolality (mOsm/kg)	675 (350 - 500)
Urinary sodium (mmol)	8 (20 - 40)
Fe_{Na}	0.2% (1%)

Which of the following is the most appropriate initial management option?

- A. IV hydration with 0.9% saline
- B. Increase prednisolone to 1mg/kg/day
- C. Start mycophenolate mofetil
- D. IV hydration with 3% saline
- E. Commence inotropic support with dobutamine

A. IV hydration with 0.9% saline

The scenario highlights the candidates understanding of the approach to the management of prerenal azotaemia. The patient described is dehydrated, owing to her fever and insensible fluid losses, due to which she is hypotensive. Her urinary electrolytes show that her kidneys are functioning as one would expect them to in the face of impaired renal perfusion, i.e. they are producing concentrated urine and retaining sodium. The derangement of renal function is due to the impaired renal blood flow and is pre-renal in nature.

All causes of pre-renal azotaemia may lead to acute kidney injury and by definition, excretory function improves once normal renal perfusion has been restored. The criteria for distinction between pre-renal azotaemia and intrinsic renal dysfunction are highlighted below:

Although she suffers from SLE and her renal impairment may be secondary to lupus nephritis, the urinary electrolytes suggest a pre-renal component rather than an intrinsic parenchymal pathology.

Question 23 of 183

A 49-year-old man was brought in by ambulance after he had a prolonged tonic-clonic seizure for 45 minutes. He had no previous history of seizures, and this episode was witnessed by people passing by, who claimed he was jerking throughout his body and wet himself during the course of the fit. On admission, he was postictal after. His seizures stopped after administration of rectal diazepam, phenytoin infusion and intubation, and he was admitted to the intensive care unit. He remained drowsy and lethargic for the next 2 days, and attempts to wean him off the ventilator was slow.

His blood results on day 2 showed:

Na+	140 mmol/l
K+	5.8 mmol/l
Urea	11.1 mmol/l
Creatinine	180 µmol/l
Serum corrected calcium	2.15 mmol/l
Serum bilirubin	21 µmol/l
Serum alkaline phosphatase	97 IU/l
Serum aspartate aminotransferase	62 IU/l
Creatine kinase (CK)	9000 IU/l
Serum lactate	4.5 mmol/l
C-Reactive protein (CRP)	5 mg/l
Haemoglobin	13.6 g/dl
White cell count	7.2 x 10^9/L
INR	1.0

Which of the following values would likely be seen on his arterial blood gas result?

A. pH 7.25 pO2 16 kPa pCO2 4.6 kPa HCO3 11mmol/l BE -10mmol/l, Cl- 101mmol/l
B. pH 7.46 pO2 16 kPa pCO2 2.4 kPa HCO3 31mmol/l BE 5mmol/l, Cl- 101mmol/l
C. pH 7.29 pO2 15 kPa pCO2 6.6 kPa HCO3 36mmol/l BE 6mmol/l Cl- 111mmol/l
D. pH 7.48 pO2 17 kPa pCO2 6.4 kPa HCO3 11mmol/l BE -10mmol/l, Cl- 101mmol/l
E. pH 7.36 pO2 17 kPa pCO2 4.6 kPa HCO3 27mmol/l BE -2mmol/l , Cl- 101mmol/l

A. pH 7.25 pO2 16 kPa pCO2 4.6 kPa HCO3 11mmol/l BE -10mmol/l, Cl- 101mmol/l

This man has rhabdomyolysis, as evidenced by his acute kidney injury, and markedly raised CK values, secondary to status epilepticus. Rhabdomyolysis is the clinical syndrome resulting from the breakdown of muscle cells, resulting in the release of the intracellular contents into the circulation eg. myoglobin and potassium, resulting in electrolyte imbalances and acute kidney injury. It can also be caused by crush injuries, overexertion, compartment syndrome, drugs eg. statins, neuroleptic malignant syndrome.

In severe cases, it results in metabolic acidosis secondary to raised serum lactic acid levels from the ischaemic muscle fibres. The serum lactate is raised which would suggest an acidotic picture over a normal blood gas picture, ruling out options (b), (d), (e). Option D is a spurious result - it is alkalotic despite a high pCO2 and low HCO3. Option (c) is a picture of respiratory acidosis, leaving option (a) as the only correct answer.

Question 24 of 183

A 58-year-old lady with an extensive psychiatric history presents to the Acute Medical unit unwell and dehydrated. She recently had a viral illness and has spent most of the past week in bed. She is mildly confused on arrival and the ambulance sheet states that for the past two weeks she has been passing urine more frequently, particularly at night. She denies any dysuria or fevers.

She has a history of bipolar affective disorder which is currently stable on lithium. She also has a history of irritable bowel syndrome, hypertension, hypercholesterolaemia and migraines. She currently takes amlodipine 10mg once a day, simvastatin 20mg at night, mebeverine 135mg three times a day and lithium carbonate 800mg at night. She has no known drug allergies.

On examination, she looks unwell and dehydrated. Her pulse is 105 bpm and regular, blood pressure 95/65 mmHg, saturations of 98% on air. Numerous linear scars are apparent on both her arms and the tops of her thighs. Her mucous membranes are dry and her JVP is not visible at 45 degrees. Her chest was clear. Abdominal examination demonstrated a soft, non-tender abdomen with no masses or organomegaly and normal bowel sounds.

A set of bloods come back as follows:

Na+	153 mmol/L
K+	4.9 mmol/L
Urea	8 mmol/L
Creatinine	110 µmol/L
Hb	160 g/L
WBC	6.0x10^9/L
LFTs	Normal
Urine osmolality	180 mOsmol/kg

Chest x-ray shows clear lung fields.

Considering the most likely diagnosis which of the following treatments are most likely to treat the underlying condition?

A. Hypertonic saline resuscitation
B. Low dose DDAVP
C. Observation
D. Thiazide diuretic and amiloride
E. 1.5L fluid restriction

D. Thiazide diuretic and amiloride

Diabetes insipidus is characterised by a **high plasma** osmolality and a **low urine** osmolality

This lady has a history of bipolar disorder which has been well controlled with lithium. She presents following a viral illness where her mobility and possibly fluid intake has been reduced. She has been passing large quantities of urine associated with a very low (diluted) urine osmolality. She is very dehydrated and hypovolaemic with associated hypernatraemia which may explain the mild confusion.

Nephrogenic diabetes insipidus (DI) is a relatively common side effect of lithium therapy and usually presents with polyuria and polydipsia. Severe dehydration only presents if patients have struggled to keep up with the oral intake as is the case here.

Unlike cranial DI, nephrogenic DI is not usually responsive to low doses of desmopressin (DDAVP) although mild to moderate cases have been successfully treated with high doses.

The patient is clearly very dehydrated, therefore fluid restriction is likely to worsen her condition. Equally, in view of her hypernatraemia, the use of hypertonic saline will likely worsen this.

Observation is likely to result in further deterioration. Ideally, the case should be discussed with the psychiatrists and the lithium discontinued and she should be encouraged to drink plenty of fluids.

As she is markedly dehydrated and affected by her DI, treatment with thiazide diuretics (which produce a paradoxical concentration of the urine) and amiloride has been shown to be effective. This is, therefore, the only correct answer here.

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A 22-year-old female presents to the Emergency Department with blood in her urine. She produces a sample, demonstrates painless rose coloured macroscopic haematuria. She reports no past medical history except a single episode of a urinary tract infection about 5 years ago and a recent 'cough and sore throat' that had got better two weeks ago. Her family has no history of bladder or kidney problems except her mother having 'shockwave treatment to her kidney tubes a few years ago'. She is sexually active with a regular partner, her last menstrual period was 3 weeks ago. On examination, her abdomen is soft and non-tender with no organomegaly. Urine dip demonstrates 4+ blood, 1+ protein. Her blood tests are as follows:

Hb	125 g/l
Platelets	259 * 10 /l9
WBC	12.1 * 10 /l9
Na +	139 mmol/l
K +	4.8 mmol/l
Urea	5.6 mmol/l
Creatinine	72 µmol/l
CRP	3 mg/l
Beta HCG	negative
HIV	negative
CMV IgG	positive
CMV IgM	negative
EBV IgG	positive
Anti-streptolysin titre	positive

Chest radiography is unremarkable. Ultrasound of her renal tract shows normal sized kidneys with no hydronephrosis.

What is the diagnosis?

A. IgA nephropathy
B. Diffuse proliferative glomerulonephritis
C. Minimal change disease
D. Membranoproliferative glomerulonephritis
E. Ureteric calculi

B. Diffuse proliferative glomerulonephritis

A recent respiratory tract infection should alert you to mesangioproliferative glomerulonephropathy, caused by IgA deposition acutely following an upper respiratory tract infection, and diffuse proliferative glomerulonephritis, caused by reduced C3 and diffuse glomerular proliferation around 2 to 3 weeks after a streptococcal infection. Minimal change disease classically results in significant proteinuria due to loss of small protein molecules through the deficient basement membrane. She has no risk factors for membranoproliferative glomerulonephritis, most commonly hepatitis C, cryoglobulinaemia and lipodystrophy.

Question 26 of 183

A 32 year-old man presented to his GP with dark coloured urine. Two weeks previously he had a sore throat but didn't see his GP at that time. He had no past medical history or family history of note.

On examination, his pulse was 60 beats per minute and his blood pressure was 160/95 mmHg. Abdominal examination was unremarkable. Urinalysis showed blood 3+ and protein 1+.

What is the most important diagnostic investigation?

A. Antinuclear antibodies
B. Anti glomerular basement membrane antibodies
C. Serum electrophoresis
D. Anti-neutrophil cytoplasmic antibodies
E. Antistreptolysin O titre

A. Antistreptolysin O titre

The diagnosis in this case is post-infectious glomerulonephritis which classically occurs two to three weeks after a streptococcal throat infection. The most useful test of the available options is Antistreptolysin O titre which would confirm the presence of anti-streptococcal antibodies.

Question 27 of 183

A 72-year-old man presents to the emergency department with frank haemoptysis. He has no past medical history. He smokes 20 cigarettes daily.

Observations:

- Spo2 95% on room air
- Respiratory rate 18/minute
- Temperature 37C
- Blood pressure 101/65 mmHg Heart rate 88 beats per minute

The examination is unremarkable. Bloods:

Hb	82 g/L	Male: (135-180)Female: (115 - 160)
Platelets	189 * 10 /L 9	(150 - 400)
WBC	4.2 * 10 /L 9	(4.0 - 11.0)
Na +	138 mmol/L	(135 - 145)
K +	5.1 mmol/L	(3.5 - 5.0)
Urea	14.2 mmol/L	(2.0 - 7.0)
Creatinine	302 µmol/L	(55 - 120)
CRP	55 mg/L	(< 5)

Urinalysis:

Blood	+++
Protein	+++
Glucose	-ve
Leucocytes	-ve
Nitrites	-ve

A chest x-ray demonstrates bilateral coalescent airspace opacification.

A renal biopsy is undertaken, which demonstrates linear IgG deposits along the basement membrane.

Given the likely diagnosis, what is the appropriate treatment?

A. Corticosteroids
B. Corticosteroids and plasmapheresis
C. Corticosteroids, cyclophosphamide and plasmapheresis
D. Corticosteroids, rituximab and cyclophosphamide
E. Corticosteroids, rituximab and plasmapheresis

C. Corticosteroids, cyclophosphamide and plasmapheresis

Anti-GBM disease is typically managed with a combination of corticosteroids, cyclophosphamide and plasmapheresis

Corticosteroids, cyclophosphamide and plasmapheresis is the correct answer. This elderly smoker presents with haemoptysis, anaemia and bilateral airspace opacifications (suggestive of pulmonary haemorrhage) along with a probable acute kidney injury and nephritis (haematoproteinuria). These findings, together with a characteristic biopsy, suggest a diagnosis of anti-glomerular basement membrane disease (anti-GBM). This is an autoimmune disease that targets the capillary beds of the lungs and kidneys. It is caused by the development of autoantibodies directed against autoantigens on the basement membranes of these particular organs. The accepted consensus treatment for this condition is urgent plasmapheresis to rapidly remove culprit antibodies and subsequent CYC and prednisolone treatment to inhibit autoantibody production and resolve inflammation.

Corticosteroids is incorrect. This approach has been shown to be insufficient in anti-GBM disease. Plasma exchange is required to remove the culprit autoantibodies and further immunosuppression is required with cyclophosphamide to inhibit further autoantibody production.

Corticosteroids and plasmapheresis is incorrect. Prevention of further antibody synthesis with CYC is an important component of management in this condition.

Corticosteroids, rituximab and cyclophosphamide is incorrect. An accepted component of management of this condition is the urgent removal of culprit pathogenic autoantibodies by plasma exchange. This regimen can be used in life-threatening ANCA vasculitis.

Corticosteroids, rituximab and plasmapheresis is incorrect. There is some suggestion that rituximab may be effective in anti-GBM disease and it may be an option in relapsing disease. However, it is not first-line treatment and CYC should be used in preference initially.

Anti-glomerular basement membrane (GBM) disease (Goodpasture's syndrome)

Anti-glomerular basement membrane (GBM) disease (previously known as Goodpasture's syndrome) is a rare type of small-vessel vasculitis associated with both pulmonary haemorrhage and rapidly progressive glomerulonephritis. It is caused by anti-glomerular basement membrane (anti-GBM) antibodies against type IV collagen. Goodpasture's syndrome is more common in men (sex ratio 2:1) and has a bimodal age distribution (peaks in 20-30 and 60-70 age bracket). It is associated with HLA DR2.

Features

- pulmonary haemorrhage
- rapidly progressive glomerulonephritis
    - this typically results in a rapid onset acute kidney injury
    - nephritis → proteinuria + haematuria

Investigations

- renal biopsy: linear IgG deposits along the basement membrane
- raised transfer factor secondary to pulmonary haemorrhages

Management

- plasma exchange (plasmapheresis)
- steroids
- cyclophosphamide

One of the main complications is pulmonary haemorrhage. Factors that increase the likelihood of this include:

- smoking
- lower respiratory tract infection
- pulmonary oedema
- inhalation of hydrocarbons
- young males

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A 42-year-old man attended for routine review in the HIV clinic. He had been taking highly-active retroviral therapy (HAART) regularly for 5 years. Over the past few weeks he had experienced intermittent left flank pain radiating to his groin.

Urinalysis performed in clinic showed blood 3+ but no protein. Urine microscopy showed needleshaped crystals.

What is the most likely diagnosis?

A. HIV-associated immune complex kidney disease (HIVICK)
B. Tenofovir nephrotoxicity
C. HIV-associated nephropathy (HIVAN)
D. Renal vein thrombosis
E. Indinavir-induced nephrolithiasis

E. Indinavir-induced nephrolithiasis

Renal complications of HAART: Indinavir can cause renal stones

Indinavir can crystallise in the urine and cause renal stones. Urine microscopy shows needleshaped crystals.

Tenofovir typically causes acute tubular necrosis or Fanconi syndrome.

HIV-associated nephropathy (HIVAN) causes a collapsing FSGS and presents with nephrotic syndrome.

HIV-associated immune complex kidney disease (HIVICK) can cause a nephritic syndrome but wouldn't present with renal colic.

Renal vein thrombosis can present with flank pain but wouldn't explain the finding of crystalluria.

Question 29 of 183

A 36 year old female patient presents to the endocrinology clinic to see you for follow up. She reports a 1 year history of thirst and drinking a lot of water. She has had a normal fasting glucose and oral glucose tolerance test. On her previous visit, you booked the patient for a water deprivation test and the results are available:

Time	Urine osmolality (mOsm/kg)	Serum osmolality (mOsm/kg)	Weight (kg)	% change in weight
0800	263	263	86.1	n/a
1000	265	278	85.7	-0.46%
1200	279	289	85.4	-0.81%
1400	280	295	84.9	-1.39%
1600	285	301	84.6	-1.74%
1700 DDAVPgiven	-	-		
1800	286	302	84.3	-2.09%
2000	289	303	84.1	-2.32%

What is the underlying cause of this lady's symptoms?

A. Nephrogenic diabetes insipidus

B. Psychogenic polydipsia
C. Cranial diabetes insipidus
D. Partial cranial diabetes insipidus
E. None of the above

A. Nephrogenic diabetes insipidus

Diabetes insipidus is characterised by a **high plasma** osmolality and a **low urine** osmolality

This lady has symptoms of polydipsia and thirst. The differentials of this are diabetes insipidus, psychogenic polydipsia and diabetes mellitus (which is ruled out in the question body). The water deprivation test is used when diabetes insipidus is suspected and helps to differentiate between nephrogenic diabetes insipidus (kidney is not sensitive enough to ADH release by the posterior pituitary), cranial diabetes insipidus (lack of release of ADH from the posterior pituitary, kidney sensitive to exogenous ADH when administered) and psychogenic polydipsia (a normal outcome to the water deprivation test). In this case we can see that as the patient is deprived of water, their serum osmolality rises, as expected. However, the urine does not concentrate that much, and there is minimal, if any response by the kidneys to DDAVP. This indicates a nephrogenic diabetes insipidus as psychogenic polydipsia would show concentrated urine much earlier in the test with high urine osmolalities and in cranial diabetes insipidus, the kidneys would show a much greater response to DDAVP by concentrating the urine.

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A 25-year-old male with no significant past medical history presents with a 4-month history of palpitations. Upon further questioning, he also notes an unintentional 7 kg weight loss and some heat intolerance over the same time frame.

Clinical examination reveals mild thyromegaly, fine tremor, brisk reflexes, and bilateral lid lag. He does not have any exophthalmos and the rest of the examination is unremarkable. He denies any family history of thyroid disease. He has thyroid function tests which reveal a low Thyroidstimulating Hormone (TSH), elevated Free thyroxine (FT4) and Free Triiodothyronine (FT3), and negative anti-TPO antibodies. Thyroid uptake scan demonstrates diffusely increased uptake.

At a follow-up visit to discuss the above results he reports that he may have discovered a mass on his testicle. Your exam confirms this finding. At this point, in addition to basic labs and imaging to further evaluate this testicular mass,

what laboratory test would be necessary to further evaluate the aetiology behind his hyperthyroidism?

A. TSH
B. Human chorionic gonatropin (hCG)
C. Thyroid Stimulating Antibodies
D. Lactate Dehydrogenase (LDH)
E. Alpha Feto Protein (AFP)

B. Human chorionic gonatropin (hCG

With hyperthyroidism and a testicular mass, this patient may have a germ cell tumour causing his hyperthyroidism. These tumours have the potential to secrete hCG, LDH, and/or AFP. hCG is homologous to TSH in structure and therefore can lead to hyperthyroidism. This patient has negative TPO antibodies so it is unlikely that he has autoimmune thyroid disease such as Graves Disease, which would have positive thyroid-stimulating Antibodies (TSA). Thyroglobulin level would be unhelpful in this setting and is frequently used in patients with thyroid cancer to assess for any residual thyroid tissue after thyroidectomy and ablation. And while this patient should certainly have LDH and AFP levels checked they are unlikely to

Question 31 of 183

A 21-year-old man presents with visible haematuria. This was preceded by 2 days of sore throat and coryzal symptoms. He has no significant past medical history. An examination is unremarkable and he appears systemically well.

Blood tests are taken and show the following:

Na +	138 mmol/L	(135 - 145)
K +	3.7 mmol/L	(3.5 - 5.0)
Urea	5.3 mmol/L	(2.0 - 7.0)
Creatinine	72 µmol/L	(55 - 120)

A urine sample is also collected over 24 hours:

24-hour urinary protein	1150 mg/day	(<100)

Another urinary protein sample is collected several days later and demonstrates a similar result

. What is the correct initial treatment?

A. Cyclophosphamide
B. No treatment
C. Phenoxymethylpenicillin
D. Prednisolone
E. Ramipril

E. Ramipril

IgA nephropathy management

- no proteinuria, normal GFR: observe
- proteinuria: ACE inhibitor
- signifcant fall in GFR/not responding to ACE inhibitor: corticosteroid

This patient's history is highly suggestive of IgA nephropathy - haematuria classically following 1-2 days of an upper respiratory tract infection (URTI). Patients may also have a degree of proteinuria, although this is rarer in IgA nephropathy than in post-streptococcal glomerulonephritis. The severity dictates management. In this case, there is some proteinuria, but the renal function is preserved as per the blood tests, and so **ramipril**, an angiotensin-converting enzyme (ACE) inhibitor, should be initiated.

If there were severe renal impairment, significant proteinuria, and haematuria, and it was not responding to steroids, then **cyclophosphamide** may be added. This is an aggressive immunosuppressant with significant side effects.

No treatment is inappropriate in this scenario. Renal function is preserved, but there is some proteinuria present. If the proteinuria were minimal instead, observation would be appropriate.

Phenoxymethylpenicillin would likely be given if the diagnosis were post-streptococcal glomerulonephritis (PSGN). There is some overlap between IgA nephropathy and PSGN, but the main symptom in PSGN tends to be proteinuria rather than haematuria. There is typically a much longer interval between the URTI and renal symptoms - 1-2 weeks as opposed to days.

Prednisolone, an oral steroid, would be appropriate if the renal function was dropping, or the patient was not responding to an ACE inhibitor. In this scenario, an ACE inhibitor should be tried first as renal function is preserved.

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A 72-year-old man attends the emergency department with a 24-hour history of diarrhoea. He has a background of osteoarthritis, benign intracranial hypertension and chronic kidney disease. Blood results are as follows.

Hb 142 g/L (135-180)
Platelets 395 * 10 /L (150 - 400)
WBC 10 * 10 /L (4.0 - 11.0)
Na 144 mmol/L (135 - 145)
K 4.8 mmol/L (3.5 - 5.0)
Urea 6.4 mmol/L (2.0 - 7.0)
Creatinine 135 µmol/L (55 - 120)
CRP 5 mg/L (< 5)
Lactate 1.4 mmol/L (0.7-1.8)
pH 7.24 7.35 - 7.45
BE -7 -2 - +2

You calculate his anion gap at 20mmol/L.

What is the most likely cause of this abnormality?

A. Acetazolamide
B. Diarrhoea
C. Paracetamol
D. Renal tubular acidosis
E. Sepsis

C. Paracetamol

Chronic paracetamol use can lead to raised anion gap metabolic acidosis caused by 5- oxoproline

The correct answer is **paracetamol**. This is the only option associated with a raised anion gap metabolic acidosis. This specific entity caused by paracetamol is known as pyroglutamic acidosis.

This results from reduced levels of glutathione, resulting in the accumulation of 5-oxoproline. It is more common in women and has also been associated with chronic renal insufficiency, malnourishment and co-administration of some medications such as flucloxacillin.

Acetazolamide is incorrect. This is a carbonic anhydrase inhibitor sometimes used in the treatment of benign intracranial hypertension and has been associated with metabolic acidosis. However, this is generally a normal anion gap metabolic acidosis.

Diarrhoea is incorrect. Gastrointestinal losses, such as diarrhoea, are associated with normal anion gap acidosis. This results from the loss of bicarbonate.

Renal tubular acidosis is incorrect. This refers to a spectrum of disorders where the kidneys do not effectively remove acid from the blood. Type 4 RTA is the most common and is associated with hyperkalaemia. This patient's potassium is normal, making this less likely. RTA is also a cause of normal anion gap acidosis, rather than the raised anion gap this patient has developed.

Sepsis is incorrect. Sepsis is a common cause of metabolic acidosis, usually resulting from tissue hypoperfusion and lactic acidosis. Although this would cause a raised anion gap, this patient's inflammatory markers and lactate are normal, excluding this as a diagnosis.

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A 24-year-old man was referred to clinic with an enlarged testicle. The patient reports that he was examining himself in the shower last week and noticed that his left testicle was markedly larger than the right. He has no specific symptoms, but does describe 4kg weight loss in the last 4 months, which he had previously attributed this to a new diet. Furthermore, he has also suffered with general fatigue over the last month.

He has no past medical history and takes no regular medication. In terms of social history, he is sexually active with his partner, with whom he has been in a relationship for 3 years now. He does not drink alcohol and smoke, and denies any recreational drug use.

There are no other symptoms of note on systems enquiry.

Other than an enlarged, non-tender, left testicle, clinical examination did not reveal any abnormality. Specifically, there was no palpable lymphadenopathy and no gynaecomastia.

What is the most appropriate next step in evaluating this patient?

A. Ultrasound testes
B. Lactate dehydrogenase
C. MRI testes
D. α-FP
E. β-hCG

A. Ultrasound testes

The first-line investigation of a testicular mass is an ultrasound

It is normal to have some degree of difference between the size of the two testes.

Ultrasound examination should first be performed to characterise the lesion and confirm the presence of a mass.

If the mass is suspicious for cancer, then tumour markers should be measured.

Imaging with MRI may be required for patients with equivocal

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You review a 42-year-old woman admitted generally unwell with dehydration. She has a past history of borderline personality disorder, alcohol excess, and three previous upper gastrointestinal bleeds requiring endoscopic intervention.

She takes lithium MR 400mg once daily, omeprazole 40mg once daily and thiamine 100mg twice daily.

Renal function and osmolalities are as follows:

Na	155 mmol/L	
K	4.9 mmol/L	
Urea	13.0 mmol/L	
Creatinine	251 µmol/L	
Plasma osmolality	329 mmol/kg	(275-295 mmol/kg)
Urine osmolality	56 mmol/kg	(500-800 mmol/kg)

Since admission, she has been receiving 5% glucose infusions with little improvement in her renal function.

A water deprivation test with desmopressin is performed. Urine osmolalities pre- and postdesmopressin are shown below.

Pre-desmopressin	64mmol/kg
Post-desmopressin	72mmol/kg

Given the likely diagnosis, what is the most appropriate treatment?

A. Desmopressin
B. Fluid restriction
C. Hydrochlorothiazide
D. Indometacin
E. Tolvaptan

C. Hydrochlorothiazide

Diabetes insipidus is characterised by a **high plasma** osmolality and a **low urine** osmolality

The above diagnosis is diabetes insipidus (DI) causing dehydration with significant hypernatraemia, high plasma osmolality, and low urine osmolality. The next step is to distinguish whether this is cranial/central DI or nephrogenic DI. Measurement of urine output and osmolalities following desmopressin administration can differentiate between the two. In this case there is no response to desmopressin, indicating a nephrogenic cause for this woman's DI.

This woman is taking lithium regularly, a well-known cause of nephrogenic DI. Hydrochlorothiazide and indometacin are used in the management of nephrogenic DI, however, indometacin is contraindicated in patients with recurrent gastrointestinal bleeding, therefore hydrochlorothiazide is the correct answer.

Desmopressin is not the correct answer as nephrogenic DI is secondary to resistance to vasopressin rather than being caused by a deficiency.

Fluid restriction should not be used in diabetes insipidus and is therefore not correct. This would only worsen this woman's hypernatraemia. It is used instead in cases of syndrome of inappropriate anti-diuretic hormone (SIADH).

As mentioned, indometacin is contraindicated in patients with recurrent gastrointestinal bleeding. Therefore, in this case, hydrochlorothiazide is the correct answer.

Tolvaptan is an aquaporin 2 antagonist that can be used to increase the serum sodium concentration in SIADH. It is not used in DI.

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You are bleeped to see a 65-year-old female on the renal dialysis unit. The nurses state that shortly after starting dialysis she developed a headache and has since become very confused. She has a past medical history of myeloma which has resulted in a rapid decline in renal function. On examination you note that her Glasgow coma score is 13 (M5, V5, E3) and there is evidence of myoclonic jerks. You identify papilloedema on fundoscopy. Her drug history includes a fentanyl patch and regular paracetamol.

Her pre-dialysis bloods were as follows:

Hb	105 g/l	Na +	138 mmol/l
Platelets	210 * 10 /l 9	K +	6.8 mmol/l
WBC	8.8 * 10 /l 9	Urea	46 mmol/l
Neuts	4.2 * 10 /l 9	Creatinine	588 µmol/l

How will you manage this patient?

A. Sodium bicarbonate
B. Intramuscular adrenaline
C. Naloxone
D. Hypertonic saline
E. Reassurance

D. Hypertonic saline

Dialysis disequilibrium syndrome results in cerebral oedema which can present as focal neurological deficits, papilloedema and a decreased level of consciousness. It can be treated with mannitol or hypertonic saline

Dialysis disequilibrium syndrome is a rare but serious complication of haemodialysis. It is more likely to occur in patients with very high levels of urea, metabolic acidosis, extremes of age, and central nervous system diseases. High levels of urea result in fluid shifts in the brain. If urea is rapidly decreased, the result is a sudden drop in serum osmolarity, and a fluid shift from interstitial to intracellular compartments. The end result is cerebral oedema.

Cerebral oedema can present with headaches, focal neurological deficits, myoclonus, papilloedema and a decreased level of consciousness.

Dialysis disequilibrium syndrome can be treated with mannitol or hypertonic saline to increase plasma osmolarity and reduce cerebral oedema.

Although opioid toxicity can present with myoclonic jerks, the headache would not be typical, and the history is more suggestive of dialysis disequilibrium syndrome. Therefore, naloxone is not indicated.

Although the potassium is high, it is important to note that this is pre-dialysis bloods, and the potassium will be normalised following dialysis. Therefore, calcium gluconate is not indicated.

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A 24-year-old-man is reviewed in the Nephrology clinic. He has a recent diagnosis of IgA nephropathy, which was proven on biopsy after multiple episodes of haematuria. He has a past medical history of coeliac disease, but is otherwise fit and well. He is not taking any medications. On review, his blood pressure is 122/84 mmHg. His latest investigation results are shown below:

Bloods:

Na +	138 mmol/L	(135 - 145)
K +	3.9 mmol/L	(3.5 - 5.0)
Bicarbonate	26 mmol/L	(22 - 29)
Urea	5.1 mmol/L	(2.0 - 7.0)
Creatinine	88 µmol/L	(55 - 120)
24h urine protein collection	1300mg	(<150)

What is the most appropriate management option?

A. Chlorambucil + cyclophosphamide
B. Observation only
C. Prednisolone
D. Ramipril
E. Ramipril + prednisolone

D. Ramipril

IgA nephropathy management

- no proteinuria, normal GFR: observe
- proteinuria: ACE inhibitor
- signifcant fall in GFR/not responding to ACE inhibitor: corticosteroid

The correct answer is **ramipril**. This patient has a confirmed diagnosis of IgA nephropathy, with significant proteinuria and a normal renal function. Even though he is normotensive, the extent of his proteinuria (> 1000mg/day) warrants treatment with an ACE inhibitor. This acts to reduce glomerular filtration pressures by inhibiting angiotensin II Ì¶ mediated efferent arteriolar vasoconstriction, which in turn helps to reduce proteinuria.

Chlorambucil + cyclophosphamide is incorrect. This, together with methylprednisolone, makes up the Ponticelli regimen, which is a possible combination therapy for membranous nephropathy.

Observation only is incorrect. Whilst this would be an acceptable strategy for a patient with confirmed IgA nephropathy, a normal GFR and either no or trivial proteinuria, the extent of this patient's proteinuria warrants treatment with an ACE inhibitor.

Prednisolone is incorrect. This patient should be offered an ACE inhibitor for the reasons outlined above. Only if his proteinuria did not respond to ACE inhibition, or his GFR began to decline significantly, would it be appropriate to consider starting prednisolone.

Ramipril + prednisolone is incorrect as this patient has a normal GFR, and has not yet received a trial of ACE inhibitor monotherapy to try to reduce his proteinuria.

Question 37 of 183

A 67-year-old woman with a renal transplant is seen in clinic. She has noted increased swelling of her legs over the past 3 weeks but is relatively well otherwise.

On examination she is hypertensive at 162/110mmHg and has pitting oedema up to the mid shin bilaterally. Urine dip shows protein 3+ and blood 2+, with a serum creatinine of 142umol/l (previously 70umol/l).

A renal biopsy reveals a recurrence of her original disease.

Which disease has the highest rate of recurrence in renal transplant patients?

A. Membranoproliferative glomerulonephritis
B. IgA nephropathy
C. Polycystic kidney disease
D. Focal segmental glomerulosclerosis
E. Alport's syndrome

A. Membranoproliferative glomerulonephritis

Membranoproliferative glomerulonephritis (notably type II) reoccurs in 80-100% of cases. This is lower in type I.

- IgA reoccurs in up to around 60% of patients.
- FSGS reoccurs in up to around 50% of patients.
- Polycystic kidney disease and Alport's syndrome do not reoccur in renal transplantation.

Awareness of the conditions that reoccur in renal transplantation is vital:

- IgA nephropathy
- Membranoproliferative glomerulonephritis
- Focal segmental glomerulosclerosis
- Membranous glomerulonephritis

Question 38 of 183

A 65-year-old man attends renal transplant clinic as part of the preliminary workup to be considered as a living kidney donor. The patient's 26-year-old daughter has stage 5 chronic kidney disease secondary to congenital hydronephrosis, and will shortly require dialysis unless she receives a donated kidney. Upon arrival at the appointment, the patient states that he is very hopeful that he will be found to be a suitable kidney donor for his daughter.

The patient reports that he is in good general health, with his only active health problem being high blood pressure diagnosed 4 years previously, and for which he is prescribed medications. The patient denies any history of cardiovascular disease, malignancy, chronic infection, nephrolithiasis or kidney disease. There is no history of the patient suffering from any substance misuse or mental health problems.

The patient's regular medications were ramipril 5 mg once daily, amlodipine 10 mg once daily and bendroflumethiazide 2.5 mg daily. He stated that he had no known drug allergies and had never had an adverse reactive to anaesthetic medications. The patient is a lifelong non-smoker.

Examination revealed a moderately overweight but otherwise generally healthy middle-aged man. Cardiovascular, respiratory and abdominal examinations were unremarkable.

Initial investigations undertaken prior to the clinic, together with some details of the proposed recipient of the patient's kidney, are given below.

Body mass index	31.2 kg / m2
Haemoglobin	160 g / dL
Mean cell volume	89.8 fl
White cell count	7.3 x 10>3 / microlitre

Neutrophils	4.7 x 10>3 / microlitre (reference 1.8-7.8)
Platelets	387 x 10>3 / microlitre
Urea	6.4 mmol / L
Creatinine	103 micromol / L
eGFR	79 ml / min / 1.73 m2
Sodium	140 mmol / L
Potassium	3.9 mmol / L
Albumin	46 g / L (reference 35-50)
Bilirubin	24 micromol / L (reference < 26)
Urinalysis	no abnormality detected
Urine microscopy	no abnormality detected
Albumin-creatinine ratio	2.8 mg / mmol (reference < 3)
Ambulatory blood pressure monitoring average	158 / 92 mmHg

Potential donor blood type	Potential recipient blood type
Rhesus-D negative	Rhesus-D positive
ABO group A	ABO group AB

What is the absolute contraindication to this patient acting as a living kidney donor to his daughter?

A. Uncontrolled hypertension
B. Age > 60 years
C. Blood type incompatibility
D. Impaired renal function
E. Obesity

A. Uncontrolled hypertension

Detailed guidelines exist for living donor kidney transplantation (see below link). There are few absolute contraindications for an individual to donate a kidney. In this individual's case, the failure of three agents to control his blood pressure would render him ineligible to donate. This is due both to the increased peri-operative risk and also the potential for nephrectomy to exacerbate hypertension following donation. Other absolute contraindications include active malignancy, chronic infection, overt proteinuria, bilateral renal artery atherosclerosis and sickle cell disease.

Other factors mentioned are relative contraindications that need to be considered when discussing the risk of nephrectomy for the potential donor. The donor's pre-operative renal function must be of a level to give a minimum expected eGFR of 37.5 ml / min / 1.73 m2 at age 80 years (for this individual at age 60, the minimum eGFR would be considered to be 68 ml / min / 1.73 m2. There is no upper age limit for an individual to act as a donor, but pre-operative assessment must be particularly rigorous for older individuals. Moderate obesity (BMI 30-35 kg / m2) would also indicate the need for rigorous pre-operative assessment.

Historically, all transplants had to occur between ABO blood groups and HLA compatible donor and recipient pairs; however, desensitisation techniques are now available to allow immunologically incompatible kidney transplants in some cases.

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You are called to see a 45-year-old male on the renal ward because the nurses are concerned. The gentleman was admitted 2 weeks earlier with acute renal failure. Blood tests revealed a positive anti-neutrophil cytoplasmic antibody (ANCA) with a myeloperoxidase (MPO) level of 131. He underwent renal biopsy 4 days after admission and the provisional pathology report has shown acute crescentic glomerulonephritis (immuno-fluorescence awaited). Five days into admission he developed frank haemoptysis. He was treated with high flow oxygen and did not require transfusion.

You have been asked to see him because he collapsed on the way back from the bathroom. His blood pressure is 85/40 mmHg and he is tachycardic. He looks pale. He denies any further haemoptysis but complains of generalised abdominal pain. Blood results reveal:

White cell count	12.6 *109/l
Haemoglobin	67 g/l
Platelets	129 *109/l
Adjusted calcium	2.01 mmol/l
Prothrombin time	14 seconds
Fibrinogen	1.1 g/dL

An urgent CT angiogram is arranged which confirms intra-abdominal bleeding at the site of his kidney biopsy.

Which treatment is most likely to have increased his risk of delayed post-biopsy bleed?

A. Aspirin
B. Plasma exchange
C. Simvastatin
D. Intravenous methylprednisolone
E. Cyclophosphamide

B. Plasma exchange

Plasma exchange increases the risk of bleeding via depletion of fibrinogen levels and reduction in platelet count. Patients receiving plasma exchange should have their fibrinogen levels monitored daily and replaced with Fresh Frozen Plasma if deplete. Furthermore, it is recognised that plasma exchange specifically increases the risk of delayed bleeding following renal biopsy. This patient has a low fibrinogen level, low platelets (NB platelets are often high in acute vasculitis) and low calcium which can also occur with plasma exchange. It is likely therefore that plasma exchange contributed to his bleeding.

While aspirin may also increase bleeding risk, it is not routinely stopped prior to renal biopsy. Cyclophosphamide and methylprednisolone are 1st line drugs in this situation but are not known to have an associated bleeding risk. Plasma exchange is used in ANCA-positive vasculitis if pulmonary haemorrhage is present.

Question 40 of 183

A 92-year-old lady is admitted to the orthopaedic ward following a fall and lying three hours on the floor. She was brought in by ambulance and diagnosed with a right intertrochanteric fractured neck of femur. She was operated on six hours later and underwent fixation with a dynamic hip screw. She was given two units of blood during surgery.

The next day she reviewed by the orthogeriatric team. Over the last twelve hours, she has passed 337ml of urine. Her creatinine from the day before was 162umol/l. Her baseline creatinine from two months ago was 45umol/l. Her weight is 61kg.

What is the most appropriate description of her current renal function?

A. No renal impairment

B. Stage I acute kidney injury
C. Stage II acute kidney injury
D. Stage III acute kidney injury
E. Chronic renal disease

D. Stage III acute kidney injury

The correct answer is stage III acute kidney injury (AKI). This diagnosis is due to her rise in creatinine compared to her baseline results. Her creatinine has increased from 45umol/l to 162umol/l, which is a ratio of 3.6, indicating stage III AKI. She has passed urine at a rate of 0.46ml/kg/hour, which would indicate a stage II AKI, but since the staging by increased creatinine shows a more significant stage that is used to assess her renal function.

KDIGO grading of AKI:

Stage	Serum creatinine	Urine output
1	1.5-1.9 times baseline OR increase > 26.5 umol/l	<0.5ml/kg/hr for 6-12 hours
2	2.0-2.9 times baseline	<0.5ml/kg/hr for =>12 hours
3	3.0 times baseline OR increase => 353.6 umol/l OR initiation of renal replacement therapy	anuria =>12 hours

Acute kidney injury: NICE guidelines and staging

This guideline discuss what increases the risk of AKI:

- Emergency surgery, ie, risk of sepsis or hypovolaemia
- Intraperitoneal surgery
- CKD, ie if eGFR < 60
- Diabetes Heart failure Age >65 years Liver disease
- Use of nephrotoxic drugs NSAIDs aminoglycosides
- ACE inhibitors/angiotensin II receptor antagonists diuretics

Diagnostic criteria

It also defines the criteria for diagnosing AKI

Rise in creatinine of 26µmol/L or more in 48 hours OR >= 50% rise in creatinine over 7 days OR

Fall in urine output to < 0.5ml/kg/hour for more than 6 hours in adults (8 hours in children) OR

>= 25% fall in eGFR in children / young adults in 7 days.

Staging criteria

The Kidney Disease: Improving Global Outcomes (KDIGO) criteria are widely used to stage the AKI:

Stage 1	Increase in creatinine to 1.5-1.9 times baseline, or Increase in creatinine by ≥26.5 µmol/L, orReduction in urine output to <0.5 mL/kg/hour for ≥ 6 hours
Stage 2	Increase in creatinine to 2.0 to 2.9 times baseline, or Reduction in urine output to <0.5 mL/kg/hour for ≥12 hours
Stage 3	Increase in creatinine to ≥ 3.0 times baseline, orIncrease in creatinine to ≥353.6 µmol/L orReduction in urine output to <0.3 mL/kg/hour for ≥24 hours, or The initiation of kidney replacement therapy, or,In patients <18 years, decrease in eGFR to <35 mL/min/1.73 m2

Referral criteria

Refer to a nephrologist if any of the following apply:

- Renal tranplant
- ITU patient with unknown cause of AKI
- Vasculitis/ glomerulonephritis/ tubulointerstitial nephritis/ myeloma
- AKI with no known cause
- Inadequate response to treatment
- Complications of AKI
- Stage 3 AKI (see guideline for details)
- CKD stage 4 or 5
- Qualify for renal replacement hyperkalaemia / metabolic acidosis/ complications of uraemia/ fluid overload (pulmonary oedema)

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A 45-year-old man is referred to ambulatory care by his GP due to bilateral leg swelling and a subsequent urine dipstick showing proteinuria. He is otherwise fit with no past medical history and works as a school caretaker. He had a throat infection and joint pain two weeks ago which was treated with penicillin by his GP. This has been associated with some arthralgia and myalgia. He was beginning to feel better when the leg swelling started.

On examination he has bilateral pitting oedema to the knees. His JVP is not raised and his heart sounds are normal. His chest is clear and his throat does not appear infected currently. His abdomen is soft with no masses. He has no rash or joint swelling.

His urine dipstick shows 3+ protein. His blood tests are as follows:

Hb	118 g/l	Na +	131 mmol/l
Platelets	190 * 10 /l 9	K +	4.7 mmol/l
WBC	8 * 10 /l 9	Urea	12 mmol/l
Neuts	5.5 * 10 /l 9	Creatinine	131 µmol/l
Albumin	29g/l	CRP	25 mg/l

He is admitted and further testing reveals the following:

US kidneys - Normal size kidneys with no evidence of obstruction in the renal system.

Antistreptolysin O titre	104 IU (negative)
Complement C3 & C4	Mildly raised
Antiphospholipase A2 antibody	Positive
Double-stranded DNA	Normal
Anti-neutrophil cytoplasmic antibody	Negative

He has a renal biopsy which shows thickened basement membranes with subepithelial deposits on electron microscopy.

What is the most likely cause of his membranous glomerulonephritis?

A. Anti-phospholipid syndrome
B. Idiopathic
C. Lupus nephritis
D. Penicillin
E. Post-infectious

B. Idiopathic

This gentleman has a nephrotic syndrome with a membranous pattern on histology, of which there are many causes.

Although he gives a history of throat infection, his ASOT titre is normal, which points away from a post-infectious GN.

Penicillin typically causes an acute interstitial nephritis without nephrotic syndrome. Phospholipase A2 is highly specific for idiopathic membranous glomerulonephritis.

Although systemic lupus and anti-phospholipid syndrome are both known to cause membranous GN, this gentleman does not display typical symptoms associated with either condition. His arthralgia and myalgia are likely related to his recent throat infection. One would expect systemic lupus to be associated with low complement levels and anti-phospholipid syndrome to he associated with anti-cardiolipin antibodies.

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A 72-year-old man was referred to the medical admissions unit by his GP. He had a 1 month history of lethargy, weight loss and night sweats. He also complained of nasal crusting and shortness of breath with one episode of haemoptysis that morning. He had a past medical history of hypertension, for which he was taking amlodipine.

On examination his blood pressure was 156/94 mmHg and he appeared unwell. There was a petechial rash on both lower limbs.

A chest x-ray was performed which showed bilateral alveolar infiltrates. Blood tests are shown below:

Hb	96 g/L	Male: (135-180)Female: (115 - 160)
Platelets	487 * 10 /L 9	(150 - 400)
WBC	15.8 * 10 /L 9	(4.0 - 11.0)
Na +	138 mmol/L	(135 - 145)
K +	5.1 mmol/L	(3.5 - 5.0)
Urea	11.2 mmol/L	(2.0 - 7.0)
Creatinine	196 µmol/L	(55 - 120)
CRP	285 mg/L	(< 5)
c-ANCA	positive	
anti-GBM antibodies	negative	

A renal biopsy was performed which showed a necrotising crescentic glomerulonephritis.

Which of the following would be an indication to start plasma exchange in this case?

A. High ANCA titres
B. Crescents on renal biopsy
C. Level of renal dysfunction
D. ENT involvement
E. Pulmonary haemorrhage

E. Pulmonary haemorrhage

Indications for plasma exchange in ANCA-associated vasculitis: rapidly progressive renal failure or pulmonary haemorrhage

The indications for plasma exchange in ANCA-associated vasculitis are:

- Severe active renal disease (serum creatinine above 354 micromol/L or who require dialysis),
- Pulmonary haemorrhage,
- Concurrent anti-GBM autoantibody disease.

The history of haemoptysis and radiological findings in this case suggest active pulmonary haemorrhage, which can be life-threatening and would be an indication to start plasma exchange.

Although this patient has renal dysfunction, he is not dialysis-dependent and the creatinine is under 354.

Neither ENT involvement, crescents on renal biopsy or the degree of ANCA positivity are indications to start plasma exchange.

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A 24-year-old woman attends with a 4-week history of worsening peripheral oedema. She also has a history of widespread joint pain.

Blood results are as follows:

Hb	105 g/L	Male: (135-180)Female: (115 - 160)
Platelets	154 * 10 /L 9	(150 - 400)
WBC	15.8 * 10 /L 9	(4.0 - 11.0)
Na +	132 mmol/L	(135 - 145)
K +	4.9 mmol/L	(3.5 - 5.0)
Urea	18.2 mmol/L	(2.0 - 7.0)
Creatinine	426 µmol/L	(55 - 120)
CRP	3 mg/L	(< 5)
ANA	1:128	(<1:40)
Anti dsDNA	68 IU/mL	(<10)

A kidney biopsy is arranged:

Kidney biopsy	Diffuse glomerulonephritis involving 70% of glomeruli

What treatment is indicated?

A. Cyclophosphamide
B. Glucocorticoids
C. Glucocorticoids with azathioprine
D. Glucocorticoids with mycophenolate
E. Mycophenolate

D. Glucocorticoids with mycophenolate

Focal or diffuse lupus nephritis: glucocorticoids with either mycophenolate or cyclophosphamide is the initial treatment of choice

The clinical features and strongly positive ANA and anti-dsDNA confirm a diagnosis of systemic lupus erythematosus. The kidney biopsy results confirm class IV diffuse lupus nephritis.

Glucocorticoids with mycophenolate is correct. The combination of steroids with either mycophenolate or cyclophosphamide has the best evidence for preventing the development of end-stage renal disease.

Cyclophosphamide is incorrect. Cyclophosphamide can be used in the acute phase to induce remission, however, it should be combined with steroid therapy.

Glucocorticoids is incorrect. Glucocorticoids can be used in the acute phase to induce remission, however, they should be combined with either mycophenolate or cyclophosphamide.

Glucocorticoids with azathioprine is incorrect. Azathioprine can be used as maintenance treatment, however, is not used in the acute phase.

Mycophenolate is incorrect. Mycophenolate can be used in the acute phase to induce remission, however, it should be combined with steroid therapy.

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A 55-year-old male of African-Caribbean ethnicity presented to the emergency department with a 6-week history of progressive leg swelling, shortness of breath, and nausea. He had recently moved to the UK from South Africa about 3 months ago. He had a background of mild peripheral vascular disease for which he was on atorvastatin and clopidogrel.

On examination, he was comfortable at rest but had marked pitting oedema of both lower limbs and bilateral dullness to percussion at the lung bases. His blood pressure was 181/101mmHg.

Urine dipstick showed 2+ blood and 4+ protein. Blood test results were as follows:

Urea	21 mmol/L	(2.0 - 7.0)
Creatinine	256 µmol/L	(55 - 120)
Albumin	24 g/L	(35 - 50)

24-hour urine protein was measured to be 8g/day. Further blood tests were carried out:

ANA	negative
ANCA	negative
Complement (C3 and C4)	negative
Hepatitis serology	negative
HIV screening serology	negative
Serum protein electrophoresis	negative

A renal ultrasound was performed:

Left kidney 10.8cm, right kidney 11.3cm. There is an increased echogenicity bilaterally. No hydronephrosis is present. Peak flow velocities normal and equal bilaterally.

A renal biopsy was performed:

There are segmental areas of scarring affecting some of the glomeruli. No hypercellularity or crescents can be seen.

How should this patient be managed given the likely diagnosis?

A. Rifampicin, isoniazid, pyrazinamide and ethambutol
B. Prednisolone
C. Renal artery revascularisation
D. Supportive management
E. Haemodialysis

B. Prednisolone

Untreated FSGS has a < 10% chance of spontaneous remission

This patient presents with nephrotic syndrome secondary to focal segmental glomerulosclerosis (FSGS). He has nephrotic-range proteinuria (>3.5g/day) with oedema and hypoalbuminaemia. His biopsy is suggestive of FSGS - typically sclerosis or scarring is seen in only parts of the glomeruli (segmental) in only some of the glomeruli (focal). FSGS can be primary (as in this case) or secondary to a variety of causes including HIV and heroin use. The overall incidence is higher in African-Americans. The first-line treatment is with steroids. Patients who are resistant to steroids are treated with immunosuppressive therapy such as calcineurin inhibitors.

Untreated FSGS has a <10% chance of spontaneous remission so whilst supportive therapy should accompany active treatment, it is not appropriate in isolation.

Whilst this patient has recently travelled from a TB-endemic area, nothing in his history is suggestive of infection requiring anti-tuberculous medication.

Whilst this patient has risk factors for renal artery stenosis (known pre-existing peripheral artery disease), his presentation and biopsy findings are not in keeping with this as the cause of his deterioration in renal function. Furthermore, his kidneys are of normal and equal size on ultrasound (significant asymmetry, such as a size discrepancy of >1.5cm is more suggestive of renal artery stenosis). He also has normal and equal peak flow velocities on doppler. Therefore revascularisation is not the correct answer.

Nothing in the stem suggests that this patient is in urgent need of haemodialysis (e.g. treatmentresistant acidosis, hyperkalaemia, uraemic complications) so this is also incorrect.

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A 43-year-old man is admitted with worsening shortness of breath, confusion, and swelling in his lower legs.

He has a medical history of rosacea and uses topical brimonidine for this.

His observations are as follows: Heart rate 94 bpm, BP 192/92mmHg SpO2 93% on 2 litres nasal cannula, respiratory rate 24 breaths per minute.

Urine dip: blood +++ protein +++.

Chest x-ray: pulmonary oedema.

Hb	98 g/L	Male: (135-180)Female: (115 - 160)
Platelets	524 * 10^9/L	(150 - 400)
WBC	8.4 * 10^9/L	(4.0 - 11.0)
Lymphocytes	0.2 * 10^9/L	(1.0 - 4.0)
Na^+	145 mmol/L	(135 - 145)
K^+	6.4 mmol/L	(3.5 - 5.0)
Urea	32.4 mmol/L	(2.0 - 7.0)
Creatinine	544 µmol/L	(55 - 120)
CRP	34 mg/L	(< 5)
C3	0.3 g/L	(0.88 - 2.01)
C4	0.04 g/L	(0.14 - 0.45)
dsDNA	26 IU/mL	(<10)
pH	6.9	(7.35 - 7.45)

After fluid resuscitation, blood results remain static, including pH. A renal biopsy is planned.

Given the likely diagnosis, what is the most appropriate initial treatment of choice for the underlying condition?

A. IV methylprednisolone and cyclophosphamide
B. IV methylprednisolone and hydroxychloroquine

C. IV methylprednisolone and azathioprine
D. Mycophenolate monotherapy
E. Oral prednisolone and mycophenolate

A. IV methylprednisolone and cyclophosphamide

Focal or diffuse lupus nephritis: glucocorticoids with either mycophenolate or cyclophosphamide is the initial treatment of choice

Here we have a patient with nephrotic range proteinuria and haematuria. There are many differentials for this, but here the likely diagnosis is lupus. Blood tests reveal anaemia, raised platelets, and lymphopenia which indicates active inflammatory disease, deteriorating renal function indicating renal involvement, and a positive dsDNA with high titres. A renal biopsy would be required to confirm. In practice, autoantibody testing is unlikely to yield speedy results.

IV methylprednisolone and cyclophosphamide is the correct answer. The patient is in extremis with hyperkalaemia, uremic encephalopathy, and refractory acidosis. Workup for renal replacement therapy would be advisable and rapid treatment of lupus would be indicated. IV methylprednisolone and cyclophosphamide would be the treatments of choice.

IV methylprednisolone and azathioprine is incorrect. Dual treatment with mycophenolate or cyclophosphamide would be recommended given evidence of lupus nephritis. Azathioprine is sometimes used as a subsequent add-on treatment, but wouldn't be used in the acute phase.

Mycophenolate monotherapy is incorrect. This could be considered in mild lupus nephritis, but this patient requires an induction regime to rapidly treat the severe disease (hyperkalaemia, uremic encephalopathy, and refractory acidosis).

Oral prednisolone and mycophenolate is incorrect. This could be considered in mild lupus nephritis, but this patient requires an induction regime to rapidly treat the severe disease (hyperkalaemia, uremic encephalopathy, and refractory acidosis).

IV methylprednisolone and hydroxychloroquine is incorrect. IV methylprednisolone would be reasonable, but it is advised to avoid hydroxychloroquine in severe renal impairment. Hydroxychloroquine is commonly used in the management of lupus, but mycophenolate is preferred in the management of renal complications for this reason.

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A 43-year-old man presents to the emergency department with abdominal pain and fever. He has a past medical history of end-stage renal failure secondary to type 1 diabetes. His method of renal replacement therapy is peritoneal dialysis. His medications include insulin and atorvastatin. He does not smoke or drink alcohol.

Observations:

Heart rate 101 beats per minute Blood pressure 120/73 mmHg Respiratory rate 18/minute Oxygen saturations 96% on room air Temperature 38.4°C

On examination, there is mild abdominal tenderness. Bowel sounds are present. The dialysate fluid appears cloudy.

Given the likely diagnosis, what is the appropriate antibiotic choice?

A. IV benzylpenicillin
B. Intraperitoneal ciprofloxacin and intravenous amikacin
C. Intraperitoneal flucloxacillin and ceftazidime
D. Intraperitoneal vancomycin + ceftazidime
E. Intravenous ciprofloxacin

D. Intraperitoneal vancomycin + ceftazidime

Peritoneal dialysis peritonitis: treat with **intraperitoneal** vancomycin + ceftazidime

Intraperitoneal vancomycin + ceftazidime is the correct answer. The patient has peritoneal dialysis peritonitis as evidenced by fever, abdominal pain and a cloudy dialysate fluid. The appropriate antibiotic choice is intraperitoneal vancomycin + ceftazidime. The antibiotic choice should give broad coverage to both gram-positive and gram-negative organisms. Intraperitoneal administration of antibiotics has been shown to be safer and more effective when compared with IV in patients with peritoneal dialysis peritonitis.

IV benzylpenicillin is incorrect. This would only provide coverage against gram-positive organisms, which would be insufficient in this scenario.

Intraperitoneal ciprofloxacin and intravenous amikacin is incorrect. This antibiotic choice would provide broad gram-negative coverage. There is some suggestion that amikacin may have some action against gram-positive organisms including *Staphylococcus aureus* and *Methicillinresistant Staphylococcus aureus* but vancomycin would be a preferable choice and the intraperitoneal route is superior, in addition.

Intravenous ciprofloxacin is incorrect. This would provide insufficient cover for gram-positive organisms.

Intraperitoneal flucloxacillin and ceftazidime . This would provide good gram-positive and gram-negative coverage in this scenario. However, intraperitoneal vancomycin would be preferable to flucloxacillin due to its activity against MRSA.

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A 63-year-old man is referred to the medical assessment unit with new leg swelling. He has a past medical history of longstanding ankylosing spondylitis for which he declined anti-inflammatory treatment.

On examination, there is peripheral oedema extending to the thighs. The sensation is also reduced in a glove stocking distribution to his knees.

Blood tests:

Hb	101 g/L	Male: (135-180)Female: (115 - 160)
Platelets	555 * 10 /L 9	(150 - 400)
WBC	4.2 * 10 /L 9	(4.0 - 11.0)

Na +	138 mmol/L	(135 - 145)
K +	4.2 mmol/L	(3.5 - 5.0)
Urea	14.2 mmol/L	(2.0 - 7.0)
Creatinine	200 µmol/L	(55 - 120)
Albumin	20 g/L	(34-54)
CRP	66 mg/L	(< 5)

Urinalysis:

Blood	negative
Protein	+++

From the listed options, what is the most appropriate initial investigation to make the diagnosis?

A. Abdominal fat pad biopsy
B. Anti-phospholipase A2 receptor antibody
C. ESR
D. Renal biopsy
E. Transthoracic echocardiogram

A. Abdominal fat pad biopsy

Amyloidosis may be confirmed by a biopsy of skin, rectal mucosa, or abdominal fat

Abdominal fat aspiration is correct. The patient presents with leg swelling and evidence of nephrotic syndrome (proteinuria, hypoalbuminemia) and deranged renal function. In addition, he has evidence of sensory peripheral neuropathy. Together with his history of longstanding untreated ankylosing spondylitis, this suggests a diagnosis of secondary (AA) amyloidosis. This condition is more common in those patients with chronic levels of systemic inflammation, which causes the production of a protein called SAA (serum amyloid A protein) in large amounts. If the inflammation persists, a small amount of SAA protein (called AA protein) will deposit in affected tissues as AA amyloid. The diagnosis is confirmed by a biopsy. While it is possible to biopsy the affected organ (i.e. renal or nerve), a biopsy of a surrogate site (i.e. abdominal fat) is preferred in the first instance due to its relative ease, lower risk of complications and great physical comfort.

Anti-phospholipase A2 receptor antibody is incorrect. This antibody is associated with idiopathic membranous nephropathy rather than amyloidosis. This is a cause of nephrotic syndrome. However, it is much less likely to be associated with peripheral neuropathy and chronic systemic inflammation and is, therefore, less likely.

ESR is incorrect. This is a non-specific marker of inflammation and may well be raised in this circumstance. However, it will not confirm or refute the diagnosis.

Renal biopsy is incorrect. Biopsy of a surrogate site is preferred in the initial investigation of amyloidosis due to the increased bleeding risk associated with this condition and greater technical expertise required for biopsy.

Transthoracic echocardiogram is incorrect. This may show indirect evidence of cardiac amyloidosis but is less specific for the diagnosis when compared with an abdominal fat pad biopsy.

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A 43-year-old man presents to the emergency department with bilateral leg swelling. He has a past medical history of hepatitis B, hepatitis C, type 2 diabetes and previous syphilis. He is a current user of intravenous heroin.

On examination, there is peripheral oedema extending to his upper thighs. Chest auscultation is normal and the jugular venous pulse is not raised. There is no clinical evidence of decompensated liver disease.

Bloods:

Hb	138 g/L	Male: (135-180)Female: (115 - 160)
Platelets	189 * 10 /L 9	(150 - 400)
WBC	4.2 * 10 /L 9	(4.0 - 11.0)
Na +	137 mmol/L	(135 - 145)
K +	4.2 mmol/L	(3.5 - 5.0)
Urea	6.2 mmol/L	(2.0 - 7.0)
Creatinine	99 µmol/L	(55 - 120)
CRP	4 mg/L	(< 5)
Bilirubin	12 µmol/L	(3 - 17)
ALP	88 u/L	(30 - 100)
ALT	32 u/L	(3 - 40)
Î³GT	44 u/L	(8 - 60)
Albumin	20 g/L	(35 - 50)
Urine protein:creatinine ratio	425 mg/mmol	(<50)

A renal biopsy is undertaken, which demonstrates focal and segmental sclerosis and hyalinosis on light microscopy.

What is the underlying cause for the likely diagnosis?

A. Hepatitis B
B. Hepatitis C
C. Heroin use
D. Syphilis
E. Type 2 diabetes

C. Heroin use

Heroin use is a risk factor for focal segmental glomerulosclerosis

Heroin use is right answer. The patient has nephrotic syndrome (oedema, hypoalbuminaemia and proteinuria) and a biopsy consistent with focal segmental glomerulosclerosis (FSGS). Heroin use is a risk factor for the development of FSGS.

Hepatitis B is incorrect. This can cause nephrotic syndrome. However, it is typically associated with membranous nephropathy. A renal biopsy, in this case, would demonstrate diffuse capillary and glomerular basement thickening rather than focal and segmental sclerosis and a 'spike and dome' appearance on electron microscopy with foot process effacement.

Hepatitis C is incorrect. This can cause nephrotic syndrome but is more likely to be associated with membranoproliferative glomerulonephritis or membranous nephropathy. Membranoproliferative glomerulonephritis causes subendothelial and mesangial electron dense deposits on electron microscopy.

Syphilis is incorrect. Again, this can cause nephrotic syndrome but like HBV is more likely to cause membranous nephropathy.

Type 2 diabetes is incorrect. This is more likely to cause diffuse thickening of the basement membrane or diffuse mesangial expansion in the early stages. Nodular glomerulosclerosis

(Kimmelstiel-Wilson nodules) or diffuse glomerulosclerosis may be seen in later stages.

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A 53-year-old woman presents to the emergency department with pain radiating from her right loin to her right groin. She has a past medical history of recurrent urinary tract infections and is on prophylactic nitrofurantoin. There are no similar previous episodes and there is no history of renal tubular acidosis.

Observations:

Heart rate 101 beats per minute Blood pressure 125/72 mmHg Respiratory rate 18/minute Oxygen saturations 96% on room air Temperature 37.1C

On examination, the patient is restless and in pain. There is no peritonism. Urinalysis:

Blood	+++	(negative)
Protein	negative	(negative)
Leucocytes	+	(negative)
Glucose	negative	(negative)
pH	8.4	(4-8)

Given the probable diagnosis, what is the likely composition of causative pathology?

A. Calcium oxalate
B. Calcium phosphate
C. Cystine
D. Struvite
E. Uric acid

D. Struvite

Struvite stones are formed in the presence of increased urinary ammonia and alkaline urine (>7.2).

Struvite is the correct answer. The patient presents with symptoms suggestive of renal colic and a urine dipstick demonstrating blood. She also has evidence of alkaline urine with a pH of 8.4. In the setting of chronic urinary tract infections, this suggests her stone is likely to be struvite in origin. Struvite stones are formed in the presence of increased urinary ammonia and alkaline urine (>7.2). These occur as a result of urease producing bacteria (and are thus associated with chronic infections).

Calcium oxalate is incorrect. This is the most common cause of a renal stone and would be the likely answer if it were not for the presence of alkaline urine and chronic infections. Hypercalciuria is a risk factor.

Calcium phosphate is incorrect. These are typically associated with renal tubular acidosis, which is absent in this case.

Cystine is incorrect. This type of stone is caused by an inherited disorder and therefore we would not expect the first episode at 53.

Uric acid is incorrect. These stones typically precipitate when urinary pH is low rather than high. They are associated with gout, malignancy and inborn errors of metabolism, which are absent here.

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A 44-year-old woman is brought into the emergency department with a 2-day history of feeling generally unwell and nauseated. She is feeling increasingly drowsy and is unable to give a coherent history.

On examination, she is tachypnoeic with a respiratory rate of 28 breaths/min and oxygen saturation of 98% on air. Her heart rate is 100 beats/min with a blood pressure of 118/70 mmHg. She is afebrile.

Arterial blood gas:

pH	7.25	(7.35 - 7.45)
pO2	11.3 kPa	(11 - 14.4)
pCO2	4.7 kPa	(4.6 - 6.4)
Sodium	145 mmol/L	(135 - 145)
Potassium	5.0 mmol/L	(3.5 - 5.5)
Chloride	95 mmol/L	(95 - 108)
Bicarbonate	15 mmol/L	(22 - 29)
Glucose	6.9 mmol/L	(4 - 7)
Lactate	1.9 mmol/L	(0.5 -2.2)
Base excess	-8	(-2 - 2)

What is the most likely cause of the above presentation?

A. Addison's disease
B. Chronic paracetamol use
C. Diarrhoea
D. Type 1 renal tubular acidosis
E. Type 2 renal tubular acidosis

B. Chronic paracetamol use

Chronic paracetamol use can lead to raised anion gap metabolic acidosis caused by 5- oxoproline

Causes of raised anion gap metabolic acidosis, think MUDPILES:

- **M**ethanol
- **U**raemia (renal failure)
- **D**iabetic ketoacidosis
- **P**aracetamol use (chronic)
- **I**soniazid
- **L**actate
- **E**thanol or propylene glycol
- **S**alicylates

This patient has a raised anion gap metabolic acidosis. This can be calculated by:

[Na (145) + K (5)] - [Cl (95) + HCO3 (15)] = 40 mmol/L. A normal anion gap is between 8-16 mmol/L.

Of the options listed in the question, only **chronic paracetamol use** is capable of producing a raised anion gap metabolic acidosis, making it the correct answer.

Addison's disease, **diarrhoea**, **type 1 renal tubular acidosis** and **type 2 renal tubular acidosis** all cause a normal anion gap metabolic acidosis and are, therefore, incorrect.

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A 25-year-old man presents with vomiting, confusion, general malaise and abdominal pain. His initial bloods show the following:

Na^+	134 mmol/L	(135 - 145)
K^+	5.6 mmol/L	(3.5 - 5.0)
Bicarbonate	18 mmol/L	(22 - 29)
Urea	20.3 mmol/L	(2.0 - 7.0)
Creatinine	342 µmol/L	(55 - 120)

Hb	85 g/L	Male: (135-180)
Platelets	93 * 10^9/L	(150 - 400)
WBC	12 * 10^9/L	(4.0 - 11.0)

ESR	68 mm/hr	Men: < (age / 2)
Prothrombin time (PT)	13 secs	(10-14 secs)
Activated partial thromboplastin time (APTT)	27 secs	(25-35 secs)
Fibrinogen	1.0 g/L	(2 - 4)
Reticulocytes	4.3 %	(0.5 - 1.5)
LDH	943 U/L	(100-190 U/L)

Assays for Shiga toxin were negative and an ADAMS-13 assay was normal. Following discussion with haematology, it was thought that this may represent an atypical haemolytic-uraemic syndrome. And the decision was made for the patient to undergo plasma exchange.

Which of the following additional treatments has been shown to have efficacy in treating atypical haemolytic-uraemic syndrome?

A. Daratumumab
B. Rituximab
C. Cyclophosphamide
D. Omalizumab
E. Eculizumab

E. Eculizumab

Eculizumab (a C5 inhibitor monoclonal antibody) has evidence of greater efficiency than plasma exchange alone in the treatment of adult atypical HUS

Eculizumab is a monoclonal antibody against the C5 component of complement and thereby blocks the complement cascade. Atypical haemolytic syndrome is a subtype of thrombotic microangiopathies, which also includes Shiga toxin-producing *E.coli* haemolytic-uraemic syndrome and thrombotic thrombocytopenic purpura, which results from genetic defects in the complement cascade resulting in uncontrolled activation. Eculizumab has been shown to be efficacious in the treatment of atypical haemolytic-uraemic syndrome and is currently recommended by NICE.

Omalizumab is a monoclonal antibody against Ig-E and used in certain types of asthma.

Daratumumab is a monoclonal antibody targeting CD38 and has use in the treatment of multiple myeloma.

Rituximab is an anti-CD20 and used in certain autoimmune disorders such as rheumatoid arthritis and malignancies such as certain types of lymphoma.

Cyclophosphamide is an immunosuppressant used in the treatment of a variety of rheumatological processes such as types of vasculitis and in certain chemotherapy regimens.

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A 53-year-old man is admitted to the hospital with dysuria and frequency. He describes two episodes of visible haematuria long with occasional loin pain with radiation into his flank.

Past medical history includes hypertension and recurrent urinary tract infections. He mentions he has previously had multiple renal stones but has never been under the urologists.

Regular medications include candesartan in the morning and cefalexin at night. Relevant investigations are below:

Abdominal xray	Mildly radio-opaque density at the level of the right renal pelvis. Advise CT KUB for further evaluation.
CT KUB	Awaited
Urinary pH	7.32
Urinary ammonia	High

What type of renal stone is accountable for the above findings?

A. Calcium oxalate
B. Calcium phosphate
C. Cystine
D. Struvite
E. Uric acid

D. Struvite

Struvite stones are formed in the presence of increased urinary ammonia and alkaline urine (>7.2).

The history of recurrent urinary infections, high urinary ammonia, and an alkaline urinary pH of >7.2 is in keeping with **struvite** renal stones. These stones contain magnesium, phosphate, and ammonia, are some of the only renal stones to form in alkaline conditions, and appear slightly radio-opaque on x-ray.

Calcium oxalate is incorrect as stones form in variable urine acidities but the pH is generally around 6. As with struvite, calcium oxalate stones appear radio-opaque.

Calcium phosphate stones occur in renal tubular acidosis, types 1 and 3. These stones are radioopaque and also form in alkaline urine. Unlike struvite stones, they are not associated with recurrent urinary infections.

Cystine stones form under normal urinary acidity with a pH of 6.5 and they appear as radiodense on x-ray due to containing sulphur. Cystine stones are associated with inherited disorders of cystine transport.

Uric acid stones form in acidic urine. They are radiolucent and are more common in children and diseases associated with tissue breakdown.

Question 53 of 183

A 25-year-old man attends renal clinic following a referral by his GP due to concerns about recent blood tests suggesting impaired renal function. The patient was a professional rugby player, and as part of pre-season training had undergone a range of screening blood tests. These tests had been entirely unremarkable, except for a reduced estimated glomerular filtration rate of 58 ml / min / 1.73 m2. The rugby club had then informed the player of the need to attend his GP with the result, who referred the patient for urgent renal review.

The patient reports feeling very well in himself, but very concerned about the possibility that he had developed renal failure. The patient had never previously been aware of any problems involving his kidneys and his only previous medical concern was a successful emergency operation for testicular torsion 10 years previously. He denied any history of diabetes, hypertension, connective tissue disease, renal calculi or prostatic symptoms. There was no family history of kidney disease. The patient took no regular medications, and occasionally took over-the-counter ibuprofen if he suffered a sports injury, but not at other times. Due to his career, the patient was extremely physically active and regularly attended the gym for strength training.

On examination, the patient appeared to be generally very healthy, with a highly developed athletic physique. Cardiovascular, respiratory and abdominal examination was entirely unremarkable. Please see below for investigations arranged around 3 months after the initial blood tests, as part of the patient's referral to the renal clinic.

Haemoglobin	169 g / dL
Mean cell volume	87 fl
White cell count	9.5 x 10>3 / microlitre
Platelets	243 x 10>3 / microlitre
Urea	7.0 mmol / L
Creatinine	126 micromol / L
eGFR	56 ml / min / 1.73 m2
Sodium	142 mmol / L
Potassium	4.3 mmol / L
Urinalysis	no abnormality detected
Urine microscopy	no abnormality detected
Albumin:creatinine ratio	1.9 mg / mmol (reference < 3)

What is the appropriate next investigation?

A. Ultrasound renal tract
B. Renal biopsy
C. Genetic testing for PKD-1, PKD-2 and PKD-3 genes
D. Serum cystatin-C
E. Anti-neutrophil cytoplasmic antibodies

D. Serum cystatin-C

In individuals with extremes of muscle mass, estimated GFR calculations based on creatinine levels need to be interpreted with caution. Estimated GFR calculations based on creatinine will underestimate true glomerular filtration rate in Individuals with a high muscle mass (such as athletes or body-builders) and overestimate true glomerular filtration rate in individuals with a low muscle mass (such as an individual with a muscle-wasting disorder).

An alternative marker of kidney function used to estimate glomerular filtration rate is cystatin C, a protein produced by all nucleated human cells. Cystatin C levels are less dependent on age, sex, race and muscle mass than creatinine. Therefore, calculating estimated glomerular filtration rate using cystatin C (eGFRcystatinC) gives a more accurate estimate of renal function than calculations using creatinine (eGFRcreatinine).

NICE guidance on chronic kidney disease recommends the measurement of cystatin C in individuals with an eGFRcreatinine of 45-59 ml / min / 1.73 m2 sustained for at least 90 days and no albuminuria (albumin:creatinine ratio < 3 mg / mmol) or another marker of kidney disease. NICE guidance states that chronic kidney disease should not be diagnosed in individuals with eGFRcreatinine of 45-59 ml / min / 1.73 m2 and no other marker of kidney disease if eGFRcystatinC is greater than 60 ml / min / 1.73 m2.

NICE guidance defines markers of kidney disease to be albuminuria, urine sediment abnormalities, electrolyte abnormalities, histological abnormalities, structural abnormalities or a history of kidney transplantation. The patient in this question has none of these features and so cystatin C is the most appropriate next investigation, with no indications for the other possible answers.

Renal ultrasound is recommended by NICE guidance for patients with chronic kidney disease who have accelerated progression of chronic kidney disease, have persistent haematuria, have symptoms of urinary tract obstruction, have a family history of polycystic kidney disease (if older than 20 years), have a estimated GFR of less than 30 ml / min / 1.73 m2 or are considered by a nephrologist to require a renal biopsy.

Question 54 of 183

A 62-year-old man attends hospital for an elective laparoscopic inguinal hernia repair. The surgical team note that the patient has impairment of his renal function on pre-operative blood tests and so refer the patient to the on-call general medical registrar. Following the referral, the surgical team were asked to organise some basic further investigations pending a medical review. The patient then underwent the planned surgical procedure, which was apparently uncomplicated and spent the following night in hospital as originally planned.

The day after the patient's operation, the patient was reviewed on the ward by the general medical team. He reported himself to be recovering well from the operation, although he was quite worried about the abnormality uncovered with his kidneys as he had been told he had 'chronic renal failure'. The patient had never previously been aware of any problems involving his kidneys and reported being in good general health, with his only previous medical concerns being wellcontrolled asthma and the hernia that lead to him being admitted to hospital. He denied any history of diabetes, hypertension, connective tissue disease, renal calculi or prostatic symptoms. There was no family history of kidney disease. The patient took a regular steroid inhaler for his asthma and denied significant use of non-steroidal anti-inflammatory drugs. The patient worked as a supermarket manager and reported smoking a cigar a couple of times per week.

On examination, the patient appeared to be in generally good health. Blood pressure was 128 / 85 mmHg. The patient was euvolaemic, with no evidence of pulmonary oedema or peripheral oedema. Examination of the patient's abdomen was unremarkable, except for the surgical incisions associated with his recent operation. The patient had been catheterised peri-operatively and was documented to be passing appropriate volumes of urine (i.e. > 0.5 ml / kg / hour).

Please see below for investigations arranged after the patient was admitted to hospital. The patient's GP surgery had been contacted but did not have records of any relevant recent blood tests.

Haemoglobin	136 g / dL
Mean cell volume	87.8 fl
White cell count	5.7 x 10>3 / microlitre
Platelets	238 x 10>3 / microlitre
Urea	7.9 mmol / L

Creatinine	131 micromol / L
eGFR	57 ml / min / 1.73 m2
Sodium	137 mmol / L
Potassium	4.1 mmol / L
Thyroid stimulating hormone	4.2 microU / mL (reference 0.4-5.0)
Urinalysis	no abnormality detected
Urine microscopy	no abnormality detected
Albumin:creatinine ratio	2.8 mg / mmol (reference < 3)

What is the appropriate classification of the patient's abnormal renal function?

A. Chronic kidney disease G2A3
B. Chronic kidney disease not confirmed
C. Chronic kidney disease G2A1
D. Chronic kidney disease G3aA1
E. Chronic kidney disease G3bA2

B. Chronic kidney disease not confirmed

The classification of chronic kidney disease (CKD) was updated by revised NICE guidance in 2014. CKD is now categorised by estimated GFR (G1-G5 including sub-categories G3a and G3b) and by albumin:creatinine ratio (A1-A3). CKD is defined as abnormalities of kidney function or structure present for more than 3 months, with implications for health. This definition, therefore, includes all individuals with markers of kidney damage and those with a glomerular filtration rate of less than 60 ml / min / 1.73 m2 on at least 2 occasions separated by a period of at least 90 days. Markers of kidney damage are defined by albuminuria, urine sediment abnormalities, electrolyte abnormalities, histological abnormalities, structural abnormalities and a history of kidney transplantation.

In the case of this patient, no markers of kidney disease are evident and only one abnormal eGFR result is available. Therefore, the diagnosis of CKD is not appropriate at this stage. NICE guidance would recommend the patient underwent a further renal function test in 2 weeks (to exclude causes of acute deterioration in eGFR), and then a further renal function test at 90 days after the original test. Should the patient's eGFR remain in the same range as the initial test (45-59 ml / min / 1.73 m2) and if there continues to be no evidence of markers of kidney damage, then NICE guidance would recommend using measuring cystatin-C levels to confirm or rule out CKD.

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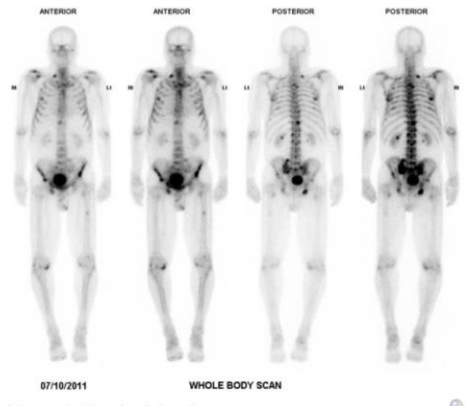
A 75-year-old man is investigated for widespread bone pain which has been getting progressively worse over the past six months. The pain is particularly bad around his back and ribs and is now no longer controlled with standard analgesia.

An isotope bone scan is requested:



Which next test is most likely to reveal the underlying diagnosis?

A. Colonoscopy
B. Adjusted serum calcium
C. Prostate specific antigen
D. Serum alkaline phosphatase
E. Electrophoresis

C. Prostate specific antigen

This technetium-99m bone scan is consistent with metastatic prostate cancer. The scan demonstrates multiple, irregular, randomly distributed foci of high grade activity involving the spine, ribs, sternum, pelvic and femoral bones. The findings are in keeping with multiple osteoblastic metastasis.

The 'spotty' nature and distribution of the lesions make metastatic cancer more likely than Paget's disease of the bone (represented by the serum alkaline phosphatase option).

Prostate cancer is the most common cause of bone metastases. Colon cancer does not as commonly metastasise to the bones.

Question 56 of 183

A 74-year-old male is reviewed in the renal outpatient clinic. Over the past 4 years, his renal function has slowly deteriorated, with his creatinine rising to 482 µmol/l and estimated glomerular filtration rate at 7 ml/min. He has noted the amount of urine he passes to be deteriorating over the past 3 months. His past medical history includes hypertension, type 2 diabetes mellitus, duodenal ulcer 35 years ago requiring laparotomy and an incisional hernia that has not been repaired. You discuss renal replacement therapies with him. He is very keen for the least lifestylerestricting option if at all possible but understands that 'his health comes first'. A recent ultrasound of his renal tract demonstrates bilateral atrophic kidneys with extensive bilateral iliac vessel calcification.

What is the most appropriate next treatment?

A. Annual follow up
B. Haemodialysis
C. Peritoneal dialysis
D. Renal transplant
E. Palliation

B. Haemodialysis

This question examines the different options of renal replacement therapy. This patient has deteriorating renal function and requires renal replacement therapy. The decision is which one, palliation and annual follow-up are definitely inappropriate. It would be inappropriate to rule out the patient for renal transplant on age alone: it is a common misconception that there is an age threshold for transplant. A recent review of transplanted patients revealed at least 13% of recipients to be over 70 years old. A contraindication may be limited life expectancy such as malignancy or degenerative diseases, which does not appear to be the case in this patient. However, the patient has a surgical contraindication to transplant: his significant bilateral renal artery calcification would prevent successful transplantation.

Although peritoneal dialysis is the renal replacement therapy that would be in keeping with his wish of 'least lifestyle-restricting' option, his previous history of laparotomy and unrepaired abdominal hernia are contraindications. Haemodialysis, via AV fistula or tunnelled catheter line, would be the most appropriate option.

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A 44-year-old Greek-Cypriot man is reviewed in the renal outpatient clinic. His renal function has been progressively deteriorating over the past 5 years secondary to poorly controlled type 2 diabetes mellitus. His eGFR is now 7 ml/min but continues to pass urine. Recent ultrasound scans have shown that he would anatomically be suitable for a renal transplant. However, you explain that in the context of his ethnicity, there is an uncertain wait for suitability of organs, should the patient require to proceed down this path. His 15-year-old son, who is present with the patients two daughters and wife, offers his father a donor kidney due to the high likelihood of compatibility, which the patient would like to proceed with.

The father's blood tests are shown below::

Hb	89 g/l
Platelets	178 * 10 /l9
WBC	6.5 * 10 /l9
Na +	144 mmol/l
K +	5.3 mmol/l
Urea	18.2 mmol/l
Creatinine	280 µmol/l
pH	7.32

What is the most appropriate next step?

A. Monitor renal function, discuss renal replacement therapy in 6 months time
B. Start haemodialysis today, discuss renal replacement therapy in 6 months time
C. Start preparation for haemodialysis and list for renal transplant on national transplant list
D. Start preparation for haemodialysis and list for renal transplant with the patients son'skidney
E. Palliation

C. Start preparation for haemodialysis and list for renal transplant on national transplant list

The underlying cause of the patient's renal dysfunction is poorly controlled diabetes and unlikely to improve spontaneously. He has no acute indications for acutely starting renal replacement therapy. The timing of renal transplantation is debated due to the variable rates of renal function decline between patients but it is accepted that once the underlying pathophysiology is deemed irreversible and eGFR has deteriorated to less than 30, transplantation should be discussed. Palliation would be wholly inappropriate.

The key issue is with regards to the ethics of using the donor kidney of a minor, despite it likely to produce the best genetic match. Renal transplantation is a rigorous process and donors under 18 years of age are almost never considered for renal transplantation, on the basis of the validity of consent and autonomy in such situations . The patient should thus be placed first on the national register with preparations for renal replacement therapy.

Question 58 of 183

A 72-year-old Indian male presents with left loin pain and occasional frank haematuria. He reports the symptoms started 2 months ago during a visit to Mumbai but have been getting progressively worse over the past 4 days. He also reports gradual weight loss and reduced appetite as well as a non-productive cough .

His past medical history includes type 2 diabetes mellitus, hypertension and hypercholesterolaemia. On examination, his temperature is 37.8 degrees and he is haemodynamically stable.

Abdominal examination reveals a significant swelling in his left scrotum, which does not empty on lying flat. The abdomen is otherwise soft and bowel sounds are present are normal. Lastly, bilateral lower limb swelling is noted to the top of both thighs. A urine dip reveals pH 6.5 4+ blood 1+ protein 1+ leucocyte 1+ nitrite. An initial chest radiograph reveals multiple round opacities in both lung fields. Blood tests are awaited.

Which investigation is most likely to reveal the underlying diagnosis?

A. CT abdomen with contrast
B. Three sets of blood cultures
C. Urinary protein-creatinine ratio
D. Ultrasound testes
E. Urine acid fast bacilli culture

A. CT abdomen with contrast

There are a number of worrying features with this patient: flank pain and haematuria, associated with round opacities in the lungs, a left varicocoele and bilateral limb swelling point towards a malignancy and unlikely to be explained by tuberculosis alone. Renal cell carcinoma (RCC) should be strongly suspected.

Be aware that while the classic triad of haematuria, abdominal mass and flank pain is frequently described in textbooks, this combination of features only present in 9% of RCC patients.

A left-sided varicocele should raise suspicion of compression on the left testicular vein. Bilateral lower limb oedema in the absence of overt right heart failure should alert you to obstruction of the inferior vena cava, a classic site of malignant tracking from renal cell carcinoma towards metastases in the lungs.

While miliary tuberculosis producing bilateral lung features with cystitis and sterile TB pyuria is possible, IVC obstruction features and bilateral rounded lung opacities would be atypical.

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A 48-year-old man presented to the emergency department with bilateral lower limb swelling which had developed over the past week. He also described having frothy urine.

On examination, he had pitting oedema up to his sacrum. His pulse was 65 beats per minute and his blood pressure was 170/96 mmHg. Urinalysis showed protein 4+. Blood tests are show below:

Hb	118 g/L	Male: (135-180)Female: (115 - 160)
Platelets	482 * 10 /L 9	(150 - 400)
WBC	8.5 * 10 /L 9	(4.0 - 11.0)
Na +	134 mmol/L	(135 - 145)
K +	3.6 mmol/L	(3.5 - 5.0)
Bicarbonate	23 mmol/L	(22 - 29)
Urea	4.5 mmol/L	(2.0 - 7.0)
Creatinine	89 µmol/L	(55 - 120)
Bilirubin	6 µmol/L	(3 - 17)
ALP	96 u/L	(30 - 100)
ALT	34 u/L	(3 - 40)
γGT	42 u/L	(8 - 60)
Albumin	18 g/L	(35 - 50)

A renal biopsy was performed, which showed diffuse thickening of the glomerular basement membrane. Silver stain showed 'spikes' on the surface of the capillary loops. Anti-PLA2R antibodies were negative.

What is the next best step in investigations?

A. Cryoglobulins
B. Bone marrow biopsy
C. Screen for malignancy
D. HIV testing
E. Renal tract ultrasound

C. Screen for malignancy

Membranous nephropathy is frequently associated with malignancy

This patient presents with nephrotic syndrome and the biopsy findings are consistent with membranous nephropathy.

Primary membranous nephropathy is associated with anti-PLA2R antibodies. The negative result in this patients suggests a secondary cause is more likely.

Secondary membranous nephropathy is frequently associated with malignancy, therefore guidelines suggest that age-appropriate cancer screening should be carried out.

HIV is associated with focal segmental glomerulosclerosis.

Cryoglobulinaemia is associated with membranoproliferative glomerulonephritis.

There is nothing to suggest a haematological malignancy, therefore a bone marrow biopsy would not be indicated.

A renal tract ultrasound would be unlikely to be helpful in this situation.

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A 27-year-old man was reviewed in the nephrology clinic. He had been diagnosed with autosomal dominant polycystic kidney disease at the age of 18 following screening. His father, aunt and sister were also affected by the condition. He was taking ramipril 10mg daily. He was asymptomatic and blood pressure was 120/70 mmHg.

Blood tests showed that his eGFR had fallen from 90 mL/min/1.73 m² to 65 mL/min/1.73 m² over the past year. A recent ultrasound scan had shown multiple bilateral renal cysts, with both kidneys enlarging from 12cm to 14cm since the previous scan two years ago.

What treatment should he be offered?

A. Angiotensin receptor blocker (ARB)
B. Tolvaptan
C. Simvastatin
D. Bilateral nephrectomy
E. Renal transplant

B. Tolvaptan

Tolvaptan has been shown to reduce the rate of CKD progression in ADPKD (and is approved by NICE)

Tolvaptan is a vasopressin receptor 2 antagonist that has been shown to slow progression of autosomal polycystic kidney disease (ADPKD). It has been recommended for use by NICE in England and Wales since 2015 for patients with ADPKD and CKD stage 2 or 3 and evidence of rapidly progressing disease, as in this case. The main side effects are polyuria and polydipsia. Patients need regular monitoring of renal function and liver function tests during treatment.

There is no evidence that dual therapy with ACE inhibitors plus angiotensin receptor blockers (ARB) offers any benefit over monotherapy, especially as this patient's blood pressure is already well controlled.

Statins are not a specific treatment for ADPKD.

Nephrectomy is not indicated in this patient, as he is asymptomatic. Nephrectomy is sometimes considered for patients with cyst complications, such as infection, pain or bleeding.

Renal transplantation is not required as he does not have end-stage renal disease yet.

Question 61 of 183

A 45 year-old man attended for routine review in the HIV clinic. He had been taking highly-active retroviral therapy for 6 years and had been well during this time. He had no other past medical history of note.

Urinalysis performed in clinic showed protein 4+ and blood 1+. Examination revealed minimal oedema of both ankles. Cardiovascular, respiratory and abdominal examinations were unremarkable.

What is the most likely diagnosis?

A. Post-infectious glomerulonephritis
B. Focal segmental glomerulosclerosis
C. Minimal change disease
D. Acute interstitial nephritis

E. IgA nephropathy

B. Focal segmental glomerulosclerosis

HIV infection is a cause of focal segmental glomerulosclerosis

HIV can lead to a secondary FSGS (Focal segmental glomerulosclerosis) which often presents with a nephrotic syndrome, as in this case.

Question 62 of 183

A 62-year-old male presents with confusion. He lives alone and you are unable to establish a collateral history. He has a past medical history of COPD and heart failure. His current medicines include ramipril, spironolactone and bendroflumethiazide.

Blood results are as follows:

Hb	135 g/l	Na +	138 mmol/l
Platelets	242 * 10 /l 9	K +	3.9 mmol/l
WBC	12.8 * 10 /l 9	Urea	10.8 mmol/l
Neuts	8.8 * 10 /l 9	Creatinine	96 µmol/l
Chloride	110 * 10 /l 9	Glucose	5.2 mg/l
Bicarbonate	10 mEq/L		

What is the most likely cause of the acidosis?

A. Lactic acidosis
B. Diabetic ketoacidosis
C. Hyperchloremic metabolic acidosis
D. Addison's disease
E. Renal tubular acidosis

A. Lactic acidosis

Lactic acidosis occurs due to tissue hypoxia. Tissue hypoxia occurs due to one of four main mechanisms: 1. Hypoxaemia (e.g. respiratory pathology), 2. Toxic (e.g. carboxyhemoglobinemia, or cyanide poisoning), 3. Perfusional (e.g. shock), or 4. Severe anaemia

The patient is acidotic. The anion gap = (Na + K) - (HCO3 + Cl) = 21.9. Therefore the patient has a high anion gap metabolic acidosis (HAGMA). Common causes of HAGMA include ketoacidosis, uraemia, lactic acidosis and toxins (e.g. salicylates, ethylene glycol and methanol).

The normal glucose level makes diabetic ketoacidosis highly unlikely.

Hyperchloremic metabolic acidosis, Addison's disease and renal tubular acidosis are all causes of normal anion gap metabolic acidosis.

Therefore the most likely answer is lactic acidosis. The cause of this should be sought. Lactic acidosis occurs due to tissue hypoxia. Tissue hypoxia occurs due to one of four main mechanisms:

1. Hypoxaemia (e.g. respiratory pathology)
2. Toxic (e.g. carboxyhemoglobinemia, or cyanide poisoning)
3. Perfusional (e.g. shock)
4. Severe anaemia

Metabolic acidosis

Metabolic acidosis is commonly classified according to the anion gap. This can be calculated by: (Na + K) - (Cl + HCO). If a question supplies the chloride level then this is often a clue that the anion gap should be calculated. The normal range = 10-18 mmol/L

Normal anion gap (= hyperchloraemic metabolic acidosis)

- gastrointestinal bicarbonate loss:
    - prolonged diarrhoea: may also result in hypokalaemia
    - ureterosigmoidostomy
    - fistula
- renal tubular acidosis drugs: e.g. acetazolamide
- ammonium chloride injection
- Addison's disease

Raised anion gap

- lactate:
    - shock
    - sepsis
    - hypoxia
- ketones:
    - diabetic ketoacidosis
    - alcohol
- urate: renal failure
- acid poisoning: salicylates, methanol

Metabolic acidosis secondary to high lactate levels may be subdivided into two types:

- lactic acidosis type A: sepsis, shock, hypoxia, burns
- lactic acidosis type B: metformin

Question 63 of 183

A 67-year-old man with a history of heart failure treated with bisoprolol, ramipril, furosemide and spironolactone comes to the clinic for review. His heart failure symptoms are stable, but his main complaint is of painful gynaecomastia that has become more prominent over the past 6 months since he had a medication change. On examination his blood pressure is 125/80 mmHg, his pulse is 65 beats per minute and regular. There are only minor basal crackles at both lung bases on auscultation, and minor pitting oedema of both ankles. Routine blood testing is unremarkable including creatinine.

Which of the following is the most appropriate way to manage his gynaecomastia?

A. Change furosemide to bendroflumethiazide
B. Change spironolactone to eplerenone
C. Stop bisoprolol
D. Stop ramipril
E. Stop spironolactone

B. Change spironolactone to eplerenone

Eplerenone can be used in patients with troublesome gynaecomastia on spironolactone

Spironolactone both decreases testosterone production and increases peripheral conversion of testosterone to oestradiol. Both of these factors drive gynaecomastia. Eplerenone is a next generation aldosterone antagonist which doesn't cause gynaecomastia, given he has stable heart failure, switching is the most appropriate thing to do.

Changing furosemide to bendroflumethiazide will not impact on gynaecomastia and may worsen control of his heart failure. Stopping the spironolactone altogether will cause worsening of fluid retention Both bisoprolol and ramipril are associated with improvement in LV function and symptoms of heart failure, and should not be discontinued.

Question 64 of 183

A 44-year-old man is referred to the acute medical unit with a one-day history of rash and fever. His past medical history includes hypertension and osteoarthritis. He takes amlodipine and naproxen when required. He does not smoke or drink alcohol.

His observations are heart rate 92 beats per minute, blood pressure 143/88mmHg, respiratory rate 18/minute, oxygen saturations 97% on room air and temperature 37.7ºC.

On examination, there is mild blanching macular erythematous rash on his trunk. Chest auscultation is normal. There is no meningism. Heart sounds are normal with no added sounds or murmurs. Neurological examination is unremarkable. He appears euvolemic.

Urinalysis:

Leucocytes	+++
Nitrites	-ve
Blood	-ve
Protein	+
Glucose	-ve

Blood tests:

Hb	138 g/L	Male: (135-180)Female: (115 - 160)
Platelets	189 * 10 /L 9	(150 - 400)
WBC	10.9 * 10 /L 9	(4.0 - 11.0)
Neuts	6.8 * 10 /L 9	(2.0 - 7.0)
Lymphs	3.1 * 10 /L 9	(1.0 - 3.5)
Mono	0.2 * 10 /L 9	(0.2 - 0.8)
Eosin	0.8 * 10 /L 9	(0.0 - 0.4)
Na +	137 mmol/L	(135 - 145)
K +	4.1 mmol/L	(3.5 - 5.0)
Bicarbonate	21 mmol/L	(22 - 29)
Urea	9.2 mmol/L	(2.0 - 7.0)
Creatinine	155 µmol/L	(55 - 120)
CRP	4g/L	(<5)

What is the most likely diagnosis?

A. Acute interstitial nephritis
B. Cholesterol embolism
C. Eosinophilic granulomatosis with polyangiitis
D. Microscopic polyangiitis
E. Post-streptococcal glomerulonephritis

A. Acute interstitial nephritis

Acute interstitial nephritis causes an 'allergic' type picture consisting usually of raised urinary WCC and eosinophils, alongside impaired renal function

Acute interstitial nephritis is the correct answer. The patient presents with fever, rash, deranged renal function and eosinophilia in association with sterile pyuria, in the context of NSAID treatment. This is a typical presentation of acute interstitial nephritis.

Cholesterol embolism is incorrect. This can cause fever, rash, eosinophilia and acute kidney injury but is classically associated with interventional procedures such as coronary angiography.

Eosinophilic granulomatosis with polyangiitis is incorrect. This can plausibly cause fever, rash, eosinophilia and deranged renal function. However, the rash is likely to be non-blanching (vasculitis) and the urinalysis will demonstrate more significant haematuria and proteinuria if renal involvement. Furthermore, there is no history of asthma or sinusitis, making this very unlikely clinically.

Microscopic polyangiitis is incorrect. This can cause fever, rash and deranged renal function but it would not explain the eosinophilia, the rash would more typically be vasculitic and urinalysis would demonstrate blood and protein.

Post-streptococcal glomerulonephritis is incorrect. There is no history of preceding streptococcal infection. Furthermore, the urinalysis results are not typical of glomerulonephritis.

Question 65 of 183

A 53-year-old man is reviewed in the renal outpatient department and fatigue and shortness of breath. He has a history of end-stage renal failure secondary to hypertension and undergoes dialysis. He takes ramipril, amlodipine and doxazosin.

The examination is unremarkable other than an arterio-venous fistula noted at his right forearm. Blood tests:

Hb	84 g/L	Male: (135-180)Female: (115 - 160)
MCV	85 fL	(80-96)
Platelets	189 * 10 /L 9	(150 - 400)
WBC	4.4 * 10 /L 9	(4.0 - 11.0)
Na +	138 mmol/L	(135 - 145)
K +	4.8 mmol/L	(3.5 - 5.0)
Urea	28.1 mmol/L	(2.0 - 7.0)
Creatinine	389 µmol/L	(55 - 120)
Ferritin	88 µg/L	(24 to 336)
Transferrin saturation (TSAT)	12%	(20-50)

What is the appropriate management strategy?

A. Erythropoiesis-stimulating agent
B. IV iron supplementation
C. IV iron supplementation followed by consideration of erythropoiesis-stimulating agent
D. Oral iron supplementation
E. Oral iron supplementation followed by consideration of erythropoiesis-stimulating agent

C. IV iron supplementation followed by consideration of erythropoiesis-stimulating agent

Anaemia in CKD: erythropoiesis-stimulating agents should be considered once a patient is iron-replete

IV iron supplementation followed by consideration of erythropoiesis-stimulating agent is the correct answer. The patient is iron deficient (low normal ferritin and low TSAT). Patients with end-stage renal failure on dialysis typically require intravenous rather than oral iron replacement due to elevated serum levels of hepcidin preventing intestinal absorption of iron. Once the iron is replete, if the patient remains anaemic then an erythropoiesis-stimulating agent can be commenced with an aim to keep the haemoglobin in the range 100-120g/L. Anaemia in CKD may cause cardiomyopathy and left ventricular hypertrophy and the correction of anaemia with an ESA has been shown to reduced mortality.

Erythropoiesis-stimulating agent is incorrect. The patient needs iron replaced in the first instance before an ESA is considered. ESA has been shown to be less effective in patients that are iron deficiency.

IV iron supplementation is incorrect. If the patient remains anaemic after iron supplementation then an ESA should be considered.

Oral iron supplementation is incorrect. Patients with end-stage renal failure on dialysis typically require intravenous rather than oral iron replacement due to elevated serum levels of hepcidin preventing intestinal absorption of iron.

Oral iron supplementation followed by consideration of erythropoiesis-stimulating agent is incorrect. Oral iron is inferior to intravenous iron in patients on hemodialysis. This is due to elevated serum levels of hepcidin preventing intestinal absorption of iron and impairment of recycling of iron through the

Question 66 of 183

A 23-year-old man presents to the emergency department with six days of malaise, headache and a sore throat. He noticed that he has been passing blood in his urine on the last three occasions. He has no abdominal pain and is passing urine 3-4 times a day. He is otherwise well but has been diagnosed with coeliac disease 3 months ago after non specific lethargy and weight loss. He smokes 20 cigarettes a day.

He feels hot and cold and his observations show blood pressure 134/89 mmHg, heart rate 65 beats per minute, respiratory rate 22 breaths per minute and temperature 39ºC. A urine sample obtained is brown with sediment in and tests positive to blood and leucocytes.

On examination, his abdomen is soft and non tender. His throat is red with pus seen on the tonsils. He is tender over the maxillary sinus on the left side and complains of congestion here. There is no meningism and he is alert. He saw his GP three days earlier who started benzylpenicillin and performed the below investigations.

Na +	138 mmol/l
K +	4.6 mmol/l
Urea	7.6mmol/l
Creatinine	145 µmol/l
Throat swab	*commensal flora*
Urine	numerous red and white cell casts, no organisms seen
C3	normal
C4	normal
IgA	3.25 (normal range 0-0.5)
IgG	2.3 (normal range 2-3)
IgE	0.25 (normal range 0-0.5)

What is the likely diagnosis?

A. Tubulointerstitial nephritis
B. IgA nephropathy
C. Post streptococcal glomerulonephritis
D. Goodpasture's disease
E. Acute tubular necrosis

B. IgA nephropathy

IgA nephropathy is more common in coeliac disease

This is IgA nephropathy, considered an adult counterpart to Henoch-Schonlein Purpura. There is a higher incidence of IgA nephropathy in patients with coeliac disease and dermatitis herpetiformis. He also shows evidence of acute kidney injury. It is unlikely acute tubular necrosis which occurs in severe nephrotoxin or shocked states. Goodpasture's has lung manifestations which are not present here. Tubulointerstitial nephritis would have raised eosinophils and possibly a rash which are not seen here. The choice between post streptococcal and IgA nephropathy is difficult. IgA nephropathy usually occurs days rather than weeks after the onset of the upper respiratory tract infection. Furthermore, there is no culture of on the throat swab pointing towards a streptococcal infection.

Question 67 of 183

A 75-year-old gentleman attends the emergency department after a fall at home. He has felt unwell the past three days with reduced oral intake, nausea and feeling weak.

He is known to have hypertension and prostate enlargement and is awaiting a TURP in two months time. He usually suffers with frequency and nocturia but has not passed urine for the last 2 days ago. He takes tamsulosin, finasteride, ramipril and occasional naproxen for joint pains.

On examination, he is pale and lethargic. On examination, he has a palpable bladder. His chest has mild crepitations at the bases and his JVP is raised at 5cm. His calves are soft and not oedematous. Observations show heart rate 80/min, temperature 36.9 ºC, respiratory rate 22/min and blood pressure 140/85mmHg. He is catheterised and drains 1400ml if dark urine.

Hb	110 g/l	Na +	130 mmol/l
Platelets	278 * 10 /l 9	K +	6.0 mmol/l
WBC	11.2 * 10 /l 9	Urea	11.3 mmol/l
Neuts	9.6 * 10 /l 9	Creatinine	493 µmol/l
Lymphs	1.0 * 10 /l 9	CRP	23 mg/l
PSA	5.3 ng/ml (0-2.0 normal range)		

Urine dip	blood +++, leucocytes ++, nitrites -
Ultrasound	catheterised bladder with no hydronephrosis, both kidneys
Chest x-ray	Increased alveolar shadowing in lower zone, small pleural

What is the likely cause of this acute kidney injury?

A. Urinary tract obstruction
B. Congestive cardiac failure
C. Sepsis
D. Medication use
E. Rapidly progressive glomerulonephritis

A. Urinary tract obstruction

Ultrasound detects only 95% of urinary tract obstruction in AKI

This man is in acute kidney injury and has developed secondary pulmonary oedema. There are many causes. He is not overtly septic. His medications which include ACE inhibitors and an NSAIDs can contribute to renal failure but this would be more likely on initiating or increasing dose. Rapidly progressive glomerulonephritis can occur but typically is on a background of a vasculitis with additional features to suggest that. We know the gentleman has an enlarged prostate with a palpable bladder on examination. Given his total anuria for two days and enlarged prostate, it is likely this caused obstruction. Ultrasound can be negative in 5% of cases of obstructive uropathy and therefore the clinical story is of great importance in acute kidney injury. The signs of congestive cardiac failure are similar but there would usually be risk factors such as an extensive cardiac history and they would be passing urine.

Question 68 of 183

A 64-year-old woman was admitted to the acute medical unit with polyuria and polydipsia. She described needing to pass urine at least three times every night, and was constantly thirsty. She was drinking regularly overnight and taking a glass of water to bed. She had a past medical history of hypertension, ischaemic heart disease, and bipolar disorder. She was taking aspirin, lithium, and bisoprolol. An overnight water deprivation test was done and the results are below

Investigations

Na$^+$	145 mmol/l(137-144)
K$^+$	4.5mmol/l(3.5-4.9)
Urea	11.5 mmol/l(2.5-7.0)
Creatinine	188µmol/l(60-110)
Random blood glucose	7.2mmol
Serum osmolality	370mosmol/kg(278-300)
Urine osmolality	165mosmol/kg(350-1000)

A d DAVP (1-deamino-8-D-arginine vasopressin) test was performed and the urinary osmolality remained unchanged.

What is the most likely diagnosis?

A. Nephrogenic diabetes insipidus
B. Cranial diabetes insipidus
C. Pre renal acute kidney injury
D. Type 1 diabetes mellitus
E. Primary polydipsia

A. Nephrogenic diabetes insipidus

Lithium is a recognised cause of nephrogenic diabetes insipidus

The clinical picture of polyuria, polydipsia, and nocturia, and the biochemical profile is consistent with diabetes insipidus. Diabetes insipidus is diagnosed with a water-deprivation test. A lack of response (failure to concentrate the urine) to desmopressin would suggest nephrogenic diabetes insipidus. Up to 20% of patients taking lithium develop impairment of urinary concentration due to dysregulation of the aquaporin system in the collecting duct. This is usually, but not always, reversed on stopping the lithium.

In cranial diabetes insipidus, the urine osmolality increases with the use of desmopressin. The random blood glucose is not in the diabetes range. For primary polydipsia and pre-renal acute kidney injury, urine osmolality will be >600mosm/kg after water deprivation test.

Question 69 of 183

A 51 year-old man presents with a twelve-hour history of generalised abdominal pain, nausea and vomiting. The abdominal pain has been increasing in severity to a 6/10 on the pain scale and there is evidence of guarding. The patient is currently on peritoneal dialysis for end-stage renal failure and he has noticed cloudy peritoneal bags over the last six hours. He is on ramipril, alfacalcidol, erythropoietin and simvastatin. His observations are all stable and peritoneal dialysis fluid has been sent off for microscopy, culture and sensitivity.

What is the next most appropriate management of this patient?

A. Give IV co-amoxiclav
B. Arrange immediate removal of catheter
C. Swab catheter site and give erythromycin
D. Give intraperitoneal vancomycin + ceftazidime
E. Perform a sepsis screen and await peritoneal dialysis fluid microscopy result

D. Give intraperitoneal vancomycin + ceftazidime

Peritoneal dialysis peritonitis: treat with **intraperitoneal** vancomycin + ceftazidime

This patient has an infection in their peritoneal dialysis fluid, common organisms being *Staphylococcus aureus*, *Pseudomonas aeruginosa* and *Staphylococcus epidermis*. This is a medical emergency and treatment should be commenced as soon as possible. The optimal treatment would be intraperitoneal vancomycin + ceftazidime.

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A 58-year-old man with progressive chronic kidney disease secondary to IgA nephropathy was reviewed in the nephrology clinic. He reported feeling well except for mild ankle swelling. Blood tests are shown below:

Hb	104 g/L	Male: (135-180)Female: (115 - 160)
Platelets	276 * 10 /L $_9$	(150 - 400)
WBC	8.2 * 10 /L $_9$	(4.0 - 11.0)
Na$^+$	133 mmol/L	(135 - 145)
K$^+$	5.8 mmol/L	(3.5 - 5.0)
Urea	18.5 mmol/L	(2.0 - 7.0)
Creatinine	912 µmol/L	(55 - 120)
eGFR	5 mL/min/1.73 m^2	>60 mL/min/1.73 m^2
Bicarbonate	17 mmol/L	(20-28)
Calcium	2.05 mmol/L	(2.1-2.6)
Phosphate	1.8 mmol/L	(0.8-1.4)

Following this appointment, it was decided that he needed to start dialysis.

What is the most likely indication for starting dialysis in this case?

A. Hyperkalaemia
B. Acidosis
C. Fluid overload

D. Low eGFR
E. Uraemia

D. Low eGFR

Dialysis should be started in CKD stage 5 if symptomatic or when eGFR drops to 5-7

According to NICE guidelines, dialysis should be started 'when indicated by the impact of symptoms of uraemia on daily living, or biochemical measures or uncontrollable fluid overload, or at an estimated glomerular filtration rate (eGFR) of around 5 to 7 ml/min/1.73 m2 if there are no symptoms'.

In this case, the patient does not have any uraemic symptoms such as lethargy, nausea, itching or confusion (encephalopathy).

His eGFR is 5 ml/min/1.73 m2, which would be an indication to start dialysis.

This patient has mild hyperkalaemia, fluid overload and acidosis, none of which would be indications to start dialysis by themselves.

Question 71 of 183

A 69-year-old-male attends clinic for review. Today he is complaining of fleeting joint pains and a progressive rash on both legs.

He suffers from chronic back pain but hasn't seen his GP, instead, taking over the counter painkillers for the past six weeks. He has a background of hypertension.

On examination, you are unable to find any evidence of active synovitis. You note a symmetrical eruption of palpable, red-purple papular lesions across the extensor surfaces of both legs. He has vesicular breath sounds with oxygen saturations at 99% on room air. His heart sounds are normal, with no murmurs. His clinic blood pressure reading is 146/88 mmHg. His abdomen is soft and non-tender.

Hb	132 g/l	Na +	135 mmol/l
Platelets	155* 10 /l 9	K +	5.1 mmol/l
WBC	9.9 * 10 /l 9	Urea	7.3 mmol/l
Neuts	5.1 * 10 /l 9	Creatinine	256 µmol/l
Lymphs	1.0 * 10 /l 9	CRP	6 mg/l
Eosin	2.5 * 10 /l9		

Which of the following is the most likely diagnosis?

A. Churg-Strauss syndrome
B. Tubulointerstitial nephritis
C. Dermatitis herpetiformis
D. Hypereosinophilic syndrome
E. Cutaneous larva migrans

B. Tubulointerstitial nephritis

This man's overzealous use of NSAIDs has led to a tubulointerstitial nephritis. Whilst an autoimmune vasculitis is plausible, Churg-Strauss would, classically, also present with respiratory features.

Question 72 of 183

A 62-year-old man is seen in renal transplant clinic. He had a cadaveric kidney transplant 1 years ago that was initially complicated by rejection and has required high dose immunosuppression. He has good graft function with creatinine 121 umol/l and no proteinuria. He complains of 2 stone weight loss in 2 months and feels tired.

There is a large painless lymph node in his right axilla and a palpable node in his left inguinal region.

Hb	8.7 g/dl
Platelets	195 * 10 /l9
WBC	6.7 * 10 /l9

Which is the most useful virology test?

A. BK virus
B. Cytomegalovirus
C. Epstein Barr virus
D. Hepatitis B
E. HIV

C. Epstein Barr virus

This patient has post-transplantation lymphoproliferative disorder (PTLD) indicated by:

- weight loss
- anaemia
- lymphadenopathy

His high dose immunosuppression have precipitated infection with EBV and subsequent B cell proliferation. PTLD incidence is highest in the first year post-transplantation, which is the time when immunosuppression is the most intense. Treatment involves reducing his current immunosuppression therapy and giving additional chemotherapy.

CMV causes systemic symptoms, colitis, pneumonitis and graft dysfunction.

BK virus causes graft dysfunction and ureteric stenosis.

Question 73 of 183

You see a 56-year-old female in the renal low clearance clinic with known progressive proteinuric diabetic nephropathy. Despite maximal medical therapy she has had poorly controlled type 1 diabetes since 18 years of age; she also has hypertension and hypercholesterolaemia. She is asymptomatic and feels fit and well but is concerned regarding the deterioration in her kidney function. Cardiovascular, respiratory and abdominal examinations are unremarkable. A sensory peripheral neuropathy, reduced dorsalis pedis pulses bilaterally and pitting oedema to both ankles are noted. Fundoscopy shows evidence of photocoagulation therapy in both eyes. Today's blood results show:

Hb	109 g/l	Na +	129 mmol/l
Platelets	111 * 10 /l 9	K +	5.1 mmol/l
WBC	5.7 * 10 /l 9	Urea	22 mmol/l
Neuts	4.6 * 10 /l 9	Creatinine	259 µmol/l
HbA1c	86 mmol/mol	eGFR	18 ml/min/1.732

Her eGFR 3 months ago was 20.

Considering her progressive stage 4 chronic kidney disease what is the gold standard treatment option in this case?

A. Haemodialysis
B. Combined renal and pancreas transplant
C. Peritoneal dialysis
D. Pancreas transplant
E. Renal transplant

B. Combined renal and pancreas transplant

Patients with type one diabetes nearing end stage renal failure should be considered for joint pancreas and renal transplants. Having said this all decisions regarding renal replacement therapy require a multidisciplinary approach and should be tailored to the patient's lifestyle and preferences. Pancreatic and renal transplantation replace renal function and treat the underlying cause of dysfunction however it commits the patient to lifelong immunosuppression. Haemodialysis usually requires thrice-weekly sessions which can be performed at home but usually at the local dialysis centre. Peritoneal dialysis allows for more mobility and reduction in symptom variation but presents a risk of peritonitis. Renal replacement therapy is a complex decision and requires extensive work up hence is must be considered early in the presence of progressive renal disease.

Pancreas transplant alone may address the diabetes but not the renal function and will complicate options further down the line should renal function deteriorate further.

Question 74 of 183

A 48-year-old male presents to his general practitioner with a 3-month history of worsening swelling of both arms and legs. He has chronic lower back pain and has noticed an accidental increase of 4 kg over 2 months. He has a past medical history of ankylosing spondylitis, hypertension and hypercholesterolaemia and currently takes regular paracetamol, amlodipine 5mg OD and simvastatin 40 mg ON. On examination observations are normal, there is a reduction in all spinal movements and pitting oedema to his mid thighs bilaterally; there is no redness, tenderness or heat. His face and arms also appear swollen although no pitting is present. Respiratory, cardiovascular and abdominal examinations are normal.

Urine dip shows 3+ protein nil else and bloods are as follows:

Hb	115 g/l	Na +	139 mmol/l
Platelets	380 * 10 /l 9	K +	4.3 mmol/l
WBC	8.1 * 10 /l 9	Urea	4.2 mmol/l
Neuts	6.2 * 10 /l 9	Creatinine	73 µmol/l
Lymphs	1.8 * 10 /l 9	CRP	49 mg/l
Eosin	0.02 * 10 /l 9	Albumin	25 g/l

What is the most likely cause of this patient's oedema?

A. Minimal change nephropathy
B. Amlodipine
C. AL amyloidosis
D. Hypertensive nephropathy
E. AA amyloidosis

A. AA amyloidosis

This patient is demonstrating the triad of oedema, low albumin, and proteinuria suggesting nephrotic syndrome. Further quantification of proteinuria via spot protein creatine ratio or 24-hour collection is required. The above subacute nephrotic picture would suggest amyloidosis or minimal change nephropathy as the most likely causes out of the five options. This patient has chronic back pain and a raised CRP suggesting poorly controlled ankylosing spondylitis. This would support AA amyloidosis as the most likely cause of his nephrotic syndrome. Amyloidosis is a rare disorder caused by deposition of abnormal amyloid protein. It can affect multiple systems including the kidneys, heart and peripheral nerves. AA amyloidosis occurs secondary to a poorly controlled chronic inflammatory condition such as ankylosing spondylitis or rheumatoid arthritis. The key to treatment is to address the underlying condition.

AL amyloidosis is not linked to another condition and results from abnormal plasma cells producing excessive amounts of light chains in a process similar to multiple myeloma.

Question 75 of 183

A 66-year-old gentleman is reviewed in the General Medical Clinic with a 4-week history of polyuria, polydipsia, and generalised muscle weakness. He denies weight loss but does describe the presence of deep aching pains in his arms, legs, and lower back.

His past medical history is remarkable for hypertension, treated with amlodipine 5mg once daily and ramipril 7.5mg once daily. He also tells you that he has an 'antibody disorder' that requires no specific treatment but necessitates yearly follow-up at the Haematology Clinic.

Examination reveals a well-looking gentleman. His pulse is 74 bpm and his blood pressure is 132/63mmHg. There is no conjunctival pallor and his chest is clear to auscultation. His abdomen is soft and non-tender.

A series of investigations are requested:

Hb	132 g/l	Na +	136 mmol/l	Bilirubin	17 µmol/l
Platelets	278 * 10 /l 9	K +	2.9 mmol/l	ALP	172 u/l
WBC	6.5 * 10 /l 9	Cl -	115 mmol/l	ALT	35 u/l
Neuts	3.8 * 10 /l 9	Urea	5.2 mmol/l	γGT	39 u/l
Lymphs	1.8 * 10 /l 9	Creatinine	87 µmol/l	Total Protein	92 g/l
Eosin	0.01 * 10 /l 9	Albumin	40 g/l		

Calcium	2.24 mmol/l
Adj. Calcium	2.24 mmol/l
Phosphate	0.38 mmol/l
Bicarbonate	18 mmol/l
Fasting glucose	4.9 mmol/l
Urine dip	Glucose ++ Protein ++
Sodium nitroprusside test	Positive

What is the most likely diagnosis?

A. Multiple myeloma
B. Fanconi syndrome

C. Diabetes mellitus
D. Cystinuria
E. Pseudohypoaldosteronism

B. Fanconi syndrome

A difficult question: the patient has presented with fairly non-specific symptoms and a raft of metabolic abnormalities. The easiest approach is to consider each of the diagnoses in turn.

The history of bone pain combined with the presence of an underlying 'antibody disorder' could point you toward a diagnosis multiple myeloma. This is supported by the large discrepancy between the total protein and albumin, indicating that the patient's globulins are grossly elevated. However, the blood tests also reveal the presence of normal haemoglobin, calcium, and renal function. Taken together, these abnormalities point instead to a diagnosis of monoclonal gammopathy of uncertain significance (MGUS).

Diabetes mellitus would explain the polyuria, polydipsia, and glycosuria, however, the normal fasting glucose makes this unlikely.

Pseudohypoaldosteronism is due to aldosterone resistance and leads to renal salt wasting and hyperkalaemia. This is inconsistent with the biochemistry in the vignette.

Fanconi syndrome is a generalised disorder of proximal renal tubular function. Reabsorption of filtered solutes is impaired and as a result, large quantities of bicarbonate, potassium, phosphate, glucose, uric acid and amino acids are lost in the urine. It is associated with the development of type 2 (proximal) renal tubular acidosis, as indicated by the elevated chloride and low bicarbonate in the patient's blood results. The remaining metabolic abnormalities can all be explained by the loss of these solutes in the urine, which in turn explain the presence of glucose and protein on urine dipstick.

A number of congenital and acquired conditions can lead to the development of Fanconi syndrome. although onset in later life suggests an acquired cause. In this case, MGUS is likely to be the predisposing factor.

The clinical features that cause patients to seek treatment are usually polydipsia and polyuria secondary to the osmotic effect of the urinary solute load. Bone pain due to osteomalacia and muscle weakness due to hypophosphataemia may also occur. Hypokalaemia may cause palpitations and can occasionally result in sudden death due to cardiac arrhythmia.

Cystinuria causes recurrent renal calculi that typically present in the first decades of life. The diagnosis was historically made by the sodium nitroprusside test, which is included here as a red herring and is positive due to the generalised aminoaciduria associated with Fanconi syndrome.

Question 76 of 183

A 51-year-old gentleman presents to the Emergency Department with a 4-hour history of severe left-sided flank pain. The pain is constant, but waxes and wanes and is associated with multiple episodes of vomiting.

His past medical history is remarkable for congestive cardiac failure, gout, and glaucoma. He has also recently been diagnosed with HIV and started highly active antiretroviral treatment (HAART) 6 months ago.

On examination, the patient is afebrile. His pulse is 96bpm and his blood pressure is 142/79mmHg. His chest is clear and his abdomen is soft, with left-sided costovertebral angle tenderness evident on palpation.

His urine dipstick is as follows:

pH	6.0
Specific gravity	1.020
Blood	+++
Protein	+

A CT KUB is requested and demonstrates inflammatory stranding around the left kidney with mild hydronephrosis but no obvious ureteric calculi.

The patient is admitted for analgesia and hydration but remains symptomatic over the next 24 hours. He undergoes intravenous pyelography which demonstrates a filling defect in the midureter.

Which of the following medications is most likely to be responsible for his symptoms?

A. Acetazolamide
B. Furosemide
C. Tenofovir
D. Indinavir
E. Allopurinol

D. Indinavir

question describes one of the rare instances in which a radiolucent stone on CT KUB may lead to a missed diagnosis.

Furosemide, acetazolamide, and allopurinol predispose to the formation of calcium oxalate, calcium phosphate, and uric acid stones respectively. Calcium stones are readily visible on both plain x-ray and CT whilst uric acid stones are only detectable with the latter. Supersaturation of urine with indinavir can, however, give rise to pure indinavir stones that are undetectable on both plain x-ray or computed tomography.

Hydration and analgesia are the recommended first-line treatment for indinavir stones, although temporary or permanent discontinuation of the drug may be required.

Tenofovir is not associated with renal stone formation.

Question 77 of 183

A 56-year-old male was admitted to the Medical Admission Unit with shortness of breath. It was of relatively sudden onset having developed over the course of the last five days. It initially occurred with severe exertion but gradually progressed such that by the time of admission he was breathless at rest with significant orthopnoea and paroxysmal nocturnal dyspnoea. His past medical history included angina for which he was awaiting hospital investigation, hypertension, hypercholesterolaemia and a transient ischaemic attack three years ago. Over the course of the last few weeks, he had been seeing his GP with poor blood pressure control. In addition to felodipine M/R 5mg OD, indapamide 25mg OD and bisoprolol 7.5 mg OD the GP introduced ramipril 2.5mg OD two weeks ago. His other medication comprised of aspirin 75 mg OD, dipyridamole M/R 200mg BD, GTN spray PRN and simvastatin 40mg OD. He smoked 40 cigarettes per day and consumed 38 units of alcohol per week.

On examination to the Medical Admission Unit, he was short of breath at rest, with a respiratory rate of 26/min and oxygen saturations of 92% on air. His blood pressure was 158/78 mmHg and heart rate 54 bpm. Examination of the cardiovascular system revealed the presence of normal heart sounds, a JVP of 4 cm and the absence of pedal oedema. Examination of the respiratory system revealed bibasal crackles, whilst examination of the gastrointestinal and neurological systems was unremarkable.

Initial investigations revealed the following results:

Na +	138 mmol/l
K +	4.6 mmol/l
Urea	18.9 mmol/l
Creatinine	245 µmol/l
Glucose	5.7 mmol/l
Troponin I	<0.05
CRP	2 mg/l
ESR	6 mm/hr

ECG: normal sinus rhythm 56 bpm Chest x-ray: bilateral pulmonary oedema, normal heart diameter

Urinalysis: NAD

Transthoracic echocardiogram: normal systolic function, presence of aortic stenosis with pressure gradient of 18mmHg, as well as mild mitral regurgitation. Treatment was commenced with IV furosemide 80mg OD and GTN, as well as IV diamorphine 2.5 mg OD with excellent effect and the patient recovered over the next two days.

What is the next best investigation to determine the cause of this gentleman's symptoms?

A. Coronary angiography
B. Renal angiography
C. Myocardial perfusion scanning
D. Renal magnetic resonance angiography
E. No further investigations required

D. Renal magnetic resonance angiography

This gentleman has exhibited flash pulmonary oedema occurring in response to the initiation of ramipril. He is a known vasculopath, and after commencing an ACE-inhibitor experienced rapid onset pulmonary oedema with a decline in renal function in the presence of normal systolic function. The most likely diagnosis is, therefore, atheromatous renal artery stenosis. Of the available options, MR angiography is less invasive than renal angiography and therefore would be most appropriate as the best next option to confirm the diagnosis. He will indeed also require coronary angiography; however, this would not be the most appropriate best next option to investigate the cause of this episode.

Question 78 of 183

A 24-year-old female with a history of sickle cell trait presents to the outpatient department with the results of her routine urine examination which was performed after she complained of an intermittent burning sensation just after passing urine. She works as a fitness instructor at a nearby gym and has said that she takes ibuprofen regularly for recurrent muscle spasms due to exercise. She has no known drug allergies but mentions that she does develop itchy skin and rhinitis when the season changes.

On examination, her blood pressure is 145/95 mmHg and there is mild periorbital puffiness. Her liver is palpable 1 finger breadth below the right costal margin with a total span of 11cms.

Lab results revealed:

Hb	80 g/l
MCV	62 fl
MCH	20 pg
WBC	9 * 10 /l9
Plt	350 * 10 /l9
Urea	8.1 mmol/l
Creatinine	275 µmol/l
Na+	140 mmol/l
K+	4.1 mmol/l
Albumin	30 g/l

Urine routine examination showed proteinuria 2+

Renal biopsy showed uniform granular capillary wall deposits of IgG and C3 complement.

What is the most appropriate management plan?

A. Oral high dose corticosteroids with azathioprine
B. Blood pressure control with ACE inhibitors and immunosuppression withchlorambucil/oral prednisolone
C. Blood pressure control with ACE inhibitors and plasmapheresis
D. Simple observation and regular monitoring of renal functions
E. Haemofiltration

B Blood pressure control with ACE inhibitors and immunosuppression withchlorambucil/oral prednisolone

The diagnosis is membranous glomerulonephritis which could be secondary either to this lady's sickle cell trait or the use of NSAIDS.

The main difference in the management of membranous glomerulonephritis and the other glomerulopathies is that high dose steroids and azathioprine are not associated with any significant benefits in the former. The main treatment strategy involves control of blood pressure with ACE inhibitors and immunosuppression with either cyclophosphamide, chlorambucil, ciclosporin or mycophenolate mofetil along with prednisolone. Rituximab has also been shown to have a benefit in the short term.

Question 79 of 183

A 71-year-old patient with end-stage renal failure secondary to adult polycystic kidney disease presents with new painful necrotic skin lesions. Her general practitioner has referred her to hospital as they have failed to improve with oral antibiotics.

On arrival she is afebrile. A bruit and thrill were noted over a left brachiocephalic fistula. She has an irregular 8cm x 4 cm, punched-out ulcer on the medial aspect of her left calf just below her knee. The centre of the ulcer is black and necrotic. There is minimal surrounding erythema.

Bloods results revealed:

White cell count	10.0 * 109/l
C-reactive protein (CRP)	17 mg/dL

Which investigation will confirm the diagnosis?

A. Skin swab
B. Parathyroid hormone (PTH)
C. Skin biopsy
D. Anti-neutrophil cytoplasmic antibody (ANCA)
E. Computer tomography angiogram of left leg

B. Parathyroid hormone (PTH)

Question 80 of 183

A 72-year-old woman is admitted with a cough and fever. She has been unwell for four days with a worsening productive cough, chest pain, fever and shortness of breath. Observations show tachycardia, fever, and hypoxia. She weighs 42kg. A chest x-ray demonstrated right middle lobe consolidation and she is diagnosed with a lower respiratory tract infection and sepsis.

She is given IV fluids, IV antibiotics and a urinary catheter is inserted. She is reviewed at the posttake ward round 8 hours later. Since then she has passed 141ml of urine, which appears dark. You calculate that she is passing urine at a rate of 0.42ml/kg/hr. Creatinine from admission is 87µmol/L. There are no previous levels of creatinine measured.

What is the most appropriate description of her renal function?

A. No renal impairment
B. Stage I acute kidney injury
C. Stage II acute kidney injury
D. Stage III acute kidney injury
E. Chronic renal disease

B. Stage I acute kidney injury

The correct answer is stage I acute kidney injury (AKI). This diagnosis is due to her reduced urine output. She has passed 141ml in 8hours and weighs 42kg. Dividing 141ml by 8hrs and 42kg gives a urine output rate of 0.42ml/kg/hr. As she has a urine output of <0.5ml/kg/hr for more than six hours but less than 12, she has a stage I kidney injury. There is no baseline creatinine to compare to making a diagnosis based on creatinine levels difficult.

KDIGO grading of AKI:

Stage	Serum creatinine	Urine output
1	1.5-1.9 times baseline OR increase > 26.5 µmol/L	<0.5ml/kg/hr for 6-12 hours
2	2.0-2.9 times baseline	<0.5ml/kg/hr for =>12 hours
3	3.0 times baseline OR increase => 353.6 µmol/L OR initiation of renal replacement therapy	anuria =>12 hours

Question 81 of 183

You are called to the haematology ward during an evening on-call to review an elective chemotherapy patient. A 23-year-old male was electively admitted to start chemotherapy for Hodgkin's lymphoma the following day and is reporting pain in his left flank. He is currently under investigation by the renal physicians for deteriorating creatinine of unknown cause for the past 4 months, does not take regular medications, smokes about 10 cigarettes every day and occasionally smokes marijuana.

On examination, the patient has bilateral lower limb non-pitting oedema to knees. You note a left varicocele and mild bilateral scrotal oedema. His heart sounds and auscultation of his chest is unremarkable. Abdominal examination reveals only mild left renal angle tenderness. A urine dip reveals 4+ blood, 2+ protein, normal pH. On review of his old clinic letters, you note a renal biopsy from 2 months prior to show IgG and complement deposits on the basement membrane.

His current serum results are:

K +	5.4 mmol/l
Urea	12.8 mmol/l
Creatinine	212 µmol/l
Albumin	18 g/l

What is the most likely acute diagnosis?

A. Renal vein thrombosis
B. Renal calculus
C. IgA nephropathy
D. Anti-glomerular basement membrane disease
E. Membranoproliferative glomerulonephritis

A. Renal vein thrombosis

An underlying diagnosis of membranous glomerulonephritis is almost given in the renal biopsy. This would be consistent with a renal complication of lymphomatous disease. However, this is insufficient to explain the acute presentation of flank pain, oedema and varicocele. Although the precise pathophysiology is unclear, there is a significant association between nephrotic syndrome and renal vein thrombus. In this case, the left renal vein thrombus would explain the left varicocele and flank tenderness. Other causes of glomerulonephritis offered are unlikely to produce pain. Renal calculi should not produce oedema and varicocele.

Question 82 of 183

A 74 year old lady with a background of type 2 diabetes mellitus, hypertension and previous myocardial infarction presents to the Emergency Department with a 2 day history of abdominal pain and profuse diarrhoea which has become bloody in the last 24 hours. Her temperature is 37.2º, heart rate 102 beats per minute and blood pressure 106/74 mmHg. On examination her heart sounds are normal, chest is clear and she has a diffusely tender abdomen.

Hb	10.4 g/dl
Platelets	64 * 10 /l9
WBC	14.2 * 10 /l9
Urea	10 mmol/l
Creatinine	154 µmol/l
Bilirubin	56 µmol/l
CRP	125 mg/l

A blood film shows fragmented red blood cells.

Given the most likely diagnosis, what is the most appropriate management for this patient?

A. Loperamide and IV metronidazole

B. Supportive management and notify a consultant in communicable disease control
C. Urgent plasma exchange
D. Platelet transfusion
E. Loperamide and PO ciprofloxacin

B. Supportive management and notify a consultant in communicable disease control

E. coli 0157 is an infectious gastroenteritis that can prove fatal particularly in the young and the elderly. Complications include haemolytic uraemic syndrome (HUS) and TTP. It may cause a spectrum of presentations from asymptomatic infection to haemorrhagic colitis and HUS. Thrombocytopenia and fragmented erythrocytes suggest the onset of HUS.

Although no clues are given in the question as to the source of this patient's infection, it is often the case that there has been contact with farm animals. Other routes of infection include personto-person contact and from contaminated water e.g. contaminated lakes/streams/swimming pools/non-chlorinated water supply.

Management is supportive and cases need to be reported to a consultant in communicable disease control (CCDC).

Question 83 of 183

A 50 year-old woman if referred by her GP due to refractory hypertension, despite having taken in the past ramipril, bendroflumethiazide and amlodipine. She has no other symptoms and past medical history includes hyperthyroidism which was treated with carbimazole ten years ago. She has a 20-year pack history and drink 5-10 units of alcohol per week. Her occupation is as an office worker and she has recently complained of pain in both of her calves when walking the 15 minutes to and from work. On examination, her blood pressure is 161/98 mmHg, she has cold peripheries and she is otherwise well.

Blood tests reveal:

Na +	145 mmol/l
K +	3.3 mmol/l
Urea	5.1 mmol/l
Creatinine	81 µmol/l
Renin activity	High
Aldosterone activity	High

What is the most likely diagnosis?

A. Renal artery sclerosis
B. Fibromuscular dysplasia
C. Cushing's disease
D. Cushing's syndrome
E. Primary hyperaldosteronism

A. Renal artery sclerosis

This patient has refractory hypertension, hypernatraemia and low potassium levels. When this is taken with her high renin and aldosterone activity, this suggests secondary hyperaldosteronism. Arteriosclerosis is the most common cause of renal artery stenosis and she demonstrates that she already has peripheral vascular disease by the pain in her calves on walking. Fibromuscular dysplasia is a rarer cause of renal artery stenosis and tends to occur in women aged 30-40 years. Both Cushing's disease and syndrome are unlikely, as she does not display any other signs or symptoms other than her calf pain. In primary hyperaldosteronism, the renin would be suppressed, rather than raised.

Question 84 of 183

A 25 year old man was admitted under section to an inpatient psychiatric hospital after a severe manic episode. The patient had suffered several previous episodes of depression but had not previously been admitted to hospital for a mental health problem. Treatment for bipolar affective disorder with lithium, lorazepam and olanzapine had been initiated.

Shortly after admission, routine blood tests showed the patient to be hyponatraemic and he was noted to be passing large volumes of urine by ward staff. Following advice from the endocrine team, basic investigations were requested as detailed below.

Sodium	129 mmol / L
Potassium	3.7 mmol / L
Urea	1.8 mmol / L
Creatinine	55 micromol / L
Calcium (adjusted)	2.41 mmol / L (reference 2.20-2.60)
Serum glucose (random)	5.4 mmol / L
Thyroid stimulating hormone	0.8 microU / L (reference 0.4-5.0)
T4 free serum	12.5 pmol / L (reference 8.5-15.2)
Plasma osmolality	265 mmol / L (reference 280-295)
Urine osmolality	80 mmol / L (reference 100-900)
Serum Lithium	0.35 mEq / L (reference 0.8-1.2)

Urine dipstick: negative glucose; negative protein; negative nitrites; negative leucocytes; negative ketones

What is the most likely cause of the patient's polydipsia?

A. Nephrogenic diabetes insipidus secondary to lithium therapy
B. Syndrome of inappropriate anti-diuretic hormone secretion secondary to olanzapine
C. Idiopathic cranial diabetes insipidus
D. Primary polydipsia
E. Excessive alcohol consumption

D. Primary polydipsia

Primary polydipsia is a rare cause of polyuria in the general population but is common among institutionalised psychiatric patients (6-20 %). The plasma and urine osmolality results imply a normal renal response to ADH making primary polydipsia the most likely cause in this case.

The plasma and urine osmolality results are not consistent with SIADH or diabetes insipid us which can be caused by the drugs mentioned in the incorrect stems. The patient has no obvious biochemical cause for his polydipsia such as diuretic medication, diabetes mellitus, hyperthyroidism or electrolyte abnormality. Lithium therapy can cause polydipsia but this is unlikely given sub-therapeutic serum levels. There is no history to suggest excess alcohol consumption.

Question 85 of 183

You are called to see a 34-year-old gentleman following an episode of haematemesis on the dialysis ward. His haemoglobin has dropped from 101g/L to 69g/L. His blood pressure is 98/54 mmHg, with a heart rate of 100bpm. You note that he is currently awaiting a renal transplant for end-stage diabetic nephropathy. He has no other past medical history. You organise an urgent OGD.

How would you manage his anaemia?

A. Hold transfusion and expedite the OGD
B. Transfuse and inform the transplant team
C. Increase the dose of erythropoietin
D. Contact the renal team for advice regarding transfusion
E. Transfuse and administer terlipressin

B. Transfuse and inform the transplant team

Transfusions in patients awaiting renal transplants are usually avoided where possible, due to the potential risk of circulating antibodies and thus organ rejection. In this example, the patient is haemodynamically unstable and therefore an urgent blood transfusion would be advised in this instance. The transplant team would need to be informed and the patient advised that a transplant will be postponed for at least 3 months, following repeat antibody screening. Terlipressin is only advised in patients with known oesophageal varices or acute hepato-renal syndrome. There is no role for increased dose of erythropoietin in the acute setting.

Question 86 of 183

A 59-year-old gentleman is admitted to hospital with lower abdominal pain. He has a past medical history includes Parkinson's disease, psoriasis and high cholesterol. Current medications include methotrexate 10mg once weekly, bromocriptine 2.5mg BD, atorvastatin 20mg ON, levodopa 200mg TDS and entacapone 200mg OD.

Observations on admission are as follows: heart rate 102 beats per minute, blood pressure 125/62 mmHg, respiratory rate 18/min, oxygen saturations 97% (room air), temperature 37.2ºC. On examination, there is bilateral loin tenderness, with no evidence of peritonism. Bowel sounds are active. Urine dipstick confirms blood 2+ with no evidence of nitrites or leucocytes. An abdominal x-ray is unremarkable.

Which medication is likely to be implicated with the underlying diagnosis?

A. Bromocriptine
B. Entacapone
C. Methotrexate
D. Atorvastatin
E. Levodopa

A. Bromocriptine

The correct answer is bromocriptine. This gentleman has presented with retroperitoneal fibrosis, resulting in secondary hydroureters and hydronephrosis. Examination findings of bilateral loin pain, with associated haematuria, is consistent with this. Though not present in this case, there is an association with inflammatory bowel disease. The exact pathogenesis has not been clearly described, but evidence supports the notion that it results from an immunological response to antigens within atherosclerotic plaques.

Medications associated with retroperitoneal fibrosis include bromocriptine, beta-blockers and methysergide.

Question 87 of 183

A 57-year-old man is admitted with general lethargy. He is currently being treated for bladder cancer, which has been complicated by paraneoplastic Guillain-Barre syndrome. Focussed examination is largely unremarkable. His bloods are printed off by the FY1:

Na+	139 mmol/l
K+	4.1 mmol/l
Urea	20.1 mmol/l (Baseline 4.2 mmol/l)
Creatinine	526 µmol/l (Baseline 86 µmol/l)
CRP	50 mg/l

An urgent KUB ultrasound is arranged, producing the following conclusion:

Severe bilateral hydronephrosis.

His urinalysis is as follows:

Nitrites	+ve
Leucocytes	3+
Blood	2+
Protein	1+

What is the most likely cause of his hydronephrosis?

A. Urinary tract infection
B. Ureteric blood clot
C. Renal tract calculi
D. Malignant infiltration of the ureters
E. Retroperitoneal fibrosis

D. Malignant infiltration of the ureters

Given this patient's history of bladder cancer the most likely answer is malignant infiltration of the ureters. Urothelial cell cancer of the bladder can metastasise to the cells lining the ureters, as well as lymph nodes in the nearby area. This can potentially cause a blockage of urine, thereby causing hydronephrosis.

Renal tract calculi and ureteric blood clot are unlikely to give bilateral hydronephrosis.

Urinary tract infection is an extremely unlikely cause of hydronephrosis but can of course complicate acute hydronephrosis.

Retroperitoneal fibrosis is a potential cause of bilateral hydronephrosis, but is a rare cause and not the most likely in this context.

Question 88 of 183

A 58-year-old man seeks advice for erectile dysfunction in the general medical clinic. He has been noticing that he has been unable to first maintain an erection and then he noticed he had difficulty in initiating an erection. He is married and normal has regular intercourse with his wife. He has not noticed any other medical problems apart from an increase in stress due to additional pressures at his work. He works as an accountant and is concerned about possible redundancies. He has a past medical history of type 2 diabetes diagnosed three years ago, which is well controlled with 1gram metformin MR alone. He also has had gout and a cholecystectomy in the past. He is overweight with a BMI of 28. On examination, there are no abnormalities to the external genitalia and a PR examination is normal. He is keen to try medical treatment. Blood tests are arranged for review in clinic, including HbA1c, renal function, liver function, testosterone and full blood count. A random capillary glucose is 7.3mmol/l.

What else would be an appropriate measure at this stage?

A. Advise weight loss and trial of sildenafil
B. Referral to endocrinology
C. Add gliclazide 40mg daily
D. Referral to cardiology
E. Referral to urology

A. Advise weight loss and trial of sildenafil

Trial of sildenafil is appropriate unless contraindicated for erectile dysfunction, regardless of underlying aetiology

The most appropriate advice would be to advise weight loss and a trial of sildenafil. This patient has erectile dysfunction which may be due to psychological stress, but could also potentially be due to an endocrine cause or due to worsening diabetes. Regardless of the cause, advising weight loss and a trial of sildenafil is recommended. In the presence of evidence of hypogonadism it is appropriate to refer to endocrinology, and if the patient had unstable cardiac disease contraindicating sildenafil, such as unstable angina, then cardiology referral would be an appropriate response. If there was any physical abnormality or history of trauma then a urological referral would be needed. There is insufficient evidence to add gliclazide - this is a big step as it would add an oral hypoglycemic agent - and waiting for the HbA1c would be a more appropriate plan in regards to his diabetes. He may need psychological support in the future.

Question 89 of 183

A 55-year-old lady with end stage renal failure is due to undergo haemodialysis. She receives 3 sessions a week and has been well without complications for these. She has felt unwell the past week with shortness of breath at night and a frothy cough.

On examination, she has crepitations in both lungs and her ankles are swollen. She feels nauseous but has not vomited. Her skin is a sallow colour with pale conjunctiva. There is a systolic murmur present and no sounds are auscultated over her arteriovenous fistula which is sited in the left forearm. She is saturating at 96% in air with a respiratory rate of 21/minute.

Na $^+$	130 mmol/l
K $^+$	5.2 mmol/l
Urea	21.1 mmol/l
Creatinine	420 µmol/l
Hb	96g/dl
ECG	ST elevation in V1-V2, tall T-waves V4-V6

What contraindication is there to her undergoing haemodialysis today?

A. Hyperkalaemia
B. Pulmonary oedema
C. Anaemia
D. Uraemic pericarditis
E. Fistula thrombosis

A. Fistula thrombosis

Arteriovenous fistula with no bruit has thrombosed

This lady has end stage renal failure with signs of decompensation. She is mildly fluid overloaded, hyperkalaemic and at risk of uraemic complications. Despite these, they are not a contraindication to dialysis. Likewise, her anaemia would not prevent her receiving dialysis. The lack of an audible bruit suggests her fistula has thrombosed and she therefore would not be able to undergo dialysis until a new fistula site is created.

Question 90 of 183

A 79-year-old man presented to his GP with bilateral lower limb swelling which had developed gradually over the past month. He also described back pain and widespread joint pains.

On examination, he had macroglossia. His pulse was 90 beats per minute and his blood pressure was 160/95 mmHg. Urinalysis showed protein 4+ and blood 1+. His blood results are shown below.

Hb	81 g/L	Male: (135-180)Female: (115 - 160)
Platelets	350 * 10 /L ^{9}	(150 - 400)
WBC	9.6 * 10 /L ^{9}	(4.0 - 11.0)
Na $^+$	140 mmol/L	(135 - 145)
K $^+$	5.2 mmol/L	(3.5 - 5.0)
Bicarbonate	19 mmol/L	(22 - 29)
Urea	13 mmol/L	(2.0 - 7.0)
Creatinine	196 µmol/L	(55 - 120)
Bilirubin	8 µmol/L	(3 - 17)
ALP	385 u/L	(30 - 100)
ALT	21 u/L	(3 - 40)
γGT	35 u/L	(8 - 60)
Albumin	21 g/L	(35 - 50)
Calcium	2.8 mmol/L	(2.1-2.6)
Phosphate	1.7 mmol/L	(0.8-1.4)

He was referred to a nephrologist who organised a renal biopsy. This demonstrated nodular glomerulosclerosis with amorphous hyaline deposits in the glomeruli. Congo red stain was positive.

What is the most likely underlying disorder?

A. Rheumatoid arthritis
B. Hepatitis C
C. Ankylosing spondylitis
D. Myeloma
E. Cryoglobulinaemia

D. Myeloma

Amyloidosis can cause nephrotic syndrome and the majority of cases are AL type (secondary to plasma cell dyscrasias)

This patient presents with nephrotic syndrome and the renal biopsy findings are characteristic of amyloidosis.

Plasma cell dyscrasias (e.g. myeloma) are the commonest cause of amyloidosis in the UK and lead to primary (AL) amyloidosis. In this case, the back pain, macroglossia, anaemia and hypercalcaemia all point towards myeloma.

Secondary (AA) amyloidosis can be related to chronic inflammatory conditions including rheumatoid arthritis and ankylosing spondylitis, but the incidence has decreased significantly with improvements in treatments for inflammatory arthritides.

Hepatitis C infection and cryoglobulinaemia can cause nephrotic syndrome but are typically associated with membranoproliferative glomerulonephritis.

Question 91 of 183

A 75-year-old retired plumber presents to the emergency department with recurrent urinary tract infections. He has been treated by his GP four times in the last six months with this complaint, with trimethoprim twice and a further two courses of amoxicillin. He is confused on this presentation and is will not comply with a full neurological examination. His past medical history includes a stroke and a known gastric ulcer. An ultrasound of his renal tract did not reveal any structural defects of hydronephrosis. His son reveals that he is under investigation for a possible haematological malignancy, and had a bone marrow biopsy two days ago. On examination, his heart rate is 115/min, respiratory rate 18/min, blood pressure 134/89mmHg. His respiratory, cardiovascular and abdominal examinations are all unremarkable, other than a soft ejection systolic murmur. He has a tender prostate. His blood reveal:

Hb	12.7 g/dl
Platelets	80 * 10 /l9
WBC	17.1 * 10 /l9
Na +	141 mmol/l
K +	3.5 mmol/l
Creatinine	77 µmol/l
CRP	99 mg/l
PSA	6 ng/dl

Given the likely diagnosis, what is the recommended treatment course?

A. No treatment required
B. One week course of co-amoxiclav
C. Tamsulosin
D. Finasteride
E. Four week course of ciprofloxacin

E. Four week course of ciprofloxacin

The correct answer is a four week course of ciprofloxacin. This patient presents with features suggestive of chronic bacterial prostatitis, with recurrent urinary tract infections, tender prostate on examination, and elevated PSA. The recommended first-line treatment in the UK for chronic bacterial prostatitis is a fluoroquinolone antibiotic, specifically ciprofloxacin, for a duration of 4 weeks. This extended duration is necessary because antibiotics penetrate the prostate poorly, and a longer course is required to effectively treat the infection.

No treatment required would be inappropriate as this patient has clear signs of infection with elevated WBC and CRP, along with symptoms requiring intervention. Leaving this untreated could lead to serious complications, particularly given his age and comorbidities.

One week course of co-amoxiclav is incorrect for two reasons. Firstly, co-amoxiclav does not penetrate the prostate gland effectively. Secondly, a one-week course would be insufficient duration for treating prostatitis, even if using an appropriate antibiotic.

Tamsulosin, an alpha-blocker, would not be appropriate as primary treatment. While it might help with urinary symptoms associated with benign prostatic hyperplasia, it does not address the underlying infection. It could be considered as an adjunctive treatment but is not the primary intervention needed.

Finasteride, a 5-alpha reductase inhibitor, is used in the management of benign prostatic hyperplasia to reduce prostate size over time. Like tamsulosin, it does not address the acute infection and would not be appropriate as primary treatment in this scenario.

The patient's recent bone marrow biopsy and thrombocytopenia (platelets 80 10^9/l) should also be noted, as these factors might influence the choice of invasive investigations, but they do not contraindicate the use of ciprofloxacin for treating the prostatitis.

Question 92 of 183

A 55-year-old known rheumatoid arthritis patient is admitted via the emergency department with sudden onset severe right upper quadrant pain. She has vomited several times and felt hot and feverish. She has had no diarrhoea. She has been a bit tired over the last few months and recently saw her GP for swelling of her ankles. He prescribed her a short course of furosemide but this has now finished after there was no improvement. She has a past medical history of hypertension. Her regular medication is penicillamine and ramipril. On examination, she is very tender in the right upper quadrant with voluntary guarding. There is no palpable hepatosplenomegaly. She has pitting oedema to the knee. She is has a low-grade fever of 37.5. Her urine dip shows 3+ of protein and 1+ RBCs.

Her blood tests show

Hb	126 g/l
Platelets	458 * 10 /l9
WBC	9.7 * 10 /l9
Neutrophils	7.7 * 10 /l9
Na +	139 mmol/l
K +	4.9 mmol/l
Urea	25 mmol/l
Creatinine	300 µmol/l
CRP	12 mg/l
Bilirubin	20 µmol/l
ALP	189 u/l
ALT	110 u/l
Albumin	26 g/l

What is the most likely cause of her right upper quadrant pain?

A. Biliary colic
B. Acute cholecystitis
C. Acute portal vein thrombosis
D. Adult polycystic kidney disease
E. Acute viral hepatitis

C. Acute portal vein thrombosis

This lady has an acute portal vein thrombosis after developing nephrotic syndrome due to her penicillamine treatment for rheumatoid arthritis. Acute portal vein thrombosis often presents with right upper quadrant pain, vomiting and fever. It is unlikely to have any features of portal hypertension and the LFTs are often only mildly deranged. The history of ankle swelling combined with proteinuria, hypoalbuminaemia and abnormal renal function should suggest proteinuria in this patient. Biliary colic is the main alternative here but the presence of renal impairment and the clinical history should make you think of nephrotic syndrome.

Question 93 of 183

A 38-year-old known ulcerative colitis (UC) patient presents to the emergency department with excruciating sudden onset left sided loin to groin pain. The patient has no other medical problems. He had a panproctocolectomy and ileostomy 5 years ago after he suffered a colitis flare which did not respond to medical therapy. On examination he is in obvious pain and his urine dip shows 3+ RBCs. He is given diclofenac and 5mg IV morphine and his pain settles. His observations after he is pain-free are normal.

His blood tests are:

Hb	145 g/l
Platelets	455 * 10 /l9
WBC	11.9 * 10 /l9
Na +	136 mmol/l
K +	4.5 mmol/l
Urea	8.8 mmol/l
Creatinine	99 µmol/l
Calcium (adj)	2.48 mmol/L
CRP	<10 mg/L

He undergoes a KUB X-ray which shows a left sided stone in the ureter.

What is the likely type of renal stone?

A. Urate
B. Magnesium pyrophosphate
C. Xanthine
D. Calcium oxalate
E. Cystine

D. Calcium oxalate

This patient has had a bowel resection for UC, both of these conditions are risk factors for developing oxalate stones. The stone is radio-opaque which excludes urate and xanthine stones.

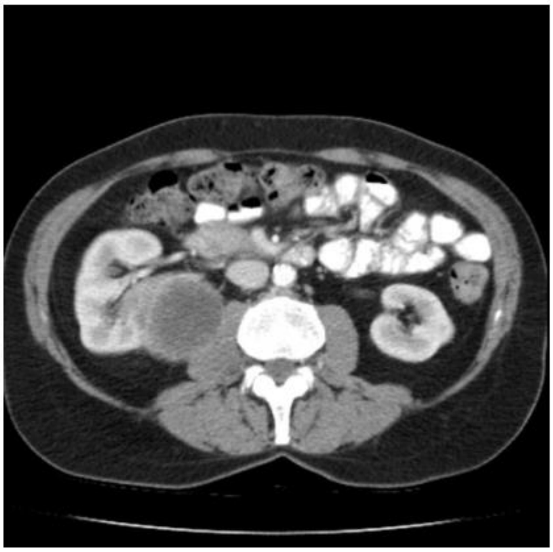
Magnesium pyrophosphate or 'Staghorn calculi' involve the renal pelvis and extend into at least 2 calyces, the presence of the stone in the ureter excludes this.

Question 94 of 183

A 65-year-old woman is investigated for intermittent visible haematuria. Her symptoms have been present for around 6 weeks and are associated with a dull ache in the right loin area. Her past medical history includes depression, hypertension and lower back pain. She has no history of urinary problems other than recurrent bouts of 'cystitis'. Blood tests show the following:

Hb	12.2 g/dl	Na +	142 mmol/l
Platelets	389 * 10 /l 9	K +	3.9 mmol/l
WBC	8.2 * 10 /l 9	Urea	6.5 mmol/l
Creatinine	90 µmol/l		
CRP	14 mg/l		

A CT is requested:



What is the most likely diagnosis?

A. Chronic pyelonephritis with abscess formation
B. Wilm's tumour
C. Renal cyst
D. Autosomal dominant polycystic kidney disease
E. Renal cell cancer

A. Renal cell cancer

The CT scan shows an heterogeneously enhancing mass at the upper pole of the right kidney. The mass is not cystic on imaging nor is it consistent with an abscess. A renal abscess would also normally be associated with more markers of systemic infection.

Wilm's tumours are almost exclusively seen in childhood.

Question 95 of 183

A 45-year-old man presents to the Emergency Department complaining of loin pain and passing blood in his urine. This is not the first time he has had similar symptoms but they are generally not as severe. The pain is concentrated around the left loin region and is described as 'dull' and 6/10 in terms of severity. He is generally fit and well but is currently awaiting ambulatory blood pressure monitoring by his GP as he had a borderline blood pressure at a recent medical.

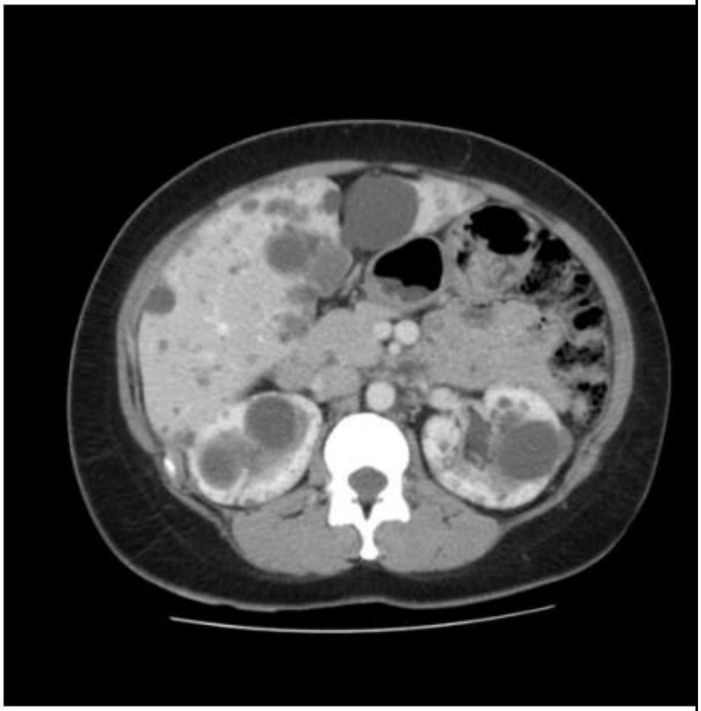
He has recently been on holiday in Greece. He works as a car mechanic and drinks around 30 units of alcohol per week.

Blood tests in the Emergency Department show the following:

Hb	13.9 g/dl	Na +	141 mmol/l

Platelets	286 * 10 /l 9	K +	4.8 mmol/l
WBC	6.9 * 10 /l 9	Urea	6.9 mmol/l
Creatinine	109 µmol/l		
CRP	14 mg/l		

A presumed diagnosis of renal colic is made and a CT requested:



What is the most likely underlying diagnosis?

A. Metastatic renal cell cancer
B. Pseudomyxoma peritonei
C. Autosomal dominant polycystic kidney disease
D. Hydatid disease
E. Medullary cystic disease

C. Autosomal dominant polycystic kidney disease

The presence of both renal and hepatic cysts on the CT make the diagnosis relatively straight forward. Note also the raised creatinine and borderline blood pressure, both clues to underlying autosomal dominant polycystic kidney disease.

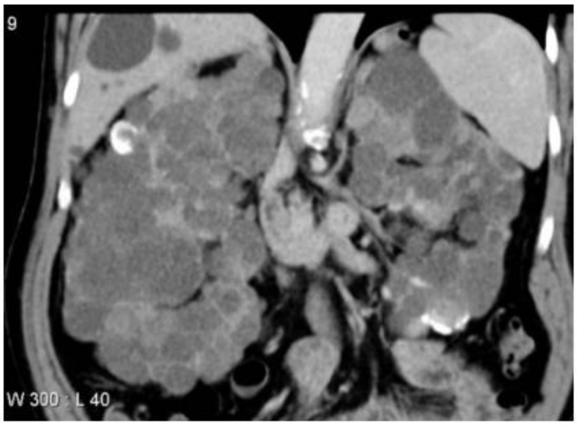
Question 96 of 183

A 45-year-old man is presents to the Emergency Department feeling sick, complaining of abdominal pain, pruritus and reduced urine output. Initial bloods show the following:

Na +	142 mmol/l
K +	5.8 mmol/l
Urea	31.3 mmol/l
Creatinine	450 µmol/l

Urine dipstick: leuc +, protein +, blood+++

His past medical history includes sensorineural hearing loss and recent treatment for *Chlamydia*. An abdominal ultrasound is quickly performed with a subsequent CT to confirm the findings:



What is the most likely cause of the renal failure?

A. Acute pyelonephritis
B. Autosomal dominant polycystic kidney disease
C. Metastatic renal cell cancer
D. Alport's syndrome
E. Nephrolithiasis with associated hydronephrosis

B. Autosomal dominant polycystic kidney disease

The coronal CT of the abdomen demonstrates both kidneys to be markedly enlarged by innumerable cysts ranging in size from a few millimeters to multiple centimeters with cysts also present in the liver. These features are consistent with a diagnosis of autosomal dominant polycystic kidney disease.

Whilst Alport's syndrome is associated with hearing loss it does not cause renal cysts.

Question 97 of 183

A 37-year-old patient presents to the Emergency Department with worsening swelling of his legs over the past few weeks. He has never had this problem before. He does not feel short of breath and has no chest pain. His past medical history includes asthma, hepatitis C and depression.

The patient appears to have sunken cheeks and a number of depressions in his skin across his upper arms and chest. On auscultation his chest is clear, heart sounds I+II are present and there are no added sounds. His abdomen is soft and non-tender. He has pitting oedema to the knees bilaterally.

Observations are as follows: temperature 36.3ºC, blood pressure 179/111 mmHg, heart rate 89/min, respiratory rate 16/min, saturations 97% on air

Initial investigations are as follows:

Hb	120 g/l
Platelets	170 * 10 /l9
WBC	9.2 * 10 /l9
Neuts	4.0 * 10 /l9
Na +	144 mmol/l
K +	4.9 mmol/l
Urea	12.6 mmol/l
Creatinine	166 µmol/l
Adjusted calcium	2.35 mmol/l
Albumin	26 g/l

Urine: blood ++, nitrites -ve, leucocytes -ve, protein +++

What is the single most likely underlying diagnosis?

A. Diffuse proliferative glomerulonephritis
B. Focal segmental glomerulosclerosis
C. IgA nephropathy
D. Mesangiocapillary glomerulonephritis
E. Renal amyloid

D. Mesangiocapillary glomerulonephritis

This young patient has presented with nephrotic syndrome and hypertension on a background of Hepatitis C.

Mesangiocapillary glomerulonephritis is associated with Hepatitis C, which makes it the most likely diagnosis in this patient. The sunken cheeks and dimples in the skin are suggestive of lipodystrophy which is seen in mesangiocapillary glomerulonephritis Type II.

Focal segmental glomerulosclerosis is more commonly seen in patients with HIV.

Question 98 of 183

A 60 year female with end-stage renal failure is referred to the medical assessment unit with painful legs. She has a past history of atrial fibrillation, rheumatoid arthritis and osteoporosis. On examination she has 3 discrete, painful, necrotic skin lesions on her left calf. There is minimal surrounding erythema and her foot appears well-perfused. Peripheral pulses are present.

Blood tests reveal:

Adjusted calcium	2.62 mmol/l
Parathyroid hormone	47 pmol/l

Which of her regular medications is contributing to her acute presentation?

A. Sevelamer
B. Methotrexate
C. Hydroxychloroquine
D. Warfarin
E. Ferrous fumarate

D. Warfarin

The correct answer is **Warfarin**. This patient's presentation is suggestive of warfarin-induced skin necrosis, a rare but serious side effect of warfarin therapy. It usually occurs within the first few days of treatment, often in patients with protein C or S deficiency. The necrotic lesions typically appear on areas with abundant subcutaneous fat such as breasts, thighs and buttocks although they can occur anywhere including the calf as seen in this patient.

Sevelamer is an incorrect option. Sevelamer is a phosphate binder used in patients with chronic kidney disease to prevent hyperphosphatemia. It does not cause skin necrosis. Its common side effects include gastrointestinal symptoms like nausea, vomiting and constipation.

Methotrexate is also an incorrect option. Methotrexate is an immunosuppressant drug used in the management of rheumatoid arthritis among other conditions. While it can cause various cutaneous reactions, it does not cause painful necrotic skin lesions as seen in this patient.

The choice of **Hydroxychloroquine** is incorrect too. Hydroxychloroquine is an antimalarial drug also used in the treatment of autoimmune diseases such as rheumatoid arthritis. Cutaneous side effects associated with hydroxychloroquine are diverse but do not typically present as discrete necrotic lesions without significant surrounding inflammation.

Finally, **Ferrous fumarate** is not the correct answer either. Ferrous fumarate is an iron supplement commonly prescribed to patients with chronic kidney disease who are often anaemic due to reduced erythropoietin production by damaged kidneys. It does not contribute to skin necrosis; its common side effects are gastrointestinal like constipation and black stools.

Question 99 of 183

A 45-year-old female presents to the hyperacute stroke unit with expressive dysphasia and mild right sided upper limb weakness without sensory disturbance seven hours after symptom onset. Although she is outside the thrombolysis window, a hyperacute CT head demonstrates multiple small infarcts in the left middle cerebral artery territory while a simultaneous CT angiogram of her extra and intracranial vessels was reported by the radiologist as a string of beads appearance.

What is the most appropriate next investigation to request to demonstrate the underlying cause of this young females strokes?

A. Factor V Leiden
B. CT angiogram renal arteries
C. Lupus anticoagulant
D. MRI head with contrast
E. MR venogram

B. CT angiogram renal arteries

Young patients under 55 years with acute ischaemic stroke should alert you to an underlying cause beyond arterial atherosclerotic or cardioembolic disease. In this case, the CT angiography findings classically describe a patient with fibromuscular dysplasia (FMD), a non-atherosclerotic, noninflammatory condition producing segmental stenoses in all vascular beds. Pathogenesis remains unknown but a strong genetic predisposition is known to FMD while hormonal influences are thought to contribute, with significant numbers of females within child-bearing age being affected. Between 35 to 60% of FMD patients have carotid artery involvement , hence the frequent presentation of cerebral ischaemia such as transient ischaemic attacks, strokes, headache and tinnitus .

However, the most commonly affected vasculature in FMD is the renal arteries, implicated in up to 70% of FMD patients . As a result of reduced renal perfusion secondary to renal artery stenoses and consequent activation of the renin-angiotensin-aldosterone axis, FMD patients frequently report preceding years of severe or refractory hypertension, often taking multiple antihypertensive agents. While digital subtraction angiography is the investigation of choice, noninvasive modalities such as CT and MR angiography are more commonly used.

Question 100 of 183

A 56-year-old male presents with oliguria, fever and proteinuria eight days after renal transplant. On examination, his abdomen is soft and non-tender, the transplant scar has a mild serous ooze but otherwise clean. A urine dip demonstrates protein 4+ blood 1+, urine output over the past 3 hours have been 27, 20 and 12 ml/ hour respectively. His blood tests are as follows (results from 24 hours ago in brackets)

Hb	99	(98) g/dl
Platelets	190	(185) * 10 /l9
WBC	3.6	(3.6) * 10 /l9
Na +	139	(136) mmol/l
K +	4.9	(4.6) mmol/l
Urea	19	(7.8) mmol/l
Creatinine	225	(117) µmol/l

A renal ultrasound demonstrates a normal sized transplanted kidney, no ureteric leak and no hydronephrosis. His tacrolimus levels are within normal range. An urgent renal biopsy of the transplanted kidney demonstrates significant lymphocytic, in particular, mononuclear cell infiltration with no clonal populations, EBV antigen negative and no light chains. His CMV titre is negative, his tacrolimus level is therapeutic.

What is the most appropriate immediate management?

A. IV methylprednisolone
B. Stop tacrolimus
C. Increase tacrolimus
D. Percutaneous nephrostomy insertion
E. Start ciclosporin

A. IV methylprednisolone

The key differential diagnoses are between acute allograft rejection, which requires aggressive increases in immunosuppression, and post-transplant lymphoproliferative disorder, which requires a reduction in immunosuppression with possible chemotherapy. In short, PTLD frequently demonstrates clonal populations of T or B cells in significant lymphoid disruption of renal architecture, EBV antigen present in many cells with significant immunoglobulin light chain expression. This is not the case in this patient, who demonstrates clinical and histological evidence of allograft rejection. The initial treatment would involve pulsed intravenous methylprednisolone before changing doses of maintenance immunosuppression such as tacrolimus after at least 3 days of steroids. There is no evidence of a post-renal obstruction and hence percutaneous nephrostomy is not indicated. There is no acute indication for starting a second immunosuppressant.

Question 1 of 84

A 71-year-old patient with end-stage renal failure secondary to adult polycystic kidney disease presents with new painful necrotic skin lesions. Her general practitioner has referred her to hospital as they have failed to improve with oral antibiotics.

On arrival she is afebrile. A bruit and thrill were noted over a left brachiocephalic fistula. She has an irregular 8cm x 4 cm, punched-out ulcer on the medial aspect of her left calf just below her knee. The centre of the ulcer is black and necrotic. There is minimal surrounding erythema.

Bloods results revealed:

White cell count	10.0 * 109/l
C-reactive protein (CRP)	17 mg/dL

Which investigation will confirm the diagnosis?

A. Skin swab
B. Parathyroid hormone (PTH)
C. Skin biopsy
D. Anti-neutrophil cytoplasmic antibody (ANCA)
E. Computer tomography angiogram of left leg

C. Skin biopsy

The diagnosis of calciphylaxis is confirmed on skin biopsy. PTH is sensitive but not specific for calciphylaxis. An arterial ulcer would be an important differential but the distribution (upper calf) makes this less likely. Taking a skin swab for bacterial culture is important as there may be superimposed infection but it does not confirm the underlying diagnosis. Vasculitis is unlikely, especially given the nature of her underlying kidney disease.

Question 2 of 84

A 42-year-old woman of Afro-Carribean ethnicity is admitted to hospital after a two-week history of non-specific fatigue and worsening lower limb swelling. She was diagnosed with systemic lupus erythematosus 6 years ago and over the 6 months, her creatinine has been slowly rising from 120 µmol/l to 260 µmol/l.

On examination, her blood pressure is 180/118 mmHg, she has significant swelling of ankles and feet, with periorbital oedema. A urine dip demonstrated 4+ protein and a 24 hour urine collection collected 4.6g protein. She undergoes a renal biopsy under the renal physicians, demonstrating membranous lupus nephritis.

In addition to angiotensin-converting enzyme inhibitors, statins and anti-hypertensives, what is the most appropriate immediate treatment?

A. Monitor
B. PO prednisolone alone
C. IV methyprednisolone alone
D. IV methylprednisolone and IV cyclophosphamide
E. Plasmaphoresis

D. IV methylprednisolone and IV cyclophosphamide

Abnormal renal function is extremely common in patients with SLE with up to 75% of all patients displaying either haematuria or proteinuria on a routine urine dip. A classification for lupus nephritis was produced in 2004, with 6 classes of possible overlapping histological findings on renal biopsy . In the case of this patient, renal biopsy has demonstrated membranous lupus nephritis, when glomerular walls are thickened and significant subepithelial immune deposits are seen, classified as class V lupus nephritis.

The treatment of lupus nephriits is guided by the class of renal histology. While more focal and mild lesions can be treated less aggressively with oral steroids alone. However, nephrotic syndrome and advanced diffuse severe lupus nephritis, as in the case of this patient, warrants early and aggressive immunosuppresion, involving both intravenous methylprednisolone and intravenous cyclophosphamide as endorsed by the KDIGO (Kidney Disease

Improving Global Outcomes) guidelines . There is no role for plasmaphoresis in lupus nephritis . Treatment must be started promptly with appropriate induction followed by maintenance therapy to ensure best long-term prognosis. Monitoring alone is not appropriate.

Question 3 of 84

A 32-year-old man attended his GP feeling generally unwell with headaches and nasal congestion. These symptoms had been ongoing for 4 weeks and did not seem to be responding to over-thecounter decongestants and flu remedies. He also had recurrent nose bleeds and had coughed up fresh blood on two or three occasions. On further questioning, he also had some night sweats. He had otherwise been well with no past medical history and took no regular medications.

His GP was concerned about his symptoms and took some blood tests:

Haemoglobin	97 g/L	Sodium	138 mmol/L
Platelets	169 x10^9/L	Potassium	5.8 mmol/L
White cell count	10.0 x10^9/L	Urea	13.5 mmol/L
Neutrophils	5.5 x10^9/L	Creatinine	367 micromol/L
Eosinophils	0.3 x10^9/L	Albumin	32 g/L
ESR	19 mm/h	Bilirubin	8 micromol/L
CRP	24 mg/L	ALT	27 iu/L
Alkaline Phosphatase	35 iu/L		

On receiving these results the GP admitted the patient to hospital to investigate reasons for the raised creatinine. Additional tests showed:

Urine dip	protein +++ and blood ++
Urinary protein creatinine ratio (PCR)	224

Chest X-ray multiple, well demarcated and round lesions in both lung fields

c-ANCA	positive with high PR3 titres
p-ANCA	negative
Anti-glomerular basement membrane (Anti-GBM) antibodies	negative
Complement levels	normal

Given the most likely diagnosis, what type of glomerulonephritis (GN) would you expect to see on a renal biopsy?

A. Membranous GN
B. Diffuse proliferative GN
C. Focal segmental glomerulosclerosis
D. Rapidly progressive GN
E. Membranoproliferative GN

D. Rapidly progressive GN

The clinical scenario is that of a vasculitic process that involves the lungs, nasal mucosa and kidneys. The history should make you think of both Goodpasture's disease and of granulomatosis with polyangiitis (GPA) - formerly known as Wegener's granulomatosis.

The key to differentiating them is in the immunological tests. Negative Anti-GBM antibodies and a positive c-ANCA will point towards GPA. Regardless, both diagnoses should show the same type of glomerulonephritis on renal biopsy. That is rapidly progressive GN.

Low complement levels are seen in diffuse proliferative GN and membranoproliferative GN so these can be excluded in this scenario.

Question 4 of 84

A 67-year-old man is admitted electively for an inguinal hernia repair. He has a history of hypothyroidism, aortitis and hypertension. His regular medication consists of levothyroxine 75 micrograms daily, mycophenolate mofetil 1 gram twice daily, prednisolone 5mg daily maintenance, ramipril 5mg daily and amlodipine 5mg daily. He has no known allergies. His operation went well with no intra-operative complications.

The next morning his bloods show:

Sodium	139 mmol/L
Potassium	5.6 mmol/L
Urea	15.5 mmol/L
Creatinine	342 micromol/L

His pre-operative values were:

Sodium	137 mmol/L
Potassium	4.4 mmol/L
Urea	6.2 mmol/L
Creatinine	121 micromol/L

When you review the observations he has had an adequate blood pressure throughout the operation and since. He appears euvolaemic clinically with adequate urine output. He had suspended his mycophenolate therapy two weeks prior to the operation as instructed by his consultant in rheumatology. Otherwise, his medications were prescribed as above with some additional analgesia including tramadol and ibuprofen as required. You decide to suspend his nephrotoxic drugs and repeat his bloods the next morning.

According to NICE guidelines on the management of acute kidney injury (AKI), which of the following should trigger a referral to nephrology?

A. AKI on a background of chronic kidney disease (CKD) stage 2 or more
B. AKI in patients over the age of 65 years
C. AKI in a patient with known vasculitis
D. AKI caused by nephrotoxic drugs, eg, non steroidal anti inflammatory drugs
E. AKI caused by hypovolaemia

C. AKI in a patient with known vasculitis

NICE guidelines are useful for your day-to-day management of patients with common problems but they are also very useful tools for revision for MRCP part two. This question relates to the management of AKI in guideline 169 (link below).

Question 5 of 84

You are seeing a 78 year old man in renal outpatient clinic with known CKD4. He is feeling much better than he did last time he saw you in clinic and only occasionally feels slightly tired, a problem which is only occasionally limiting. You saw him three months ago with similar problems and had started him on iron replacement therapy (ferrous fumarate 210mg three times daily). His bloods at clinic three months ago and today are shown below:

Three months ago:

Hb	77 g/l
Platelets	178 * 10 /l9
WBC	3.2 * 10 /l9
MCV	71 fl
Ferritin	78 ng/ml
Transferrin saturations	12%
B12	451 pg/ml
Folate	4.8 nmol/nl
TSH	2.1 mIU/L

Today:

Hb	101 g/l
Platelets	172 * 10 /l9
WBC	4.5 * 10 /l9
MCV	78 fl
Ferritin	621 ng/ml
Transferrin saturations	21%
B12	721 pg/ml
Folate	3.8 nmol/nl
TSH	2 mIU/L

What would you do next?

A. Increase his ferrous fumarate to 210mg four times daily
B. Decrease his ferrous fumarate to 210mg twice daily
C. Do not change any of his medications
D. Transfuse him 1 unit of packed red cells
E. Start an ESA (erythrocyte colony stimulating agent)

B. Decrease his ferrous fumarate to 210mg twice daily

This question requires some knowledge of management of anaemia in patients with chronic kidney disease. The key things that one needs to know to answer this question revolve around the target haemoglobin of patients in this group and how to manage a patient's iron status.

One should typically aim to keep the haemoglobin of these patients between 100 and 120 g/L

One should also be aware of the patient's iron status, and NICE guidelines recommend these tests (in order of preference) for determining the patient's iron status.

1) % hypochromic red cells (requires analysis within 6 hours) a value of >6% indicates iron deficiency

2) Reticulocyte Hb content where < 29pg is diagnostic of iron deficiency anaemia

3) A combination of transferrin saturations (<20%) and ferritin (<100 micrograms/litre)

There is a significant risk of over-replacement of iron in these patients, and the NICE guidelines suggest that a patient's ferritin should be kept below 800 ng/ml, and one should consider changing the patient's iron replacement therapy in order to prevent this when their ferritin is higher than 500 ng/ml.

Thus this patient should not be transfused, nor an erythrocyte colony stimulating agent started, as his symptoms are mild and his Hb is in the target range.

The patient should not have his iron increased as this would further increase his ferritin resulting in iron overload.

And we should not leave his medications as they are as his ferritin has passed the 500 ng/ml marker.

Thus decreasing his iron is the only available option.

Question 6 of 84

A 52-year-old man is referred to the Emergency Department by his General Practitioner with the following renal profile:

Na +	140 mmol/l
K +	3.4 mmol/l
Urea	25.2 mmol/l
Creatinine	380 µmol/l

His past medical history includes neurofibromatosis type 1, hypertension and type 2 diabetes mellitus. While the hypertension and diabetes had previously been controlled with diet alone, this was unsuccessful, and the following medicines were started two weeks ago: amlodipine, irbesartan and metformin.

Urinalysis is negative for blood, protein and nitrites.

What is the likely diagnosis?

A. Cushing's syndrome
B. Bartter's syndrome
C. Polycystic kidney disease
D. Acute glomerulonephritis
E. Renal artery stenosis

A. Renal artery stenosis

A rapid deterioration in renal function soon after starting an angiotensin-2 receptor blocker should raise concerns over possible renal artery stenosis. While the association is more typically described for angiotensin-converting enzyme inhibitors, there is significant risk of deteriorating renal function in patients with renal artery stenosis with the use of angiotensin-2 receptor blockers as well.

There is also an association between renal artery stenosis and neurofibromatosis.

Question 7 of 84

A 64-year-old woman is seen in the low clearance renal clinic. She has a history of chronic kidney disease stage IV secondary to previous bilateral ureteric injury during hysterectomy with nonresolving acute kidney injury.

She is currently feeling well aside from some mild ankle swelling. She is currently taking losartan, alfacalcidol, calcium, cholecalciferol and sodium bicarbonate.

On examination she has a blood pressure of 175/91 mmHg and a heart rate of 92 beats per minute. She has normal heart sounds and her chest is clear. Her JVP is visible at 4cm and she has pitting oedema to the ankles.

Her blood tests are as follows:

Hb	121 g/l	Na +	137 mmol/l
Platelets	241 * 10 /l 9	K +	5 mmol/l
WBC	5 * 10 /l 9	Urea	18 mmol/l
Neuts	2 * 10 /l 9	Creatinine	387 µmol/l
Ca 2+	2.24 mmol/l	Phos	1.1 mmol/l

Which medication should be added to control her hypertension?

A. Amlodipine
B. Bendroflumethiazide
C. Furosemide
D. Metolazone
E. Ramipril

C. Furosemide

This lady has CKD stage IV with as estimated glomerular filtration rate (eGFR) of <30ml/min.

First line treatment for hypertension in this group remains an ACE inhibitor or angiotensin receptor blocker.

However, chronic kidney disease represents one of the special case where the usual NICE hypertension algorithm does not apply for second line management. Diuretics are used to help excrete salt and diminish hypertension. In patients with an eGFR <30ml/min loop diuretics, such as furosemide, are preferred.

Question 8 of 84

A patient presents to the MAU with a worsening dyspnoea. He has a past history only significant for chronic hepatitis B for which he receives regular follow up but is currently on no treatment. Further examination shows widespread oedema with peripheral oedema extending to involve the scrotum and abdominal wall.

Urinalysis

Blood - Protein +++ Glucose + White cells -

Blood results:

Hb	9.8 g/dl
Platelets	540 * 10 /l9
WBC	10.2 * 10 /l9
ESR	72 mm/hr
Na +	133 mmol/l
K +	4.8 mmol/l
Urea	18.9 mmol/l
Creatinine	220 µmol/l
CRP	6mg/l
Chest X-Ray	Normal

What is the most likely diagnosis?

A. Hepato-renal syndrome
B. Membranous glomerulnephritis
C. Rapidly progressive glomerulonephritis
D. Focal segmental glomerulonephritis
E. Minimal change disease

B. Membranous glomerulnephritis

Hepatitis B infection can be associated with a variety of renal diseases, but membranous glomerulonephritis is the most common. Membranous nephropathy is the second commonest glomerulnephritides, with focal segmental glomerulonephritis being the most common. The development of membranous glomerulnephritis is thought to be secondary to immune complexes containing HbsAg being deposited in the glomeruli.

Nephrotic syndrome is associated with a hyper-coaguable state and can be seen with an increased platelet level. There is also a dissociation between CRP and ESR in this condition with ESR being increased in the presence of a normal CRP.

Question 9 of 84

A 39 year-old woman presents with a four month history of abdominal distension, facial swelling and malaise. Her past medical history includes a diagnosis of membranous glomerulonephritis. On examination, heart sounds 1 and 2 were present with no added sounds and the pulse was regular, lung fields were clear, abdominal exam reveals mild ascites and there was bilateral periorbital oedema with pitting oedema of the shins.

Blood tests reveal:

Hb	8.8 g/dL
Platelets	115 * 10 /l9
WBC	4.2 * 10 /l9
Mean corpuscular volume (MCV)	82 fl
Na +	148 mmol/l
K +	3.3 mmol/l
Urea	9.2 mmol/l
Creatinine	176 µmol/l
Bilirubin	14 µmol/l
ALP	91 u/l
ALT	19 u/l
γGT	68 u/l
Albumin	32 g/l
Cholesterol	7.9 mmol/l

Urinalysis reveals protein +++ and a 24-hour protein excretion is 5.2g.

High-dose diuretics and immunosuppression was started. Two weeks later the patient presents with abdominal pain which is 6/10 on the pain scale and worse in her flanks. Her observations were normal apart from a temperature of 38.3ºC. Blood tests are performed and are similar to before except a raised creatinine to 314 µmol/l and a urea of 15.1 mmol/l.

What is the most likely reason this patient has had a deterioration in her renal function?

A. Renal infarction
B. Renal artery stenosis
C. Renal calculus
D. Renal vein thrombosis
E. Acute pyelonephritis

D. Renal vein thrombosis

The original diagnosis is nephrotic syndrome, as demonstrated by the high protein content in her urine, secondary to her membranous glomerulonephritis. Patients who have a diagnosis of nephrotic syndrome are in a hypercoagulable state as they are they lose clotting factors in their urine and are often volume depleted due to the high dose diuretics they are placed on.

The most likely diagnosis here, is therefore renal vein thrombosis, occurring in around 15% of patients with nephrotic syndrome.

Question 10 of 84

A 75 year old woman was admitted to the Emergency Department after collapsing in her General Practitioner's surgery. She had been unwell for five days with profuse diarrhoea up to 10 times per day and regular vomiting. Her husband reported that she had been unable to keep down oral fluids for the past two days. There was no history of foreign travel but she had visited her grandson the previous weekend who had been suffering from a vomiting illness. Past medical history was unremarkable except for a hysterectomy at age 55 years for fibroids and a laparoscopic cholecystectomy for biliary colic at age 65 years. The patient takes no regular medications.

The initial assessment of the patient documented in the Emergency Department is detailed below. Airway: patient's own

Breathing: respiration shallow but no overt respiratory distress; respiratory rate 24 / min; oxygen saturations 100 % (15 L O2); chest clear and resonant; trachea central

Circulation: very dry mucous membranes; capillary refill time 7 seconds; blood pressure 70/30 mmHg; heart rate 130 / minute regular

Disability: GCS 12; temperature 35.9 oCelsius, pupils equal and reactive to light; spontaneous movements of all limbs; plantars down going bilaterally

Exposure: abdomen soft and generally mildly tender; nil other abnormality Results from an arterial blood sample (15 L O2) were as follows:

pH	7.31
PaCO2	35 mmHg (reference 32-43)
PaO2	162 mmHg (reference 70-100)
Bicarbonate	10.4 mmol / L (reference 20.0-26.0)
Sodium	130 mmol / L
Potassium	3.4 mmol / L
Calcium	2.12 mmol / L (reference 2.20-2.60)
Chloride	105 mmol / L (reference 99-108)
Haemoglobin	12.2 g / dL
Lactate	6.5 mmol / L

During initial assessment, IV access was secured and a 500 mL bolus of 0.9 % saline was given.

Repeat observation after initial fluid showed BP 72/34 mmHg with heart rate 126 / minute. The patient remained drowsy.

What is the most appropriate next fluid prescription for this patient?

A. 1000 mL 0.9 % saline over 60 minutes
B. 500 mL 0.9 % saline over 15 minutes
C. 500 mL Gelofusine over 15 minutes
D. 1000 mL Gelofusine over 60 minutes
E. 100 mL 20 % human albumin solution over 15 minutes

B. 500 mL 0.9 % saline over 15 minutes

The patient is severely dehydrated and is hypotensive and tachycardic with a lactic acidosis. She needs emergency fluid resuscitation as treatment for hypovolaemic shock. NICE guidance on intravenous fluid prescription for adults in hospital recommends an initial fluid bolus of 500 mL of crystalloid (containing 130-154 mmol / L sodium) over 15 minutes.

Reassessment after the initial bolus is recommended with a further bolus of 250-500 mL crystalloid if patient still requires fluid resuscitation. If patient still has signs of shock after 2000 mL of fluid have been given then recommendation is to seek expert help.

Human albumin solution and colloids such as Gelofusine have no place in the NICE guidance on IV fluid resuscitation.

Question 11 of 84

A 68-year-old woman was admitted for an elective coronary angiogram. She had a past medical history of type 2 diabetes mellitus, hypertension and chronic kidney disease. Her blood results are below:

Na +	140 mmol/L	(135 - 145)
K +	5.1 mmol/L	(3.5 - 5.0)
Bicarbonate	21 mmol/L	(22 - 29)
Urea	11.8 mmol/L	(2.0 - 7.0)
Creatinine	194 µmol/L	(55 - 120)

Which of the following measures is most likely to prevent this patient from developing contrastinduced nephropathy?

A. Oral rehydration
B. Stop metformin
C. Haemodialysis following angiography
D. N-acetylcysteine
E. IV 0.9% sodium chloride

E. IV 0.9% sodium chloride

Prevention of contrast-induced nephropathy: volume expansion with 0.9% saline

According to NICE guidelines, intravenous volume expansion should be offered to adults having iodinated contrast agents if a) they are at increased risk of contrast-induced acute kidney injury because of risk factors or b) they have an acute illness. Offer either isotonic sodium bicarbonate or 0.9% sodium chloride.

Metformin is not nephrotoxic but can lead to lactic acidosis in renal impairment (eGFR <30). ACE inhibitors or angiotensin receptor blockers (ARBs) should be stopped however.

There is no evidence that either N-acetylcysteine or early haemodialysis following contrast administration are beneficial.

Question 12 of 84

A 42-year-old man with chronic kidney disease (stage 4) attends the clinic with worsening fatigue. He is awaiting a kidney transplant.

Blood results are as follows:

Hb	84 g/L	Male: (135-180)Female: (115 - 160)
Platelets	162 * 10 /L 9	(150 - 400)
WBC	8.2 * 10 /L 9	(4.0 - 11.0)
Ferritin	84 ng/mL	(20 - 230)
Vitamin B12	420 ng/L	(200 - 900)
Folate	3.5 nmol/L	(> 3.0)
Reticulocytes	0.9 %	(0.5 - 1.5)
Na +	136 mmol/L	(135 - 145)
K +	4.8 mmol/L	(3.5 - 5.0)
Bicarbonate	20 mmol/L	(22 - 29)
Urea	45.2 mmol/L	(2.0 - 7.0)
Creatinine	389 µmol/L	(55 - 120)

What treatment is indicated?

A. Active surveillance
B. Erythropoietic stimulating agent (ESA)
C. Iron
D. Pyridoxine
E. Red cell transfusion

C. Iron

In a patient with suspected anaemia of chronic disease secondary to CKD, iron status should be checked prior to commencing EPO

Iron is correct. The patient has a ferritin < 100 and thus requires iron to ensure adequate replete stores prior to the initiation of an ESA. Evidence has demonstrated better efficacy with IV iron in this cohort of patients, which is reflected in the NICE guidelines.

Active surveillance is incorrect. The patient has symptomatic anaemia which therefore requires treatment making this a less preferable option.

Erythropoietic stimulating agent (ESA) is incorrect. An ESA should not be started in the presence of absolute iron deficiency without also managing the iron deficiency. A ferritin of >100 is required prior to initiation of an ESA.

Pyridoxine is incorrect. Vitamin B6 (pyridoxine) deficiency anaemia should be considered in any patient on haemodialysis who does not respond to standard ESA and iron therapy. The patient in this clinical scenario is not on dialysis, making pyridoxine deficiency a less likely diagnosis.

Red cell transfusion is incorrect. Blood transfusions carry risks that are relevant to patients awaiting a kidney transplant. Through exposure to non-self human leukocyte antigens (HLA), the blood recipient is at risk of sensitisation to HLA through the development of anti-HLA antibodies. This can result in the rejection of the donor's kidney. Thus red cell transfusions should be avoided if at all possible in this cohort. If the patient has worsening anaemia despite adequate ion replacement and an ESA, then of course a transfusion may be deemed necessary.

Question 13 of 84

A 48-year-old man attends the clinic with a debilitating itch. He has a history of chronic kidney disease (CKD) stage 4. He takes regular alfacalcidol.

Blood results are as follows:

Calcium	2.2 mmol/L	(2.1-2.6)
Phosphate	2.4 mmol/L	(0.8-1.4)
Parathyroid hormone (PTH)	18.2 pmol/L	(1.6 - 6.9)

What treatment is indicated?

A. Aluminium-based phosphate binders
B. Calcium-based phosphate binders
C. Cholecalciferol
D. Dietary changes
E. Parathyroidectomy

D. Dietary changes

Initial management of CKD-mineral bone disease: Correct hyperphosphataemia first; start with dietary changes before starting a phosphate binder

Dietary changes is correct. The patient likely has CKD-mineral bone disease with associated hyperphosphataemia. In the first instance, this should be treated with dietary changes before the addition of a phosphate binder.

Aluminium-based phosphate binders is incorrect. In the first instance, a dietary reduction of phosphate should be implemented prior to the initiation of a phosphate binder.

Calcium-based phosphate binders is incorrect. In the first instance, a dietary reduction of phosphate should be implemented prior to the initiation of a phosphate binder.

Cholecalciferol is incorrect. The patient is already taking active vitamin D supplementation (alfacalcidol) so there would be no benefit from the addition of cholecalciferol.

Question 14 of 84

A 54-year-old man with chronic kidney disease (stage 4) attends the clinic with worsening fatigue. He is awaiting a kidney transplant.

Blood results are as follows:

Hb	74 g/L	Male: (135-180)Female: (115 - 160)
Platelets	182 * 10 /L 9	(150 - 400)
WBC	10.2 * 10 /L 9	(4.0 - 11.0)
Ferritin	64 ng/mL	(20 - 230)
Vitamin B12	620 ng/L	(200 - 900)
Folate	3.8 nmol/L	(> 3.0)
Reticulocytes	1.2 %	(0.5 - 1.5)
Na +	136 mmol/L	(135 - 145)
K +	4.8 mmol/L	(3.5 - 5.0)
Bicarbonate	20 mmol/L	(22 - 29)
Urea	41.8 mmol/L	(2.0 - 7.0)
Creatinine	368 µmol/L	(55 - 120)

What treatment is indicated?

A. Active surveillance

B. Erythropoietic stimulating agent (ESA)
C. Iron
D. Pyridoxine
E. Red cell transfusion

C. Iron

Anaemia in CKD: erythropoiesis-stimulating agents should be considered once a patient is iron-replete

Iron is correct. The patient has a ferritin < 100 and thus requires iron to ensure adequate replete stores prior to the initiation of an ESA. Evidence has demonstrated better efficacy with IV iron in this cohort of patients, which is reflected in the NICE guidelines.

Active surveillance is incorrect. The patient has symptomatic anaemia which therefore requires treatment making this a less preferable option.

Erythropoietic stimulating agent (ESA) is incorrect. An ESA should not be started in the presence of absolute iron deficiency without also managing the iron deficiency. A ferritin of >100 is required prior to initiation of an ESA.

Pyridoxine is incorrect. Vitamin B6 (pyridoxine) deficiency anaemia should be considered in any patient on haemodialysis who does not respond to standard ESA and iron therapy. The patient in this clinical scenario is not on dialysis, making pyridoxine deficiency a less likely diagnosis.

Red cell transfusion is incorrect. Blood transfusions carry risks that are relevant to patients awaiting a kidney transplant. Through exposure to non-self human leukocyte antigens (HLA), the blood recipient is at risk of sensitisation to HLA through the development of anti-HLA antibodies. This can result in the rejection of the donor's kidney. Thus red cell transfusions should be avoided if at all possible in this cohort. If the patient has worsening anaemia despite adequate ion replacement and an ESA, then of course a transfusion may be deemed necessary.

Question 15 of 84

A 47-year-old lady presented to the Emergency Department with a three-day history of rapid deterioration. She complained of feeling unwell for the last three days with extreme fatigue and reduced appetite, followed by a gradual but rapid onset of swelling in both her legs. She complained further of changes in her urine over the last few days, stating that it appeared frothy and over the last day noticed the presence of blood in her urine, but she denied the presence of urinary frequency, urgency or dysuria. She complained of feeling short of breath on exertion but not at rest and did not suffer from chest pain. She had a past medical history comprising SLE which was diagnosed 12 years ago and hypertension for which she was prescribed ramipril 10mg OD, azathioprine 50mg OD, methotrexate 20mg once per week and folic acid 5mg once per week.

Examination revealed the presence of an unwell lady with a blood pressure of 166/86 mmHg, heart rate of 102, a temperature of 37.1 Celsius and respiratory rate of 22/min. Examination of her cardiovascular system revealed the presence of gross bilateral pitting oedema in her lower limbs to the level of her mid shins, with a JVP of 3cm and normal heart and breath sounds. Examination of her gastrointestinal system was unremarkable.

Initial investigations reveal the following results. Results from DMARD monitoring bloods 2 months ago are shown in brackets.

Hb	122 g/l (126)
Platelets	242 * 10 /l (248)9
WBC	7.8 * 10 /l (8.2)9
ESR	26 mm/hr
Na +	131 mmol/l (138)
K +	6.2 mmol/l (5.2)
Urea	22.2 mmol/l (6.4)
Creatinine	268 µmol/l (77)
CRP	42 mg/l
Bilirubin	18 µmol/l (18)
ALP	76 u/l (82)
ALT	72 u/l (21)
Total protein	52 g/l (78)
Albumin	18 g/l (39)
Glucose	8.2 mmol/l

Chest x-ray: normal heart size and lung fields ECG: sinus tachycardia 108bpm no acute changes

Urinalysis: blood ++, protein ++++, leuc trace, nit negative, gluc negative, ketone trace

What is the most likely diagnosis?

A. Berger's nephropathy
B. Membraneous glomerulonephritis
C. Focal segmental glomerulosclerosis
D. Rapidly progressive glomerulonephritis
E. Mesangiocapillary glomerulonephritis

D. Rapidly progressive glomerulonephritis

This lady has presented with acute nephrotic syndrome and (presumably) acute severe kidney failure. Options 2,3 and 5 are all associated with SLE and can cause a nephrotic syndrome; indeed membranous glomerulonephritis is the most common cause of nephrotic syndrome in adults. They are all associated with end stage renal failure to some degree, though this tends to be a chronic gradual decline in renal function. RPGN, also known as crescentic glomerulonephritis, also associated with focal segmental glomerulosclerosis, can present with acute nephritic or nephrotic syndrome and is associated with a fulminant renal failure. Early recognition is essential to delay renal further renal damage; this lady will require aggressive immunosuppressive treatment.

Question 16 of 84

A 64-year-old man presents to the endocrinology clinic. He has been seen previously in the clinic and recent investigations have confirmed a diagnosis of panhypopituitarism, He has found to have low thyroid function, low adrenal function and low gonadal function. He has been feeling increasingly tired as well and finds himself easily fatigued. He has a past medical history of prostate cancer, fractured wrist, osteoporosis, gout, type 2 diabetes and high cholesterol. He currently takes metformin, colecalciferol and atorvastatin. He is to be started of hydrocortisone and levothyroxine. He wonders if he needs to have testosterone replaced.

What is the most appropriate plan?

A. Check morning serum testosterone in one month
B. Start testosterone replacement
C. Testosterone replacement is contraindicated
D. Replace testosterone if sexually active
E. Check evening serum testosterone in one month

C. Testosterone replacement is contraindicated

A patient who has an elevated PSA should not have testosterone even if deficient

This is a patient with panhypopituitarism and low serum testosterone. Normally replacement would be indicated due to the history of osteoporosis, regardless of sexual function and problems with erectile dysfunction. The fact that the patient has a history of prostate cancer means that treatment is contraindicated. Other contraindications include a PSA >4ng/ml, male breast cancer, severe sleep apnoea and severe LUTS due to BPH.

Question 17 of 84

A 38 year-old man presented to his GP with weakness and arthralgia. He had a past history of liver cirrhosis secondary to hepatitis C virus infection which was contracted 10 years ago, however he had failed to attend any clinic appointments over the past 18 months.

On examination, his pulse was 85 beats per minute and his blood pressure was 140/80 mmHg. There was a purpuric rash affecting his lower limbs. Urinalysis showed blood 2+ and protein 2+.

Hb	128 g/l
Platelets	577 * 10 /l9
WBC	10.9 * 10 /l9
Na +	137 mmol/l
K +	4.1 mmol/l
Urea	7.3 mmol/l
Creatinine	186 µmol/l

What is the most important diagnostic investigation?

A. Antinuclear antibodies
B. Anti glomerular basement membrane antibodies
C. Anti-neutrophil cytoplasmic antibodies
D. Serum cryoglobulins
E. Antistreptolysin O titre

D. Serum cryoglobulins

The main clues in this case are the history of hepatitis C infection and the onset of a purpuric rash. This should make you think of cryoglobulinaemia, which can cause a type 1 mesangiocapillary glomerulonephritis (most commonly in association with hepatitis C infection).

Question 18 of 84

An 84 year-old lady is referred to the Renal team. She was admitted 3 days previously with a community-acquired pneumonia and was being treated with co-amoxiclav and doxycycline. Over the past 24 hours she has developed a rash and reduced urine output. She was otherwise fit and well apart from mild asthma which was well controlled with a salbutamol inhaler.

On examination, her pulse was 105 beats per minute and her blood pressure was 112/64 mmHg. Urinalysis showed protein 2+ and blood 2+.

Hb	122 g/l
Platelets	468 * 10 /l9
WBC	8.3 * 10 /l9
Eosinophils	2.1 * 10 /l9
Na +	133 mmol/l
K +	5.4 mmol/l
Urea	18.5 mmol/l
Creatinine	440 µmol/l

What is the most likely diagnosis?

A. Focal segmental glomerulosclerosis
B. Post-infectious glomerulonephritis
C. Membranous nephropathy
D. Minimal change disease
E. Acute interstitial nephritis

E. Acute interstitial nephritis

Acute interstitial nephritis (AIN) is an immune-mediated hypersensitivity reaction to an antigen, most commonly drugs or infections. The commonest drugs implicated are antibiotics, NSAIDs, proton pump inhibitors, diuretics and allopurinol.

The clues to the diagnosis of AIN in this case are the presence of a rash and eosinophilia in the context of recent antibiotic administration. Urinary eosinophils may also be seen.

Question 19 of 84

A 75 year-old man presented to his GP with bilateral lower limb swelling which had developed over the past week. He also described having frothy urine. He had a past history of lung cancer for which he was about to begin a course of palliative chemotherapy.

On examination, his pulse was 88 beats per minute and his blood pressure was 150/90 mmHg. Urinalysis showed protein 4+ and blood 1+.

Hb	110 g/l
Platelets	375 * 10 /l9
WBC	4.9 * 10 /l9
Na +	136 mmol/l
K +	4.6 mmol/l
Urea	23.0 mmol/l
Creatinine	420 µmol/l
Serum Albumin	18 g/L
24 hour urine protein	4.5 g (<0.2)

What is the most likely diagnosis?

A. Post-infectious glomerulonephritis
B. Acute interstitial nephritis
C. Focal segmental glomerulosclerosis
D. Minimal change disease
E. Membranous nephropathy

E. Membranous nephropathy

The clue to the diagnosis in this case is the history of lung cancer. Malignancies can cause secondary membranous nephropathy, and the onset of nephrotic syndrome in an elderly patient should raise the possibility of underlying malignancy.

Question 20 of 84

An 18 year-old man presented to his GP with facial puffiness and leg swelling over the past few weeks. He had no past medical history and no family history of note. He took no regular medications.

On examination, his pulse was 90 beats per minute and his blood pressure was 140/80 mmHg. Cardiovascular, respiratory and abdominal examination was unremarkable. Urinalysis showed protein 4+ and blood 1+ but was negative for glucose.

Hb	138 g/l
Platelets	185 * 10 /l9
WBC	6.6 * 10 /l9
Na +	144 mmol/l
K +	4.0 mmol/l
Urea	5.5 mmol/l
Creatinine	78 µmol/l
Serum albumin	20 g/L
24 hour urine protein	5.1 g (<0.2)

Renal biopsy showed podocyte fusion on electron microscopy

What is the most appropriate next step in treatment?

A. Rituximab
B. Prednisolone
C. Ramipril
D. Intravenous immunoglobulin
E. Plasmapheresis

B. Prednisolone

management of minimal change disease would include prednisolone 1mg/kg/day in the first instance. (http://www.kdigo.org/clinicalpracticeguidelines/pdf/KDIGO-GN-Guideline.pdf)

Question 21 of 84

A 50 year-old lady presented with generalised oedema and shortness of breath. Urinalysis revealed nephrotic range proteinuria. A clinical diagnosis of nephrotic syndrome was made.

Which of the following conditions is most commonly associated with nephrotic syndrome?

A. IgA nephropathy
B. Membranous nephropathy
C. Post-infectious glomerulonephritis
D. Acute interstitial nephritis
E. Wegener's granulomatosis

B. Membranous nephropathy

Nephrotic syndrome consists of heavy proteinuria >3 g/day, low serum albumin <25 g/L and oedema. It can also lead to hyperlipidaemia, venous thromboembolism (including renal vein thrombosis) and increased susceptibility to bacterial infections.

There are many causes of nephrotic syndrome.The commonest causes can be remembered by the acronym 'MMF' (Minimal change disease, Membranous nephropathy, and Focal segmental glomerulosclerosis).

Question 22 of 84

A 56-year-old man with long-standing hypertension medicated with enalapril and diuretics presents with right flank pain. Temperature is 37.2°C, blood pressure is 145/95 mmHg.

On examination: Tenderness in right costovertebral angle and bilaterally enlarged kidneys. Urine dip-stick noted micro-haematuria.

Which of the following is the most appropriate next step in diagnosis?

A. Cytological examination of urine
B. Ultrasonography
C. CT scan of the abdomen
D. Intravenous pyelography (IVP)
E. Renal biopsy

B. Ultrasonography

The findings of hypertension with bilaterally enlarged kidneys is suggestive of autosomal dominant (adult) polycystic kidney disease, where hypertension is frequently the initial presenting sign. A positive family history may be noted. Ultrasound of the kidneys is the initial diagnostic procedure of choice as it is very sensitive for detecting cystic formation within the kidneys.

Question 23 of 84

An 80-year-old gentleman presents with gross peripheral oedema and lethargy. Further history reveals a history of dysphagia and a subsequent CT scan reveals oesophageal carcinoma. Despite initial treatment with furosemide and prednisolone, his renal function continues to deteriorate. His blood results are as follows:

Na +	132 mmol/l
K +	5.6 mmol/l
HCO3 -	17 mmol/l
Urea	27.5 mmol/l
Creatinine	352 µmol/l
albumin	19 g/L

He agrees to undergo a renal biopsy.

What do you expect the biopsy to show?

A. IgA nephropathy
B. Membranous glomerulonephritis
C. Normal biopsy
D. Focal segmental glomerulonephritis
E. Rapidly progressive glomerulonephritis

B. Membranous glomerulonephritis

Membranous glomerulonephritis is one of the most common causes of nephrotic syndrome. It typically occurs in adults over the ago of forty and is more common in caucasian males. Secondary membranous glomerulonephritis is often associated with malignancy (especially lung or colon), systemic lupus erythematosus (SLE) and viral hepatitis. Anyone who is found to have membranous glomerulonephritis on biopsy, especially those over 50, should undergo routine age-appropriate cancer screening.

Question 24 of 84

You are the medical registrar on-call. A 70-year-old male presents to the Emergency Department (ED) with clinical findings consistent with critical limb ischaemia. The patient has a background of diabetes and chronic kidney disease (Stage 4). He is admitted under the vascular surgeons who wish to undertake a CT angiogram. They contact you with the following bloods results:

Today	1 month ago	
Na +	141 mmol/l	138 mmol/l
K +	4.7 mmol/l	4.4 mmol/l
Urea	15.8 mmol/l	9.8 mmol/l
Creatinine	241 µmol/l	158 µmol/l

What evidence-based advice should you give to minimise the risk of contrast-induced nephropathy?

A. Oral pre-hydration is superior to intravenous pre-hydration
B. Intravenous fluids should be administered with a diuretic
C. Intravenous N-acetylcysteine is superior to oral N-acetylcysteine
D. Prophylactic haemofiltration should be undertaken post-scan
E. Intravenous pre- and post-hydration should only be undertaken with isotonic fluid

E. Intravenous pre- and post-hydration should only be undertaken with isotonic fluid

The only evidence showing benefit in intravenous pre- and post-hydration is for isotonic fluids.

There is evidence that oral fluids may be as efficacious as intravenous fluids in certain populations (e.g. CKD Stage 1 and 2), however in higher-risk groups this has not been proven and hence intravenous fluids are the the preferred option.

There is no evidence that co-administration of a diuretic improves outcomes, and evidence that some diuretics (e.g. furosemide, mannitol) may cause harm.

There is no evidence to show that intravenous is superior to oral N-acetylcysteine (NAC). Since intravenous NAC is associated with increased frequency of drug reactions, the oral route is preferred if possible.

The evidence suggests no benefit for prophylactic haemodialysis or haemofiltration pre- or post procedure in CKD Stage 3 and Stage 4.

In addition to these measures, it is of course advisable to stop nephrotoxic medications (NSAIDs) and metformin.

Question 25 of 84

A 42-year-old man attends for follow-up in renal clinic. The patient has been under surveillance for autosomal dominant polycystic kidney disease for the previous 10 years. The patient is currently asymptomatic but concerned about an ongoing decline in his renal function as monitored by his general practitioner. He has no other past medical history. The patient took ramipril 5 mg daily and had no allergies.

Clinical examination demonstrated bilateral palpable kidneys with a euvolaemic fluid status. Blood pressure in clinic was documented as 105 / 70 mmHg.

Please see below for serial renal function and ultrasound results over the period of follow-up.

Date	Estimated globular filtration rate mL / min / 1.73 m3
10 years previous	72
8 years previous	68
5 years previous	62
2 years previous	59
Present	41

Date	Increase in total kidney volume relative to previous measurement
10 years previous	n/a
8 years previous	+ 2 %
5 years previous	+ 2 %
2 years previous	+ 7 %
Present	+ 12 %

What is the appropriate management to reduce the rate in decline of the patients renal function?

A. Desmopressin
B. Increase Ramipril dose
C. Pravastatin
D. Octreotide
E. Tolvaptan

E. Tolvaptan

The patient has ADPKD with recent significant deterioration in both renal function and increase in kidney volume. Tolvaptan is a selective vasopressin receptor 2 antagonist and has been shown to reduce the annual rate of kidney growth and reduce the rate of decline of renal function over a three year period. In England and Wales, tolvaptan is recommended for patients with rapidly progressive disease at CKD stages 2-3. There is no current definition of rapidly progressive disease although an increase in total kidney volume of > 5 % per year is a predictor of rapid disease progression.

The patients blood pressure is already well controlled, so increasing ramipril dose would not be expected to yield additional benefit. Pravastatin was shown to slow disease progression in children with ADPKD, but there is no evidence of benefit in adult patients. Somatostatin analogues such as octreotide are being evaluated in the treatment of ADPKD but do not yet have proven benefit. Desmopressin has no role in the treatment of ADPKD.

Question 27 of 84

A 62-year-old man is admitted due a three week history of penile swelling. He has a past medical history of colorectal cancer, obesity and asthma. Limited examination demonstrates global pitting oedema and a mild expiratory wheeze. The FY1 prints off his admission blood tests:

Na+	142 mmol/l
K+	3.6 mmol/l
Urea	6.9 mmol/l (Baseline 4.2 mmol/l)
Creatinine	126 µmol/l (Baseline 84 µmol/l)

What is the most likely diagnosis?

A. Nephrotic syndrome
B. Testicular cancer
C. Malnutrition
D. Mondor's disease
E. Eosinophilic granulomatosis with polyangiitis (Churg-Strauss syndrome)

A. Nephrotic syndrome

Nephrotic syndrome can commonly present in men with penile swelling. This particular symptom will have occurred following oedema of other areas but may be the first noted problem by the patient. The further clue in this question relates to the patient's history of colorectal cancer. A significant cause of nephrotic syndrome in older adults is membranous nephropathy. This can be secondary to a malignancy. Therefore in this patient it would be prudent to perform lower GI endoscopy.

Churg-Strauss syndrome is a triad of late-onset asthma, vasculitis and eosinophilia. It is a multisystem disease, with the symptoms presented dependent on the organs affected; oedema would not be an expected presenting complaint. The degree of renal impairment may also be expected to be greater. His co-existing diagnosis of asthma is a distractor.

Malnutrition may indeed cause gross global oedema. However, this would not be expected in a patient who is overweight in a Western society.

Testicular cancer would not be expected to cause oedema.

Mondor's disease is a thrombophlebitis of superficial veins typically in the breast or anterior chest wall. It may also occur in the penis.

Question 28 of 84

A 46 year-old man with a diagnosis of chronic hepatitis C is reviewed in renal clinic.

He had initially been referred via the hepatology clinic after blood results reveal raised urea and creatinine. A urine sample had revealed haematuria and proteinuria.

A renal biopsy had been arranged at the initial renal outpatients appointment.

The biopsy has revealed increased mesangial matrix and increased mesangial cellularity in the glomerulus.

What is the most likely diagnosis?

A. Goodpasture's syndrome
B. IgA nephropathy
C. Membranoproliferative glomerulonephritis
D. Granulomatosis with polyangiitis
E. Systemic lupus erythematosus

C. Membranoproliferative glomerulonephritis

Membranoproliferative glomerulonephritis (also known as mesangiocapillary glomerulonephritis) is a type of glomerulonephritis caused by deposits in the glomerular mesangium and basement membrane.

Histological features include increased mesangial matrix and increased mesangial cellularity.

It is associated with chronic viral, bacterial and protozoal infections including:

- Viral Hepatitis B and hepatitis C
- Bacterial Endocarditis, infected ventriculoatrial (or jugular) shunt, multiple visceral abscesses, leprosy
- Protozoal Malaria, schistosomiasis

Question 29 of 84

A 36 year male presents with sudden onset, sharp, right sided chest pain and a 4 month history of increasing leg swelling, weight gain and abdominal distension. He has no past medical history of note. On examination he is tachycardic at 105/min with otherwise normal observations. Cardiovascular and respiratory examinations are normal; there is shifting dullness on examination of the abdomen with no masses palpable. There is also bilateral pitting oedema to the groins. Electrocardiogram shows sinus tachycardia. Urine dip shows 3+ protein nil else. Chest X-ray is unremarkable. Blood tests show the following:

Hb	137 g/l	Na +	141 mmol/l	Bilirubin	12 µmol/l
Platelets	275 * 10 /l 9	K +	4.4 mmol/l	ALP	89 u/l
WBC	9.2 * 10 /l 9	Urea	4.3 mmol/l	ALT	33 u/l
Neuts	7.3 * 10 /l 9	Creatinine	86 µmol/l	γGT	47 u/l
Lymphs	1.4 * 10 /l 9	Albumin	20 g/l		

What is the most likely cause of his chest pain?

A. Pulmonary embolism
B. Acute coronary syndrome
C. Aortic dissection
D. Pneumothorax
E. Musculoskeletal chest pain

A. Pulmonary embolism

This patient has signs and biochemisty suggestive of nephrotic syndrome which presents as a triad of oedema, proteinuria and hypoalbuminemia. Patients with nephrotic syndrome are at an increased risk of venous thromboembolism and the nature of the chest pain would support pulmonary embolism as the diagnosis. The underlying mechanism is felt to be a combination of reduced antithrombin III due to glomerular dysfunction, platelet hyperactivity and renal loss of plasmin impairing fibrinolysis.

Chest X-ray rules out pneumothorax and there are no clear risk factors for acute coronary syndrome. The nature of the pain is not suggestive of aortic dissection. Musculoskeletal pain is a possibility but in the presence of the clinical and biochemical findings pulmonary embolism must be ruled out first.

Question 30 of 84

A 49-year-old known rheumatoid arthritis patient is admitted via the emergency department with a one-month history of intermittent left flank pain and bloody urine. She has had no diarrhoea or rectal bleeding. She has been a bit tired over the last few months and recently saw her GP because she was worried her ankles were swollen. She has a past medical history of hypertension. Her regular medication is penicillamine and valsartan. On examination, her abdomen is soft with mild left flank tenderness. There is no palpable hepatosplenomegaly. She has pitting oedema to the knee. Her urine dip shows 3+ of protein and 1+ RBCs.

Her blood tests show

Hb	118 g/l
Platelets	419 * 10 /l9
WBC	4.7 * 10 /l9
Na +	133 mmol/l
K +	4.3 mmol/l
Urea	28 mmol/l
Creatinine	328 µmol/l
CRP	11 mg/l
Bilirubin	17 µmol/l
ALP	89 u/l
ALT	11 u/l
Albumin	23 g/l

What is the most likely cause of her flank pain?

A. Inflammatory bowel disease
B. Renal vein thrombosis
C. Pyelonephritis
D. Adult polycystic kidney disease
E. Renal colic

B. Renal vein thrombosis

This lady has a renal vein thrombosis after developing nephrotic syndrome due to her penicillamine treatment for rheumatoid arthritis. Renal vein thrombosis can be asymptomatic or present with flank pain and frank haematuria. The history of ankle swelling combined with proteinuria, hypoalbuminaemia and abnormal renal function should suggest proteinuria in this patient. Renal colic would be the main differential in this patient but the presence of swollen ankles and low albumin in a rheumatoid patient on penicillamine should point you towards nephrotic syndrome and renal vein thrombosis.

Question 31 of 84

A 58-year-old Caucasian man with a background of type 2 diabetes mellitus presents to the GP with worsening pedal oedema. On further questioning, he reports having previously had weight loss and alteration in his bowel habit over the past six months.

On examination, he is unkempt, and there is pitting oedema to both thighs and sacral oedema, Lungs are clear, the cardiac examination is unremarkable and the JVP is not elevated. Abdominal exam is unremarkable, as is a digital rectal examination. Urine dip shows protein +++ but is otherwise negative.

Hb	78 g/l	Na +	135 mmol/l	Bilirubin	20 µmol/l
Platelets	340 * 10 /l 9	K +	3.7 mmol/l	ALP	14 u/l
WBC	12 * 10 /l 9	Urea	6 mmol/l	ALT	80 u/l
Neuts	3 * 10 /l 9	Creatinine	67 µmol/l	gamma #GT	30 u/l
Lymphs	7 * 10 /l 9	Albumin	18 g/l		
Eosin	0.1 * 10 /l 9	MCV	82 fl		

What is the most likely diagnosis?

A. Alcoholic cirrhosis
B. Hypoalbuminaemia secondary to poor nutritional status
C. Diabetic kidney disease
D. Congestive cardiac failure
E. Membranous glomerulonephritis secondary to colorectal cancer

E. Membranous glomerulonephritis secondary to colorectal cancer

In this question, all the options are potential causes of the clinical presentations.

Diabetic kidney disease can cause fluid overload, and result in pedal oedema, and could explain the proteinuria, but the hypoalbuminaemia would not be accounted for by this.

Congestive cardiac failure can present with fluid overload; oedema can also result from hypo albuminuria, and similarly alcoholic cirrhosis can cause oedema, but these options do not account for the proteinuria.

Given the clinical findings and the history, there is a high suspicion for malignancy, and the diagnosis fo membranous glomerulonephritis secondary to colorectal cancer is the most likely answer.

Question 32 of 84

A 67-year-old man with chronic kidney disease secondary to diabetes mellitus is seen in the clinic. He reports no new symptoms and has good glycaemic control.

His current medications are metformin 1g BD, aspirin 75mg OD, bisoprolol 1.25mg OD On examination, there is mild peripheral oedema and his blood pressure is 135/80mmHg; this has been confirmed by ambulatory blood pressure monitoring.

What is the most appropriate next step?

A. Monitor blood pressure
B. Add atorvastatin 40mg ON PO
C. Add in ramipril 1.25mg OD PO
D. Add furosemide
E. Increase bisoprolol to 2.5mg OD

C. Add in ramipril 1.25mg OD PO

The most appropriate next step in this scenario is to **add in ramipril 1.25mg OD PO**. This is the correct answer because the patient has chronic kidney disease (CKD) secondary to diabetes mellitus and hypertension. In such patients, angiotensin-converting enzyme inhibitors (ACEIs) like ramipril are recommended as first-line treatment for blood pressure control and renal protection, according to UK guidelines. ACEIs have been shown to slow down the progression of CKD in diabetic patients by reducing proteinuria, lowering blood pressure, and providing direct renal vasodilatory effects.

Monitor blood pressure alone would not be sufficient in this case, as the patient's blood pressure is above the target range for someone with CKD and diabetes (ideally below 130/80 mmHg). Additionally, monitoring without intervention may lead to further deterioration of his renal function.

Adding **atorvastatin 40mg ON PO** might be beneficial for this patient's cardiovascular risk profile due to his age, diabetes, and CKD; however, it would not directly address his elevated blood pressure or provide renal protection. Statins are mainly used for lipid-lowering purposes and primary or secondary prevention of cardiovascular events.

Add furosemide is not the best choice in this situation. Furosemide is a loop diuretic that can help reduce peripheral oedema but does not offer significant renal protection compared to ACEIs or angiotensin II receptor blockers (ARBs). Furthermore, furosemide may cause electrolyte imbalances which could exacerbate CKD if not monitored closely.

Lastly, **increase bisoprolol to 2.5mg OD** would help lower the patient's blood pressure but would not provide optimal renal protection compared to adding an ACEI like ramipril. Bisoprolol is a beta-blocker, which can be used in combination with ACEIs or ARBs for blood pressure control, but it does not have the same renoprotective effects.

Question 33 of 84

A 72-year-old lady who has a background of multiple myeloma is being investigated for bilateral carpal tunnel syndrome, worsening shortness of breath and oedema. Her investigation results are as follows:

Hb	105 g/l
Platelets	145 * 10 /l9
WBC	7.2 * 10 /l9
Ca +	2.70 mmol/l
Urea	4.3 mmol/l
Creatinine	150 µmol/l

Urine protein >3.5/day

Chest x-ray shows cardiomegaly with fluid overload

What are her symptoms most likely to be due to?

A. Membranous nephropathy
B. Amyloidosis
C. Heart failure
D. Minimal change disease
E. Treatment side effects

B. Amyloidosis

Amyloidosis may be associated with lymphoproliferative disorders and has clinical features relating to the organs involved heart failure, nephrotic syndrome with proteinuria. Given her background of multiple myeloma, bilateral carpal tunnel syndrome, heart failure and proteinuria, the answer that accounts for all of this is amyloidosis.

Although this lady has heart failure based on her clinical history and chest x-ray findings, it does not explain all of her symptoms.

Membranous nephropathy is less likely to account for this lady's symptoms as it tends to be associated with lymphoma or chronic lymphoid leukaemia rather than multiple myeloma. Minimal change disease is most common in children and renal function is usually preserved. This pattern of findings is also unlikely to be associated with treatment.

Question 34 of 84

A 41-year-old lady was admitted feeling generally unwell with a headache, generalised aches and pains, lethargy and fevers. She had a previous medical history of type one diabetes and end-stage renal failure. She had undergone a simultaneous pancreas-kidney transplant ten months prior to admission with good graft function. Four months previously she had been admitted with neutropaenia which had led to the early cessation of her valganciclovir and a reduction in her immunosuppression. Her medications comprised of tacrolimus, prednisolone, mycophenolate and aspirin. On examination, she appeared pale, lethargic and unwell. She had a mid-line laparotomy scar with a palpable renal transplant in the left iliac fossa. Her temperature was 39.4 degrees Celsius, her pulse was 115 beats per minute and regular, her blood pressure was 102/59 mmHg, her respiratory rate was 22 breaths per minute and her oxygen saturations were 95% on room air.

Investigations:

Haemoglobin	108g/L
White cell count	9.2x10^9 /L
Platelet Count	347x10^9/L
Neutrophil Count	7.6x10^9/L
Lymphocyte count	0.5x10^9/L
Serum sodium	129mmol/L
Serum potassium	5.4mmol/L
Serum urea	7.4mmol/L
Serum creatinine	174micromol/L
Albumin	32g/L
Alkaline phosphatase	204 IU/L
Alanine transaminase	46 IU/L
CRP	101mg/L

Chest X-ray: clear lung fields

What is the most likely diagnosis?

A. Urinary tract infection
B. Hepatitis C infection
C. Cytomegalovirus infection
D. Upper respiratory tract infection
E. BK virus infection

C. Cytomegalovirus infection

This lady has presented with the classical symptoms of cytomegalovirus(CMV) infection which is a cause of increased morbidity and mortality in renal transplant patients. CMV infection normally presents one to four months after renal transplantation or stopping cytomegalovirus prophylaxis (valganciclovir) as in this case. All renal transplant patients and donors will have their CMV status checked prior to transplantation and recipients will receive prophylaxis unless both they and the donor test negative. CMV infection presents with a mononucleosis-like syndrome with fever, myalgia and arthralgia. There is often a leukopaenia, atypical lymphocytosis with a mild rise in transaminases and graft dysfunction. Specific organ involvement can lead to hepatitis, pancreatitis, gastrointestinal ulceration and bleeding, pneumonitis, colitis and meningoencephalitis. The diagnosis is confirmed with CMV polymerase chain reaction (PCR). Treatment for patients with invasive disease is initially IV ganciclovir.

Although immunosuppression increases the risk of bacterial infections there are no symptoms of urinary tract infection in the history, making this diagnosis unlikely. Hepatitis C infection would be unlikely to present in this manner. An upper respiratory tract infection would not explain the deranged liver function tests or deterioration in renal function. BK virus is a polyomavirus which can cause asymptomatic latent infection in renal tissue in immunocompetent individuals. After renal transplantation this virus can reactivate causing fever and graft dysfunction. BK virus infection could account for some of this lady's symptoms, however, cytomegalovirus infection is more likely and fits the presentation better.

Question 35 of 84

A 64-year-old lady was admitted to hospital with haematuria. She had been feeling unwell in the preceding weeks with lethargy, vomiting, weight loss and fevers. A day prior to admission she developed haematuria with reduced urine output. She had no previous medical history but was an ex-smoker. She was fit and active working as a retail shop manager. Examination revealed a pale and lethargic woman with mild peripheral oedema. Her chest was clear, the abdomen was soft and non-tender with no palpable masses.

Investigations:

Urine dip: blood+++, protein++

Haemoglobin	86 g/L
White cell count	8.7 x10^9/L
Platelet Count	201x10^9/L
INR	1.0
Serum sodium	139mmol/L
Serum potassium	6.3mmol/L
Serum urea	34.0mmol/L
Serum creatinine	789 micromol/L
CRP	32
Antinuclear antibody	negative
Anti-neutrophil cytoplasmic antibody	negative
Anti-glomerular basement membrane antibody	positive

Chest radiograph: Clear lung fields.

She was diagnosed with renal limited anti-GBM disease and started on methylprednisolone and cyclophosphamide. What other treatment should be initiated?

A. Tacrolimus
B. Blood transfusion
C. IV tazocin
D. Plasma exchange
E. 3-way catheter and bladder irrigation

D. Plasma exchange

Anti-GBM (Goodpasture's) disease classically presents with haematuria and a rapidly progressive glomerulonephritis due to the production of an antibody that targets the basement membrane in the kidney. This antibody can also target the basement membrane in the lung leading to pulmonary haemorrhage. Left untreated there will be a rapid decline in renal function leading to end-stage renal failure. Treatment has three main principles:

- Remove any identifiable cause of antibody production. Exposure to organic solvents, hydrocarbons, metal dust and smoking are known to increase the risk of developing the disease.
- Stop further antibody production using immunosuppressive medication (cyclophosphamide and methylprednisolone).
- Removal of the anti-GBM antibodies rapidly by plasmapheresis to prevent further damage. This is, therefore, the correct answer to this question.

Plasmapheresis is the removal of the patient's plasma from the blood and replacement with another fluid. It is designed to remove large molecular weight molecules from the blood and therefore will remove the circulating anti-GBM antibodies. Plasmapheresis is indicated in the following:

- anti-GBM disease in patients with pulmonary haemorrhage
- those with renal involvement who do not require renal replacement therapy at presentation (the indication in this case)
- selected patients who may require dialysis but present acutely, are young and have a proportion of viable glomeruli on renal biopsy.

Other conditions where plasma exchange is indicated include Guillain-Barre syndrome, hyperviscosity in monoclonal gammopathies and thrombotic thrombocytopenic purpura.

Tacrolimus is used in solid organ transplantation to prevent rejection but is not used to treat acute autoimmune conditions. This patient's haemoglobin is acceptable and she, therefore, does not require a blood transfusion. There is no evidence of infection and antibiotics are not routinely given in anti-GBM disease. A three-way catheter and irrigation is used in haematuria with clot retention and is, therefore, incorrect in this case.

Anti-glomerular basement membrane (GBM) disease (Goodpasture's syndrome)

Anti-glomerular basement membrane (GBM) disease (previously known as Goodpasture's syndrome) is a rare type of small-vessel vasculitis associated with both pulmonary haemorrhage and rapidly progressive glomerulonephritis. It is caused by anti-glomerular basement membrane (anti-GBM) antibodies against type IV collagen. Goodpasture's syndrome is more common in men (sex ratio 2:1) and has a bimodal age distribution (peaks in 20-30 and 60-70 age bracket). It is associated with HLA DR2.

Features

- pulmonary haemorrhage

- rapidly progressive glomerulonephritis
    - this typically results in a rapid onset acute kidney injury
    - nephritis → proteinuria + haematuria

Investigations

- renal biopsy: linear IgG deposits along the basement membrane
- raised transfer factor secondary to pulmonary haemorrhages

Management

- plasma exchange (plasmapheresis)
- steroids
- cyclophosphamide

One of the main complications is pulmonary haemorrhage. Factors that increase the likelihood of this include:

- smoking
- lower respiratory tract infection
- pulmonary oedema
- inhalation of hydrocarbons
- young males

Question 36 of 84

A 52-year-old gentleman complains of severe abdominal pain. He has a background of stage III chronic kidney disease, aspergillosis, type two diabetes mellitus and peripheral vascular disease. Following review by the surgical team, a contrast CT scan is booked. He takes aspirin, clopidogrel, metformin, paracetamol, amphotericin B and insulin. His capillary blood glucose is 8.2mmol/L. He is given IV 0.9% saline and is prescribed to have this before and following contrast. His metformin is also stopped.

Blood tests:

Na +	139 mmol/l
K +	4.1 mmol/l
Urea	5.2 mmol/l
Creatinine	145 µmol/l

Which additional measure is most likely to be effective in preventing contrast-induced acute kidney injury?

A. Start insulin sliding scale
B. Stop paracetamol
C. Give contrast intra-arterially
D. Stop amphotericin
E. Stop aspirin

D. Stop amphotericin

The correct answer is to stop amphotericin. This is most likely to help prevent contrast-induced acute kidney injury (CI-AKI). Whilst stopping metformin is also a correct thing to do, the reason for doing so is that metformin may lead to lactic acidosis when renal function is worsening. Current guidelines do not have a role for n-acetylcysteine but this may change. It is still commonly prescribed and given. There is no reason to start a insulin sliding scale unless the patient becomes nil by mouth.

Question 37 of 84

A 17-year-old male presents with bilateral lower limb and periorbital swelling of gradual onset about 3 days ago. He reports no pain or tenderness elsewhere. He has no past medical, drug allergies and does not take any medications on a regular basis. He has been smoking cigarettes and marijuana for 2 years 'occasionally' but refuses to acknowledge an estimate.

About 5 weeks ago, he had a few days of a 'cough and cold', during which he developed swollen glands in his neck, a sore throat and a temperature of 37.6ºC. He sought medical attention from his GP, who prescribed antibiotics to little avail. The illness spontaneously resolved after 3 days.

A urine dip reveals 4+ protein, no blood, leucocytes or nitrites. A 24-hour urine collection demonstrated protein of 3.8g.

His selected blood tests demonstrate:

Urea	6.5 mmol/l
Creatinine	90 µmol/l
Albumin	21 g/l
Complement	normal
HIV p24 antigen	negative

His renal biopsy demonstrates podocyte fusion. What is the cause of his presentation?

A. Mesangioproliferative glomerulonephropathy
B. Minimal change disease
C. HIV seroconversion
D. Diffuse proliferative glomerulonephritis
E. Secondary focal segmental glomerulosclerosis

B. Minimal change disease

This patient presents with a nephrotic syndrome, characterised by oedema, proteinuria and hypoalbuminuria. The underlying diagnosis is actually given to you by the renal biopsy findings of podocyte fusion, pathognomonic of minimal change disease. His recent viral illness seems to be classical for infectious mononucleosis, which can predispose to minimal change disease. A recent respiratory tract infection should alert you to mesangioproliferative glomerulonephropathy, caused by IgA deposition acutely following an upper respiratory tract infection, and diffuse proliferative glomerulonephritis, caused by reduced C3 and diffuse glomerular proliferation around 2 to 3 weeks after a streptococcal infection. Both however, classically present as nephritic syndrome or at least haematuria. Focal segmental glomerulosclerosis is a consequence of diffuse renal damage and is linked to HIV, sickle cell anaemia and intravenous heroin abuse, not marijuana.

Question 38 of 84

A 47-year-old male is referred by his GP in the urgent care centre to the medical team for review. The patient describes 5 months of non-specific symptoms, describing fatigue and malaise with no focal symptoms. He reports that his 'urine looks a bit weird' and a sample reveals frothy urine. A urine dip reveals 4+ protein 4+ blood no leucocytes or nitrites. His resting blood pressure is 190/120 mmHg. On examination, you note bilateral periorbital oedema, a right varicocele and prominent abdominal veins. You also note significant swelling around both eyes. His past medical history includes intravenous drug use, one previous admission for cellulitis around a groin injection site, positive hepatitis B and atopic dermatitis. He denies drug use over the past 2 months and denies any recent infections.

His first serum results are as follows:

Hb	105 g/l
Platelets	426 * 10 /l9
WBC	11.2 * 10 /l9
Na +	138 mmol/l
K +	4.3 mmol/l
Urea	16 mmol/l
Creatinine	230 µmol/l
Albumin	19 g/l
CRP	16 mg/l

An urgent ultrasound of his renal tract reveals a right renal vein thrombus. What is the most likely underlying diagnosis?

A. Minimal change disease
B. Membranous glomerulonephritis
C. Primary focal segmental glomerulosclerosis
D. Right renal vein thrombosis
E. Goodpasture's syndrome

B. Membranous glomerulonephritis

The patient has presented with proteinuria, hypoalbuminaemia and oedema, collectively termed nephrotic syndrome and a consequence of pathology of the glomerular capillary walls and basement membrane. In separating the different causes of nephrotic syndrome, several risk factors direct you towards membranous glomerulonephritis, during which IgG and complement deposit on a thickened glomerular membrane: firstly, the patient is hepatitis B positive.

Secondly, the patient has presented with a renal vein thrombus, present in 5% of membranous glomerulonephritis patients.

Thirdly, the significant hypoalbuminaemia reflects significant protein loss via leaky nephrons, with membranous glomerulonephritis patients commonly presenting with albumin of less than 20.

Minimal change disease is caused by podocyte pathology and most commonly found in children.

Focal segmental glomerulosclerosis is not a diagnosis but a histological description, caused by primary or secondary disorders. The former typically presents acutely or subacutely with renal failure, secondary FSGS is a consequence of diffuse renal insult, increased in risk by HIV, heroin use and sickle cell pathology. Similarly, Goodpasture's disease typically presents acutely with antibodies against the glomerular basement membrane.

While right renal vein thrombosis is the correct diagnosis, it does not provide the underlying cause of the procoagulant state due to loss of anti-coagulation factors.

Question 39 of 84

A 40-year-old man with end-stage chronic kidney disease is seen in the Emergency Department with a swollen right arm. He has dialysis 3 times per week through a fistula in the right arm but dialysis has been painful on the last two sessions and he has had to stop 30 minutes early on both occasions. He does not complain of swelling elsewhere. He has no shortness of breath and no fevers. His past medical history is of reflux nephropathy and hypertension. He has no history of thromboembolism. He has been on dialysis through this fistula for 2 years and the swelling has been developing over the last 2 weeks.

On examination his heart rate is 83 beats per minute, blood pressure 157/94 mmHg, temperature 36.8 ºC and oxygen saturations 98% on air. He has a diffusely swollen right arm with no erythema. His fistula site is clean with recent evidence of needle marks and no discharge. There is a palpable thrill and an audible bruit. His chest is clear and JVP is 2cm. There is no oedema of the contralateral arm and mild oedema of both feet.

Chest x-ray shows mild chronic cardiomegaly with clear lung fields. Blood results are as follows:

Hb	105 g/l	Na +	135 mmol/l
Platelets	170 * 10 /l 9	K +	5.1 mmol/l
WBC	9 * 10 /l 9	Urea	21 mmol/l
Neuts	5.5 * 10 /l 9	Creatinine	753 µmol/l
Lymphs	2.2 * 10 /l 9	CRP	18 mg/l

What is the most likely likely diagnosis?

A. Cellulitis
B. Deep vein thrombosis
C. Fistula stenosis
D. Post-surgical lymphoedema
E. Superior vena cava obstruction

C. Fistula stenosis

This gentleman has acute pain and swelling in his fistula arm in the absence of infective symptoms. The fistula is still working but dialysis is painful. This points to a diagnosis of a stenosis.

Cellulitis is unlikely with normal inflammatory markers. A diagnosis of superior vena cava obstruction is more likely in someone with a tunnelled dialysis line and is typically associated with bilateral arm and face swelling, shortness of breath and widened mediastinum on x-ray. Lymphoedema of the arm is more common following surgery to axillary lymph nodes and is unlikely to present this late. A thrombosis associated with the fistula is possible although there is no history. However, the fistula itself is not totally thrombosed given the thrill and bruit.

Question 40 of 84

A 32-year-old female patient presents with a generalised maculopapular rash accompanied with fever and lower back pain. She has also noted swelling in both her legs. She had been seen by her GP three weeks ago and was given amoxicillin for a chest infection. A urinalysis was carried out and the result is shown below -

Urinalysis

Blood +++

White cells +++ (eosinophils+++)

No organisms seen Glucose - Nitrates -

Blood tests:

Hb	10.6 g/dl
MCV	86 fl
Platelets	390 * 10 /l9
WBC	9.2 * 10 /l9

Na +	136 mmol/l
K +	4.2 mmol/l
Urea	12.4 mmol/l
Creatinine	135 µmol/l

What is the most likely diagnosis?

A. Minimal change nephropathy
B. Membranous glomerulonephritis
C. Acute interstitial nephritis
D. Drug-induced lupus
E. Acute proliferative glomerulonephritis

C. Acute interstitial nephritis

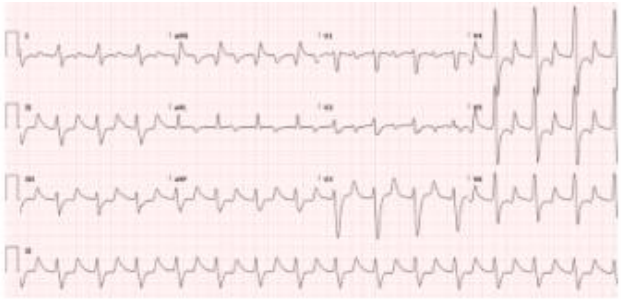
Acute interstitial nephritis (AIN) is a intrinsically renal cause of acute kidney injury that is often brought on by a hypersensitivity reaction (type 4 T-cell mediated) to medications. Medications that can be implicated in this condition include antibiotics (penicillins, cephalosporins, ciprofloxacin and rifampicin), NSAIDs, allopurinol and furosemide. The condition is often asymptomatic and can be found in the presence of normal renal function. Fever is a presenting complaint in up to one third of cases. As well as the acute kidney injury shown here, the condition can cause hyperkalaemia and a metabolic acidosis. Other causes of AIN are infections (legionella, CMV, streptococcal), sarcoidosis, while other cases are idiopathic. It is identified by the presence of eosinophils in the urinalysis which has a sensitivity of 67% and specificity of 83%.

Anaemia in patients with a nephritic picture is associated with an expansion of extracellular fluid volume. The white cell and platelet counts tend to remain in the normal range, although occasionally the white cell count can rise or a mild thrombocytopenia can develop.

Question 41 of 84

A 61-year-old woman is admitted to the acute medical unit with community-acquired pneumonia and stage 2 acute kidney injury from reduced oral intake. She is being treated with intravenous antibiotics and intravenous fluids. Her past medical history includes mild COPD, type 2 diabetes mellitus and hypertension for which she takes metformin, ramipril and regular inhalers.

During her admission, she complains of chest discomfort and palpitations. Her observations are within normal limits. An ECG is taken, as shown below:



What is the next best step in the management of this patient?

A. Aspirin
B. Calcium chloride
C. Insulin and dextrose solution
D. Intravenous sodium chloride
E. Salbutamol nebuliser

B. Calcium chloride

Calcium chloride is correct. This patient has features of hyperkalaemia on her ECG including tall T waves seen in leads II, III, aVF and V3-V6. There are also associated broad QRS complexes which are another feature of hyperkalaemia. Given the admission presentation of pneumonia and an acute kidney injury and ramipril ingestion, hyperkalaemia is quite likely to be present. The management of hyperkalaemia and ECG changes includes immediate administration of intravenous calcium chloride or calcium gluconate to stabilise the cardiac membrane and prevent deterioration into life-threatening arrhythmias.

Aspirin is incorrect. This patient's ECG shows peaked T waves, indicative of hyperkalaemia. Aspirin would be indicated if there were signs of ischaemic heart disease (e.g. ST-segment elevation, depression or T wave inversion).

Insulin and dextrose solution is incorrect. Although this is the treatment of choice to lower the serum potassium level, this patient has signs of ECG changes that are characteristic of hyperkalaemia and, therefore, stabilisation of the cardiac membrane with calcium chloride or calcium gluconate is required.

Intravenous sodium chloride is incorrect. Intravenous fluids are not used in the treatment of hyperkalaemia.

Salbutamol nebuliser is incorrect. Similarly to insulin and dextrose, although this treatment can be used to lower the serum potassium level, this patient has signs of ECG changes that are characteristic of hyperkalaemia and, therefore, stabilisation of the cardiac membrane with calcium chloride or calcium gluconate is required.

Question 42 of 84

A 25 year-old man is referred by his GP with a four week history of bilateral pitting oedema of his legs up to his knees. The only other symptoms he has noticed is some puffiness around the eyes that occurs several times a week and lasts a couple of hours each time. He has no past medical history and reports no rash or musculoskeletal problems. He also has no family history.

Examination reveals bilateral pitting oedema of the legs up to the knees, facial oedema and mild ascites.

Blood tests reveal:

Na +	135 mmol/l
K +	3.4 mmol/l
Urea	4.1 mmol/l
Creatinine	71 µmol/l
Albumin	18 g/l
Cholesterol	11.2 mmol/l

What is the most likely cause of this patient's symptoms?

A. IgA nephropathy
B. Post-infectious glomerulonephritis
C. Minimal change disease
D. Systemic lupus erythematosus
E. Diabetic nephropathy

C. Minimal change disease

This patient most likely has minimal change disease, which will require a renal biopsy to definitively diagnose the condition. IgA nephropathy, post-infectious glomerulonephritis and SLE usually present with a nephritic picture, rather than a nephrotic picture. Although diabetic nephropathy can present as a nephrotic syndrome, the patient does not have any symptoms of diabetes and the patient is young.

Question 43 of 84

A 57-year-old woman, originally from Thailand, presents with a 3 week history of haemoptysis, a dry irritating cough and progressive shortness of breath that has reduced the her ability to climb stairs and walk distances greater than roughly 50 meters. She has no other medical history of note apart from an appendectomy when younger. She takes no regular medication except for over the counter herbal remedies that she gets from a local Chinese medicine shop, although she cannot remember what these are called. She smokes 10 cigarettes per day and does not drink alcohol.

Examination reveals heart sounds 1 and 2 present with no added sounds. Some crackles across the chest. Observations are normal.

Blood tests reveal:

Hb	99 g/l
MCV	74 fL
Platelets	196 * 10 /l9
WBC	14.8 * 10 /l9
Na +	133 mmol/l
K +	5.0 mmol/l
Urea	15 mmol/l
Creatinine	193 µmol/l
ESR	92 mm/hr

A chest x-ray is performed which reveals some diffuse alveolar infiltrates but no focal areas of consolidation. A sputum sample is analysed for MC and S and shows no malignant cells.

What is the most likely diagnosis?

A. Polyarteritis nodosa
B. Pulmonary embolism
C. Goodpasture's syndrome
D. Churg-Strauss syndrome
E. Sarcoidosis

C. Goodpasture's syndrome

It is unusual for Polyarteritis Nodosa to affect the lung and Churg-Strauss syndrome normally presents with vasculitis and asthma. Pulmonary embolism and sarcoidosis also do not typically fit with the above picture. The most likely diagnosis therefore is Goodpasture's syndrome which presents with pulmonary haemorrhage, microcytic anaemia and shortness of breath caused by circulating antibodies against antigens in the alveolar basement membrane. Renal progression of the disease may follow due to crescentic glomerulonephritis.

Question 44 of 84

A 68-year-old man with who has recently been commenced on regular haemodialysis secondary to diabetic nephropathy has come for a regular review. He is concerned about being anaemic as his neighbour who is also on haemodialysis told him he needs treatment for anaemia.

When should this patient be investigated for anaemia?

A. If his haemoglobin falls below 110g/L AND he has symptoms suggestive of anaemia
B. If his haemoglobin falls below 110g/L OR he has symptoms suggestive of anaemia
C. If his haemoglobin falls below 120g/L
D. Only when he has symptoms suggestive of anaemia
E. If his haemoglobin falls below 120g/L OR he has symptoms suggestive of anaemia

B. If his haemoglobin falls below 110g/L OR he has symptoms suggestive of anaemia

As per NICE guidelines, for patients with chronic kidney disease, investigations for anaemia should be considered if their haemoglobin falls below 110g/L OR they develop symptoms suggestive of anaemia. This should begin with testing for iron deficiency using percentage of hypochromic red cells or reticulocyte haemoglobin content. In patients with anaemia of CKD, do not request transferrin saturation or serum ferritin measurement alone to assess iron deficiency status.

Question 45 of 84

A 66-year-old gentleman presents to the emergency department with a fractured humerus following direct trauma. During routine blood tests, it is found that he has stage II chronic renal impairment when compared to previous blood tests. Urinary albumin is found to be within normal range. He has a background of previous ischaemic heart disease and hypertension. His blood pressure ramipril is increased to maximum dose and he is referred to smoking cessation clinic. He is keen to control his blood pressure.

Blood tests:

01/11/2016	08/09/2016	
Na +	144 mmol/l	139 mmol/l
K +	4.8 mmol/l	4.7 mmol/l
Urea	5.2 mmol/l	5.0 mmol/l
Creatinine	112 µmol/l	104 µmol/l

On future review, what should his target blood pressure be in clinic?

A. Less than 130/80mmHg
B. Less than 135/80mmHg
C. Less than 135/85mmHg
D. Less than 140/85mmHg
E. Less than 140/90mmHg

E. Less than 140/90mmHg

The correct answer is 140/90mmHg. If he was diagnosed with diabetes or had urinary ACR >70 mg/mmol is would be 130/80mmHg, and if he was monitoring at home his target would be 135/85mmHg.

Question 46 of 84

A 17 year old female patient presents to the acute medical unit with a seizure. She has no past medical history of note. On assessment, she is post-ictal but maintaining airway, breathing, circulation independently. Her capillary blood glucose is 5.4.

CT brain reveals no bleed, no infarct and no space occupying lesion. ECG: normal sinus rhythm

Bloods are as follows:

Hb	130 g/dl
Plt	60 x10^9/l
WCC	14.5 x10^9/l
Na+	133 mmol/l
K+	5.5 mmol/l
Ur	39 mmol/l
Cr	1001 µmol/l

Blood gas analysis:

pH	7.34
pCO2	4.0 kPa
pO2	13.0 kPa
BE	-4 mmol/l
HCO3-	16 mEq/l

You note her renal function and arrange for further investigation by booking a renal ultrasound and performing immunology bloods to assess for the underlying cause. You catheterise the patient - urine dipstick shows PRO +++, BLD ++. Therefore, a mid-stream sample of urine is sent for microscopy, culture and sensitivity(results awaited). Following discussion with the renal team, the decision is made to dialyse the patient.

Which of the following is an indication for dialysis?

A. Creatinine >1000 µmol/l
B. pH 7.34
C. Potassium >/= 5.5 mmol/l
D. Urea > 25 mmol/l
E. Seizure

E. Seizure

We don't yet know what the underlying diagnosis is yet but it is clear that this patient has an acute kidney injury. We also dont know why she has had a seizure. Given the normal CT head, the next logical cause for the seizure would be the uraemia. The renal team decide to dialyse this patient; the main reason is that they suspect the patient is experiencing symptomatic uraemia (seizure). An elevated urea in of itself would not be an indication without the concomitant symptoms. A creatinine >1000 µmol/l , whilst indicating severe renal impairment, is not an indication for dialysis. A urea of >25 mmol/l is not an indication for dialysis (>27 is the point you could consider dialysis to prevent uraemic symptoms if progressive). You would try to medically treat the potassium first before jumping to dialyse. Potassium >6.5 mmol/l would be an indication. a pH of 7.34 is not severe enough to warrant dialysis. A pH of <7.1 would be an indication.

Question 47 of 84

You review a 65-year-old gentleman in the renal outpatients clinic with diabetic nephropathy. He has opted for hospital haemodialysis and is awaiting fistula creation. He complains of tiredness and poor exercise tolerance. He is clinically euvolaemic. His blood results are as follows:

Na +	134 mmol/l
K +	5.6 mmol/l
HCO3 -	21 mmol/l
Urea	29 mmol/l
Creatinine	480 µmol/l
Hb	89 g/l
MCV	81fL
Ferritin	500 ng/mL

What would be the best management of his anaemia?

A. Ferric carboxymaltose (IV iron)
B. Oral ferrous sulphate
C. Erythropoietin (intravenous)
D. Erythropoietin (subcutaneous)
E. Regular blood transfusion

D. Erythropoietin (subcutaneous)

Anaemia in CKD: erythropoiesis-stimulating agents should be considered once a patient is iron-replete

Weekly subcutaneous erythropoietin is used in pre-dialysis patients for the treatment of renal anaemia. Renal anaemia is typically associated with an eGFR <30 and with a normal MCV. Once serum ferritin stores are optimised (using either oral or IV ferritin), erythropoietin would be considered appropriate. Subcutaneous administration is used in pre-dialysis patients and the intravenous route is used in patients undergoing haemodialysis. Blood transfusions are used in patients with anaemia that is not responsive to erythropoietin when other causes of anaemia have been excluded.

Question 48 of 84

A 73-year-old gentleman, with a known diagnosis of multiple myeloma, is under investigation for declining renal function.

1 year ago	Today	
Na +	142 mmol/l	139 mmol/l
K +	3.9 mmol/l	4.6 mmol/l
Urea	6.5 mmol/l	8.6 mmol/l
Creatinine	132 µmol/l	243µmol/l

A renal biopsy is performed.

What stain should be performed to determine if the decline is associated with a complication of his myeloma?

A. Congo red
B. Gram stain
C. Ziehl-Neelsen
D. Silver staining
E. Auramine

A. Congo red

Myeloma can cause secondary amyloidosis

Light chain myeloma is associated with AL amyloidosis, caused by deposition of light chains in the tissues. Congo red staining is diagnostic of amyloidosis and can be performed on any biopsied tissue.

Gram stain is used to detect bacteria and silver staining is a fungal stain. Ziehl-Neelsen and auramine are stains used to detect Mycobacterium.

Question 49 of 84

A 28-year-old male is referred to endocrinology outpatient clinic for 3 months of increasing anxiety, weight loss and palpitations. His GP suspects hyperthyroidism and would appreciate your review. He has no past medical history except a fibula fracture sustained playing football aged 14 and a right orchidopexy aged 11. On examination, he looks anxious with bilateral sweaty palms. His body mass index is 13.8 kg/m² and you note a sinus tachycardia of 120 beats per minute with a systolic murmur. No neck swelling can be visualised or palpated.

His blood tests are as follows:

Hb	147 g/l
Platelets	278 * 10 /l9
WBC	8.9 * 10 /l9
Na +	141 mmol/l
K +	3.8 mmol/l
Urea	4.5 mmol/l
Creatinine	47 µmol/l
TSH	< 0.01 mu/l
Free T4	33.3 pmol/l (normal 10-24)
Beta HCG	16000 (normal range < 5 mIU/ml for men)

What is the most appropriate next investigation?

A. Ultrasound neck +/- fine needle aspiration
B. Serum anti-thyroid peroxidase antibodies
C. Serum free T3
D. Serum FSH and LH
E. Ultrasound testes

A. Ultrasound testes

The first-line investigation of a testicular mass is an ultrasound

Clinically, the presentation fits with hyperthyroidism. However, the two key unusual bits of information are the previous orchidopexy, which increases the risk of testicular tumours, and the grossly elevated beta HCG in a male patient. Remember that beta HCG is a glycoprotein similar in structure to thyroid stimulating hormone, hence in this case suppressing TSH centrally while augmenting thyroid release of free T4. The investigation most likely to reveal the underlying diagnosis of a testicular germ cell tumour is an ultrasound of the testes. The classic cause of hyperthyroidism and germ cell tumour in a male patient is classically choriocarcinoma due to its typically greatly elevated beta HCG, normally characterised by haemorrhage and necrosis on ultrasound.

Question 50 of 84

A 32-year-old gentleman presented to your acute medical unit with right flank pain. The pain started 2 weeks ago and has been constant. He has tried paracetamol tablets but this has not relieved the pain. He denies any dysuria and is otherwise fit and well. He is a smoker and tells you that his sister recently died from a subarachnoid haemorrhage, which was caused by a berry aneurysm.

He is tachycardic at 110 bpm but other observations are normal. Urine dipstick has 2+ blood, nil else. His blood tests reveal a Sodium 141 mmol/l, Potassium 4.6 mmol/l, Urea 3.6 mmol/l, Creatinine 84mol/l, CRP 34mg/l. His FBC is normal.

What is the most likely diagnosis?

A. Renal stone
B. Pyelonephritis
C. Renal cell carcinoma
D. Polycystic kidney disease
E. Wilms' tumour

D. Polycystic kidney disease

Adult polycystic kidney disease is an autosomal dominant condition. Patients typically present with flank pain, gross haematuria and/or palpable kidneys. The flank pain is thought to be caused by stretching of the wall of the cysts in the kidneys or pressure on other organs nearby. The condition is also associated with berry aneurysms which may rupture to cause a subarachnoid haemorrhage. Some patients may also have liver and pancreatic cysts.

Given he was a young male with a 2-week history of constant pain and a family history of subarachnoid haemorrhage, the most likely answer here would be polycystic kidney disease.

Renal stone would typically present with a shorter history of pain and would be colicky in nature. Patients with pyelonephritis tend to be more unwell and have raised inflammatory markers. Wilms' tumour affects children. Renal cell carcinoma would be more likely if he had weight loss and was older.

Question 51 of 84

A 32-year-old male with a history of end-stage renal failure (ESRF) secondary to Alport syndrome received a deceased donor renal transplant. He had undergone genetic testing previously which demonstrated a mutation in the COL4A5 gene. Two weeks post transplant his creatinine stabilised at 110 µmol/L.

Six months later, he noticed a sudden decrease in urine output. Blood tests showed a rise in creatinine to 450 µmol/L. A transplant biopsy was undertaken which showed a crescentic glomerulonephritis with linear deposition of immunoglobulin G (IgG) along the glomerular capillaries.

What is the most likely diagnosis?

A. CMV infection
B. Acute rejection
C. ANCA-associated vasculitis
D. Interstitial nephritis
E. Anti-GBM antibody disease

E. Anti-GBM antibody disease

Alport syndrome can cause anti-GBM disease following renal transplantation

De novo anti-GBM antibody disease develops in approximately 5 percent of patients with Alport syndrome following renal transplantation. Antibodies are directed primarily against the collagen alpha-5(IV) chain, not the Goodpasture antigen.

ANCA-associated vasculitis can cause a crescentic glomerulonephritis but there are no other features suggesting this in this case.

Acute rejection, tubulointerstitial nephritis and CMV infection can all cause graft dysfunction but would produce different biopsy findings.

Alport's syndrome

Alport's syndrome is usually inherited in an X-linked dominant pattern*. It is due to a defect in the gene which codes for type IV collagen resulting in an abnormal glomerular-basement membrane (GBM). The disease is more severe in males with females rarely developing renal failure.

A favourite question is an Alport's patient with a failing renal transplant. This may be caused by the presence of anti-GBM antibodies leading to a Goodpasture's syndrome like picture.

Alport's syndrome usually presents in childhood. The following features may be seen:

- microscopic haematuria
- progressive renal failure
- bilateral sensorineural deafness
- lenticonus: protrusion of the lens surface into the anterior chamber
- retinitis pigmentosa
- renal biopsy: splitting of lamina densa seen on electron microscopy

Diagnosis

- molecular genetic testing
- renal biopsy electron microscopy: characteristic finding is of the longitudinal splitting of the lamina
- densa of the glomerular basement membrane, resulting in a 'basket-weave' appearance

*in around 85% of cases - 10-15% of cases are inherited in an autosomal recessive fashion with rare autosomal dominant variants existing

Question 52 of 84

A 20-year-old gentleman goes to his GP complaining of haematuria on several occasions over the past year. Every episode occurred with an upper respiratory tract infection or a flu-like illness. There were no abnormalities on examination.

A urine dipstick test showed micro-haematuria with mild proteinuria. The patients urea and electrolytes were within normal range. Serum IgA was elevated.

Which of the following conditions is the most likely diagnosis?

A. Berger disease
B. Goodpasture syndrome
C. Henoch-Schonlein purpura
D. Minimal change disease
E. Post-infectious glomerulonephritis

A. Berger disease

The patient's presentation is consistent with Berger disease (or IgA nephropathy). Microhaematuria or mild proteinuria can occur incidentally or following recurrent upper respiratory/intestinal infections after 1-2 days. This is distinct from post-infectious glomerulonephritis, which commonly occurs 1-2 weeks after infection by group A Streptococcus (pharyngitis or impetigo) and manifests with nephritic syndrome. Haematuria found in the setting of IgA nephropathy however, is concomitant with an upper respiratory tract infection (known as synpharyngitic haematuria).

Question 53 of 84

Beth is a 78-year-old woman who has a longstanding history of chronic kidney disease. She presents to the clinic today for a routine follow-up of her blood tests. She reports that she has been well up to this point. She denies any significant bony pains along her body and has not experienced any issues with passing urine. No recent bony fractures were noted.

Blood tests taken 1 week ago were as below:

Na +	138 mmol/L	(135 - 145)
K +	4.8 mmol/L	(3.5 - 5.0)
Urea	8.0 mmol/L	(2.0 - 7.0)
Creatinine	260 µmol/L	(55 - 120)
Calcium	1.8 mmol/L	(2.1-2.6)
Phosphate	2.0 mmol/L	(0.8-1.4)
Magnesium	0.8 mmol/L	(0.7-1.0)
PTH	75pg/ml	(10-55)

Given the results of her blood tests, what is the most appropriate management for her abnormal blood tests?

A. Advice regarding reduction in foods like chocolate, nuts, shellfish and cola
B. Refer for parathyroidectomy
C. Start cinacalcet
D. Start lanthanum carbonate
E. Start sevelamer

A. Advice regarding reduction in foods like chocolate, nuts, shellfish and cola

Initial management of CKD-mineral bone disease: Correct hyperphosphataemia first; start with dietary changes before starting a phosphate binder

Chronic kidney disease (CKD) is becoming a more common chronic condition and management of its eventual sequelae is important. Mineral bone disease in the context of CKD is often asymptomatic and this is picked up on routine blood tests.

Hyperphosphatemia and hypocalcemia are driving the hyperparathyroidism here. Supplementation like calcium and vitamin D is important to restore the lack of active vitamin D produced by the kidneys and restore normal calcium homeostasis.

Management of hyperphosphatemia will begin with limiting dietary intake of phosphates. The food listed above are examples of food to avoid. It is worth noting that avoiding all these classes of foods are often challenging and difficult for patients to adhere to.

Use of phosphate binders is usually reserved for patients who have not improved on dietary changes. Sevelamer and lanthanum are examples of such phosphate binders.

Cinacalcet will be useful in the management of secondary hyperparathyroidism but its use is usually reserved if the secondary hyperparathyroidism is unresponsive to dialysis.

Lastly, parathyroidectomy will help manage the secondary hyperparathyroidism but this is usually reserved for cases not responding to medical management and dialysis.

Question 54 of 84

A 45-year-old man presents to the nephrology outpatient for a routine follow up. He underwent a renal transplant three months previously. He has a past medical history of hypertension. His medications are tacrolimus, prednisolone, amlodipine, ramipril, spironolactone and doxazosin. He does not smoke or drink alcohol.

The examination is unremarkable and there is no tenderness over the transplanted kidney. Bloods (one month ago):

Urea	5.2 mmol/L	(2.0 - 7.0)
Creatinine	88 µmol/L	(55 - 120)

Bloods (current):

Urea	14.2 mmol/L	(2.0 - 7.0)
Creatinine	189 µmol/L	(55 - 120)

Urinalysis:

Protein	++
Blood	negative
Leucocytes	+++
Nitrites	negative
Glucose	negative

What is the likely diagnosis?

A. Acute graft failure
B. Chronic graft failure
C. Doxazosin use
D. Hyperacute graft failure
E. Urinary tract infection

A. Acute graft failure

Acute graft failure happens within months, is usually asymptomatic and is picked up by a rising creatinine, pyuria and proteinuria

Acute graft failure is the correct answer. The patient is not reporting any symptoms of infection or worsening renal failure. However, we can see that there is worsening graft function in association with pyuria and proteinuria. Three months after a renal transplant, worsening renal function raises the suspicion of acute graft failure, which typically occurs < 6 months after a transplant.

Chronic graft failure is incorrect. This typically occurs at > 6 months post-transplant and is caused by both antibody and cell-mediated mechanisms causing fibrosis to the transplanted kidney (chronic allograft nephropathy). In some other cases, it may be caused by the recurrence of original renal disease.

Doxazosin use is incorrect. This is not classically considered a nephrotoxic medication. If it caused a period of profound hypotension it may plausibly cause an acute kidney injury but there is no suggestion of that from this scenario.

Hyperacute graft failure is incorrect. This occurs within minutes to hours as is due to pre-existing antibodies against ABO or HLA antigens. It is a type II hypersensitivity reaction and leads to widespread thrombosis of graft vessels. This causes ischaemia and necrosis of the transplanted organ. No treatment is possible and the graft must be removed.

Urinary tract infection is incorrect. While the patient has some leucocytes in the urine, he has no symptoms of a urinary tract infection and nitrites are negative, making this diagnosis unlikely.

Question 55 of 84

A 17-year-old boy is admitted to the Acute Medical Unit with a 5-day history of vomiting and persistent diarrhoea which is associated with a cramping abdominal pain. He has now noticed fresh blood in his stool and is struggling to maintain his oral intake. He reports 1-2kg in weight loss. He has previously been well with no past medical history.

On examination he is febrile with a temperature of 38.2ºC. His heart rate is 119bpm with a blood pressure of 100/75mmHg. Chest sounds are clear and heart sounds are normal. He has reduced skin turgor and dry mucous membranes. He is generally tender over his whole abdomen but there is no guarding or palpable masses.

Investigations:

Hb	118 g/L	Male: (135-180)Female: (115 - 160)
Platelets	98 * 10 /L 9	(150 - 400)
WBC	14.7 * 10 /L 9	(4.0 - 11.0)
Na +	148 mmol/L	(135 - 145)
K +	5.4 mmol/L	(3.5 - 5.0)
Urea	13.2 mmol/L	(2.0 - 7.0)
Creatinine	179 µmol/L	(55 - 120)
CRP	152 mg/L	(< 5)

Blood film: schistocytes.

Given the likely diagnosis, what is the next most appropriate investigation?

A. Urinalysis
B. Stool antigen for Clostridium difficile toxin
C. Faecal calprotectin
D. Blood culture
E. Stool microscopy and culture

E,. Stool microscopy and culture

Stool culture looking for Shiga toxin-producing *Escherichia coli* should be sent in patients with suspected haemolytic uraemic syndrome

This patient has a bloody diarrhoea associated with an acute kidney injury (AKI), thrombocytopenia and microangiopathic haemolytic anaemia which points toward a diagnosis of haemolytic uraemic syndrome. The next investigation of choice is to send a stool microscopy and culture to confirm the strain *Escherichia coli* O157.

Urinalysis would be appropriate to send to look for the presence of haematuria or proteinuria which may appear in early disease. However, it is not the next best investigation to send.

Faecal calprotectin is an investigation needed for the diagnosis of inflammatory bowel disease (IBD). Whilst cramping abdominal pain, bloody diarrhoea and weight loss are features of IBD, it does not explain the AKI, haemolytic anaemic and thrombocytopenia.

A blood culture is a necessary investigation given the presence of fever but a stool culture is more appropriate in this scenario.

Clostridium difficile is a less likely diagnosis, making stool antigen testing less appropriate.

Question 56 of 84

A 45 year old woman attends her General Practitioner to report a three month history polyuria. Having previously not been troubled by urinary symptoms, the patient now needs to pass urine up to every 60 to 90 minutes during the day and is being woken from sleep on at least two occasions per night. There is no history of increased urinary urgency or dysuria. The patient had not changed her fluid intake in recent weeks and consumes minimal caffeine and alcohol. Otherwise the patient had been fairly well in herself although had noticed a tendency to loose stools and 4 Kg weight loss over recent weeks.

The patient was normally fit and well and worked as an accountant. Past medical history included only an appendicectomy as a child and there were no previous pregnancies. The patient reported that her twin sister had been diagnosed with coeliac disease 5 years previously. There were no regular medications or allergies.

On examination the patient was slim and appeared slightly anxious. Cardiovascular, respiratory and ocular examination was normal. There were no skin rashes.

Initial investigations requested by the General Practitioner are listed below.

Haemoglobin	12.4 g / dL
White cell count	6.5 * 10 /l9
Platelets	329 * 10 /l9
Urea	3.5 mmol / L
Creatinine	75 micromol / L
Sodium	142 mmol / L
Potassium	3.7 mmol / L
Calcium (adjusted)	2.4 mmol / L
Haemoglobin A1C	5.3 % (reference 4-6)
Serum glucose (random)	4.7 mmol / L

Urine dipstick: negative for nitrites; negative for leucocytes; negative for glucose; negative for ketones; negative for protein; negative beta-HCG

Urine microscopy, culture and sensitivities: white cell count < 10 / mm^3; no growth

What is most appropriate next test to diagnose cause of polyuria?

A. Paired serum and urine osmolality
B. Paired serum and urine osmolality after desmopressin
C. Urodynamic studies
D. Thyroid function tests
E. Serum tissue transglutaminase IgA

D. Thyroid function tests

The patient is presenting with polyuria in the absence of other urinary symptoms associated with diarrhoea and weight loss. There is no evidence of a 'common' (> 1 in 10) cause of polyuria, for example diuretics, caffeine or diabetes. Therefore the most appropriate next test is thyroid function tests, particularly in the setting of other possible symptoms of thyrotoxicosis. Hyperthyroidism is an 'infrequent' cause of polyuria (> 1 in 100).

While paired serum and urine osmolalities may be indicated they are to detect diabetes insipidus, a 'very rare' cause of polyuria (< 1 in 10,000) and are therefore much less likely to make a positive diagnosis of this patient's polyuria. Desmopressin administration is performed after identification of diabetes insipidus to distinguish between a cranial or nephrogenic origin and would not be appropriate at this stage.

The clinical case does not point towards pelvic floor dysfunction necessitating urodynamic studies. While testing for coeliac disease may be indicated given positive family history and diarrhoea, this diagnosis would not explain the patient's polyuria.

Question 57 of 84

A 62-year-old woman with a history of type 1 diabetes and end stage renal failure comes to the renal clinic for review. Over the past few months she has been feeling increasingly lethargic with proximal muscle pains and weakness. She is managed with a basal bolus insulin regime, Examination reveals a blood pressure of 155/82 mmHg, pulse is 70 beats per minute and regular. Chest is clear, there is no ankle swelling. Abdomen is soft and non-tender. You confirm proximal muscle weakness.

Investigations

Hb	101 g/l	Na +	137 mmol/l	Bilirubin	11 µmol/l
Platelets	95 * 10 /l 9	K +	3.7 mmol/l	ALP	185 u/l
WBC	8.4 * 10 /l 9	Urea	13.2 mmol/l	ALT	23 u/l
Neuts	5.1 * 10 /l 9	Creatinine	382 µmol/l	γGT	57 u/l
Lymphs	2.1 * 10 /l 9	Ca ++	2.1 mmol/l	Albumin	30 g/l

You suspect that she has secondary hyperparathyroidism.

At what level of PTH would you begin supplementation with calcium and vitamin D?

A. In the normal range
B. Just above the normal range
C. Twice the normal range
D. Three times the normal range
E. Four times the normal range

C. Twice the normal range

Supplementing calcium and vitamin D in secondary hyperparathyroidism runs the risk of adynamic bone disease if this is begun at less than twice the upper limit of the normal range for PTH. For this reason levels are usually tracked until they cross this threshold, where upon supplementation is commenced. In the event that patients with PTH levels greater than twice the upper limit of normal are left untreated, there is significant risk of progression to tertiary disease, and increased propensity to complications associated with hyperparathyroidism including bone resorption, fracture and ectopic calcification.

Question 58 of 84

A 50-year-old female presented to her GP for a routine check-up. She has a background of obesity and chronic kidney disease stage 3 secondary to poorly controlled type 2 diabetes. Her regular medications include:

- Cholecalciferol 400 units OD
- Insulin (Lantus) 120 units ON
- Insulin (Humalog) 20-25 units before meals
- Gliclazide 120mg OD
- Metformin 1g BD
- Atorvastatin 20mg ON

The clinical examination was unremarkable. Blood test results are as follows:

Na +	136 mmol/L	(135 - 145mmol/L)
K +	4.6 mmol/L	(3.5 - 5.0mmol/L)
Urea	15 mmol/L	(2.0 - 7.0mmol/L)
Creatinine	132 µmol/L	(55 - 120µmol/L)
eGFR	39ml/min/1.73m2	(>90ml/min/1.73m2)
Calcium	2.2 mmol/L	(2.1-2.6mmol/L)
Phosphate	1.68 mmol/L	(0.8-1.4mmol/L)
PTH	65 pg/mL	(14 to 65 pg/mL)
25(OH)D	72nmol/L	(>70nmol/L)

What is the most appropriate first-line management for this patient?

A. Stop vitamin D
B. Change cholecalciferol to alfacalcidol
C. Commence cinacalcet
D. Dietary phosphate restriction
E. Commence sevelamer

D. Dietary phosphate restriction

Initial management of CKD-mineral bone disease: Correct hyperphosphataemia first; start with dietary changes before starting a phosphate binder

This patient has CKD stage 3b. Her bone profile indicates that she is hyperphosphataemic with a calcium level at the low end of normal and a borderline raised PTH. Hyperphosphataemia (due to reduced phosphate excretion in CKD) and hypocalcaemia (due to dysfunctional production of active vitamin D by the kidneys) are both potent stimuli to increase PTH secretion. This patient is at risk of secondary hyperparathyroidism and CKD-related mineral bone disease.

To prevent secondary hyperparathyroidism, the three driving forces should be targeted: hyperphosphataemia, hypocalcaemia, and vitamin D deficiency.

In this patient, the most obvious driving force for PTH secretion is her raised phosphate. Whilst management will depend on the individual patient, NICE guidelines suggest that hyperphosphataemic patients should start with dietary phosphate restriction. If a phosphatebinder is considered to be appropriate (often in combination with dietary restriction), NICE suggests calcium acetate as first-line.

Whilst supplementary vitamin D can have the effect of increasing phosphate (as well as calcium), it would not be appropriate to stop this patient's vitamin D. She is on a low maintenance dose and her vitamin D stores are only just within the required range.

Commencing alfacalcidol is not appropriate for this patient. As CKD progresses, there is a reduction in the activity of the enzyme 1-α hydroxylase in the kidneys, which converts 25- hydroxyvitamin D (25(OH)D) to its more active form, 1,25-dihydroxy-vitamin D (1,25(OH)2D). For this reason, patients are given active vitamin D such as alfacalcidol or calcitriol. This is generally required in later stages of CKD and has the effect of increasing calcium and phosphate resorption from the gut and reducing PTH secretion. KDIGO guidelines state 'It is reasonable to reserve the use of calcitriol and vitamin D analogues for patients with CKDG4-G5 with severe and progressive hyperparathyroidism'. Furthermore, active vitamin D is potent, resulting in an increased risk of hypercalcaemia and hyperphosphataemia. This patient is already hyperphosphataemic; it would be wise to correct her high phosphate in the first instance.

Cinacalcet is a calcimimetic, mimicking the action of calcium on the parathyroid gland such that the gland 'sees' more calcium than is present and reduces PTH secretion. NICE guidelines recommend that this is reserved for severe hyperparathyroidism in whom surgical parathyroidectomy is contraindicated.

Question 59 of 84

A 65-year-old man presents to the emergency department with pain in the right forearm. He has a past medical history of end-stage renal failure and hypertension. He takes ramipril, amlodipine and doxazosin.

On examination, there is an arteriovenous fistula in the right forearm. There is an audible bruit at the fistula. There is no other swelling or erythema. There is no pain on passive movement of the forearm. The colour of the hand is normal.

What is the likely diagnosis?

A. AV fistula infection
B. AV fistula stenosis
C. AV fistula thrombosis
D. AV steal syndrome
E. Compartment syndrome

B. AV fistula stenosis

Arteriovenous fistula stenosis presents with acute limb pain

AV fistula stenosis is the correct answer. Stenosis of an AV fistula can present with acute limb pain at the site of the fistula.

AV fistula infection is incorrect. Typically, patients with this type of infection would present with fevers, chills and malaise. There may be localized erythema over the site of the AV fistula.

AV fistula thrombosis is incorrect. This is classically identified by the absence of a bruit in the AV fistula. There is a bruit present here so this is not likely.

AV steal syndrome is incorrect. This syndrome causes ischaemia of the hand as a consequence of reversal or reduction or reversal of flow through the arterial segment of the fistula distal to the AV fistula. It may cause pallor and necrosis of the hand, which are not present here.

Compartment syndrome is incorrect. This is caused by increased pressure within a muscle compartment causing muscle +/- nerve injury. Typically this occurs after a significant injury.

Furthermore, there is typically pain on passive movement of the region, which is absent in this case.

Question 60 of 84

A 45-year-old man presents to the emergency department complaining of feeling generally unwell with lethargy. He is recovering from a recent diarrhoeal illness. He has no past medical history and there is no family history of note.

The examination is unremarkable apart from an elevated blood pressure of 174/72 mmHg. Blood tests:

Hb	98 g/L	Male: (135-180)Female: (115 - 160)
MCV	92 fl	(80-98)
Platelets	45 * 10 /L 9	(150 - 400)
WBC	4.8 * 10 /L 9	(4.0 - 11.0)
Na +	138 mmol/L	(135 - 145)
K +	4.8 mmol/L	(3.5 - 5.0)
Urea	14.2 mmol/L	(2.0 - 7.0)
Creatinine	168 µmol/L	(55 - 120)
CRP	15 mg/L	(< 5)
Blood film	schistocytes	
Haptoglobin	0.1 g/L	(0.5 - 2.0)
Antinuclear antibody	negative	
ADAMTS13 activity	85%	(> 67%)

What is the likely diagnosis?

A. Atypical haemolytic uraemic syndrome
B. Haemolytic uraemic syndrome
C. Lupus nephritis
D. Scleroderma renal crisis
E. Thrombotic thrombocytopenic purpura

B. Haemolytic uraemic syndrome

Normocytic anaemia, thrombocytopaenia and AKI following diarrhoeal illness - consider HUS

Haemolytic uraemic syndrome is correct. The patient presents with fatigue, hypertension and evidence of thrombotic microangiopathy (anaemia, thrombocytopenia and schistocytes). Additionally, there is further evidence of haemolysis with low haptoglobin. In the setting of a recent diarrhoea illness, this is likely to represent haemolytic uraemic syndrome (HUS) caused by Shiga toxin-producing *Escherichia coli* (STEC). Many of the thrombotic microangiopathies share similar clinical features but it is the association with a diarrhoeal illness that characterizes HUS.

Atypical haemolytic uraemic syndrome is incorrect. This shares many of the same features as HUS but is not caused by STEC but has a genetic component. It is caused by chronic uncontrolled activation of the complement system. It can also mimic thrombotic thrombocytopenic purpura initially. It should be suspected in those patients without a recent history of a diarrhoeal illness who have severe renal impairment, fail to respond to plasma exchange (treatment for TTP) and have normal or near-normal ADAMST13 activity. The treatment is eculizumab, which is a complement inhibitor. There may be a family history. The history of diarrhoea, in this case, makes a diagnosis of HUS much more likely than atypical HUS.

Lupus nephritis is incorrect. This can cause renal failure and proteinuria and in rare cases thrombotic microangiopathy. However, an antinuclear antibody result and an absence of other typical features make this diagnosis unlikely in this case.

Scleroderma renal crisis is incorrect. This can also cause thrombotic microangiopathy and hypertension. However, this is associated with a positive ANA in the vast majority of cases, which is absent here. Additionally, the typical skin findings of scleroderma are not found here.

Thrombotic thrombocytopenic purpura is incorrect. This is a life-threatening cause of thrombotic microangiopathy characterized by a pentad of renal failure, fever, neurological signs and symptoms, schistocytes and anaemia. If suspected, plasma exchange should be commenced without delay as it may be life-saving. Severely reduced levels of ADAMST13 are typical of TTP and can distinguish it from atypical HUS. The normal ADAMST13 levels exclude it here. Additionally, neurological features are often prominent and they are absent here.

Question 61 of 84

A 68-year-old man presents to the nephrology outpatient department for his annual review appointment. His past medical history includes chronic kidney disease stage IIIb and hypertension. He is on amlodipine, ramipril and doxazosin. He doesn't smoke or drink and lives alone. He is a non-executive director of a multi-national company.

His observations are heart rate 84 beats per minute, blood pressure 156/88 mmHg, respiratory rate 16/minute, oxygen saturation 98% on room air and temperature 36.7ºC.

Clinical examination reveals a well-looking man. He is clinically euvolemic. Cardio-respiratory and abdominal examinations are unremarkable.

His consultant reviews his recent blood tests.

Hb	124 g/L	Male: (135-180)Female: (115 - 160)
Platelets	189* 10 /L 9	(150 - 400)

WBC	9.2 * 10 /L 9	(4.0 - 11.0)
Na +	137 mmol/L	(135 - 145)
K +	4.2 mmol/L	(3.5 - 5.0)
Urea	8.9 mmol/L	(2.0 - 7.0)
Creatinine	184 µmol/L	(55 - 120)
CRP	4 mg/L	(< 5)
Adjusted calcium	2.01 mmol/L	(2.20 - 2.60)
Phosphate	1.81 mmol/L	(0.8-1.5)
Albumin	38 g/L	(35-50)
ALP	145 IU/L	(30-130)
Parathyroid hormone	7.6 pg/L	(1.6 - 6.9)

What is the most appropriate initial management of his chronic kidney disease-associated mineral bone disease?

A. Cinacalcet
B. Low calcium diet
C. Low phosphate diet
D. Low potassium diet
E. Phosphate binder

C. Low phosphate diet

Initial management of CKD-mineral bone disease: Correct hyperphosphataemia first; start with dietary changes before starting a phosphate binder

Low phosphate diet is the correct answer from the options listed. The first step in management is the correction of chronic kidney disease (CKD) associated hyperphosphataemia. Initially, this is with a trial of dietary changes. Foods such as milk and cheese, dried beans, colas, canned iced teas and lemonade, nuts, and peanut butter are high in phosphorus. Patients with chronic kidney disease and mineral bone disease benefit from a dietician review regarding dietary modification. If this fails, a phosphate binder can be used.

Cinacalcet is incorrect. Calcimimetic drugs such as cinacalcet suppress PTH secretion by mimicking the effect of calcium on tissues. This is an option in treating secondary hyperparathyroidism that is refractory to initial measures such as dietary restriction of phosphate, phosphate binders and vitamin D analogues such as alfacalcidol or calcitriol.

Low calcium diet is incorrect. The calcium is already low due to deficiency of hydroxylated vitamin D, and hyperphosphataemia. A low calcium diet will make this problem worse. However, correcting vitamin D deficiency and hyperphosphataemia is the route to correcting hypocalcemia in this context rather than adding calcium (either via dietary increase or supplements).

Low potassium diet is incorrect. While this might become important as chronic kidney disease progresses towards end-stage renal failure, it does not have a role in correcting CKD-associated mineral bone disease.

Phosphate binder is incorrect. If dietary measures fail, then a phosphate binder can be considered. These work by binding to dietary phosphate and preventing absorption.

Question 62 of 84

A 31-old-man with known sickle cell anaemia presents with three days of left sided loin pain and haematuria. The pain does not radiate into the groin. He has been well for a number of years since his last crisis. He is well apart from recent exercise related knee pains for which he is taking ibuprofen for with good effect.

On examination, he is tender in the left loin and a sample of urine is red with pieces seen within it. There is no jaundice or scleral icterus and he has a palpable spleen felt 2 cm below the costal margin. He is investigated further.

Na +	136 mmol/l
K +	4.6 mmol/l
Urea	5.2 mmol/l
Creatinine	89µmol/l
Hb	96 g/l
Platelets	178 * 10 /l9
WBC	9.7* 10 /l9
Urine	positive for blood only
Ultrasound renal tract	clubbed appearance of calyces and debris noted in the renal pelvis

What is the likely diagnosis?

A. Sickle cell crisis
B. Renal cell carcinoma
C. Renal calculus
D. Papillary necrosis
E. Retroperitoneal fibrosis

D. Papillary necrosis

Renal papillary necrosis presents with loin pain and visible haematuria

This gentleman has sickle cell anaemia and recent NSAID use which are both risk factors for renal papillary necrosis in which the renal medulla becomes ischaemic and starts to slough away . A renal calculus is possible but the history is more suggestive of papillary necrosis. A renal cell carcinoma and retroperitoneal fibrosis do not typically give loin pain. A sickle cell crisis is possible but the disease here is limited to the renal tract only.

Question 63 of 84

A 21-year-old female presented to her general practitioner with a 2 week history of fevers, lethargy and weight loss. She also complained of painful eyes and visual disturbance. She had no past medical history and wasn't taking any medications.

On examination, her blood pressure was 130/78 mmHg and heart rate was 100 bpm. Her temperature was 37.9ºC. Both eyes were red, with conjunctival injection. Urinalysis showed protein 2+ and leukocytes 3+.

Hb	108 g/L	Male: (135-180)Female: (115 - 160)
Platelets	438 * 10 /L 9	(150 - 400)
WBC	13 * 10 /L 9	(4.0 - 11.0)
Na +	142 mmol/L	(135 - 145)
K +	4.8 mmol/L	(3.5 - 5.0)
Urea	17 mmol/L	(2.0 - 7.0)
Creatinine	320 µmol/L	(55 - 120)
CRP	87 mg/L	(< 5)

What is the most likely diagnosis?

A. Sarcoidosis
B. Tubulointerstitial nephritis and uveitis (TINU syndrome)
C. Nephrocalcinosis secondary to Sjogren's syndrome
D. Scleroderma renal crisis
E. Chinese herb nephropathy

B. Tubulointerstitial nephritis and uveitis (TINU syndrome)

Young woman presenting with AKI and visual disturbance think TINU (tubulointerstitial nephritis with uveitis)

This is a classic presentation of tubulointerstitial nephritis and uveitis (TINU syndrome). It typically occurs in young women and presents with fevers, weight loss and uveitis. Urinalysis shows pyuria and proteinuria. Acute kidney injury is caused by tubulointerstitial nephritis and usually responds well to steroids.

Scleroderma is associated with Sjogren's syndrome, which can cause dry eyes, however scleroderma renal crisis would typically present with accelerated hypertension.

Nephrocalcinosis can be seen in sarcoidosis and Sjogren's syndrome and can cause chronic kidney disease but would not present acutely as in this case.

Chinese herb nephropathy can cause interstitial nephritis, but there is no mention of ingestion of over-the-counter medicines in the history.

Question 64 of 84

A 35-year-old banker attends the general medical clinic for erectile dysfunction. He has noticed progressively finding it more difficult to sustain an erection since starting a new job. He is normally very fit, having started to use his bicycle to get to work, which is over an hour in each direction, and has thereby managed to reduce his BMI from 27 to 23. He has found his new work somewhat stressful but not exceedingly so. He does not think that he has any mental health problems. He denies any previous medical problems apart from an appendectomy at 19 years old. On examination, his genitalia appears normal with normally sized testicles. Blood tests are arranged, including testosterone, HbA1c and routine blood tests.

What is the most appropriate advice to give him?

A. Advise weight loss
B. Advise to take time off work
C. Advise to try stop cycling
D. Advise trial of vacuum erection device
E. Advise alprostadil

C. Advise to try stop cycling

People with erectile dysfunction who cycle for more than three hours per week should be advised to stop

This is a patient who has developed erectile dysfunction after starting to cycle. There is evidence that cycling more than three hours per week (and this patient cycles far in excess of this) can cause reversible nerve damage leading to erectile dysfunction. Therefore a trial of stopping cycling is advised. As the patient is already a healthy weight further weight loss should not be advised. Vacuum erection devices can be considered for elderly men with infrequent use, and alprostadil can be considered as a second line treatment, especially in erectile dysfunction secondary to spinal cord compression. It would also be appropriate for this man to have a trial of sildenafil.

Question 65 of 84

You are the endocrinology registrar in the diabetic clinic. You see a 45-year-old female with type 2 diabetes mellitus. She is generally well in herself. She currently takes metformin 500 mg bd and has no other regular medications. She has good hypoglycaemia awareness and rarely gets hypoglycaemic episodes. Blood sugars on home monitoring range between 5 and 9.

Routine blood tests show:

Hb	133 g/l
Plt	141 x10^9/l
WCC	13.4 x10^9/l
Na+	133 mmol/l
K+	3.1 mmol/l
Urea	3.1 mmol/l
Creatinine	33 µmol/l
eGFR	79 ml/kg/1.73m²
HbA1c	46 mmol/mol (6.4%)

Urinalysis: PRO+, GLUC +, nil else.

Urinary ACR (albumin:creatinine ratio) is 3.6 mg/mmol. On examination, her BMI is 21 kg/m²; she has good peripheral sensation, fundi look normal. The chest is clear to auscultation, heart sounds are normal with no murmurs and her abdomen is soft and non-tender. Her heart rate is 72 beats per minute and regular, blood pressure is 124/75 mmHg, respiratory rate is 20/min, oxygen saturations are 98% on air and temperature 36.8 degrees.

What medication should be commenced in this lady for her ongoing management?

A. Commence gliclazide 80mg BD
B. Commence ramipril 1.25mg OD
C. Increase metformin to 500 mg TDS
D. Commence candesartan 2 mg OD
E. Commence antibiotics for suspected UTI

B. Commence ramipril 1.25mg OD

All diabetic patients with a urinary ACR of 3 mg/mmol or more should be started on an ACE inhibitor or angiotensin-II receptor antagonist

The only abnormalities, in this case, are an unexplained raised white cell count and increased urinary albumin:creatinine ratio (>3.5 mg/mmol for women or >2.5 mg/mmol for men).

There are no symptoms or other tests to suggest a cause for the elevated white cell count and giving antibiotics for a suspected UTI would not be advised given the absence of nitrites and leukocytes on urine dipstick.

The patient does not need referring to the renal team at the moment as the renal function is fine.

The patient's diabetic control is good with a HbA1c of 46 mmol/mol and she does not therefore need an increased dose of oral hypoglycaemic agent.

This leaves us with two options - a) and b).

B is the correct answer due to the patient's microscopic proteinuria - an ACE inhibitor would be commenced despite the normal blood pressure as a renal protective agent as an elevated ACR is a predictor of deterioration in renal function due to diabetic nephropathy in the future. There is no need to add gliclazide into this woman's medications for improved glycaemic control as her HbA1c is within acceptable limits.

Question 66 of 84

A 60-year-old woman is seen in an outpatient renal clinic. She has hypertension and has had type 1 diabetes mellitus since the age of 20. She has an eGFR of 15mls/min/1.73m², it is expected that she will need to commence renal replacement therapy in the next year.

She takes the following medications: Atorvastatin 40 mg nocte, ramipril 2.5mg daily, amlodipine 5mg daily, ferrous sulphate 200mg daily and a basal bolus regimen of insulin. She had previously been taking alfacalcidol and calcium acetate but they had been recently stopped.

On examination, she has some pitting ankle oedema up to her mid-tibia, a few bibasal crepitations on examination of her chest and appears comfortable at rest. There are no signs of uraemia.

Investigation results:

Sodium	140 mmol/L
Potassium	5.0 mmol/L
Serum corrected calcium	2.59 mmol/L
Serum phosphate	1.9 mmol/L
Plasma parathyroid hormone concentration	4pmol/L (0.9-5.4)

What would be the most appropriate step to correct the phosphate concentration?

A. Start renal replacement therapy now
B. Calcium carbonate
C. No action needs to be taken
D. Sevelamer
E. Alendronic acid

D. Sevelamer

This patient needs a phosphate binder; there are no indications to start renal replacement therapy as the patient is relatively well with no signs of uraemia. Calcium carbonate can be used as a phosphate binder but would be inappropriate here due to the impending possible hypercalcaemia. Sevelamer as a non-calcium based phosphate binder would thus be the most appropriate treatment. Alendronic acid has no role in the management of hyperphosphataemia.

Question 67 of 84

A 27-year-old woman with chronic kidney disease stage 5 secondary to focal segmental glomerulosclerosis presents with left forearm pain. This has been getting gradually worse over the past few weeks and is no longer controlled with a combination of paracetamol and codeine. An xray of the left forearm is shown below:

What is the most likely cause of this appearance?

A. Sarcoma
B. Osteomyelitis
C. Secondary hyperparathyroidism
D. Fibrous dysplasia
E. Complication of phosphate binder therapy

C. Secondary hyperparathyroidism

The correct answer is **Secondary hyperparathyroidism**. This condition is a common complication of chronic kidney disease (CKD), particularly in stage 5. It arises due to phosphate retention and decreased calcium levels, which stimulate the parathyroid gland to produce more parathyroid hormone (PTH). Over time, this leads to high PTH levels, causing bone resorption and subsequent bone pain as well as radiological changes known as renal osteodystrophy. The x-ray shows a Brown tumour caused by secondary hyperparathyroidism.

Sarcoma is incorrect because although sarcomas can cause bone pain and abnormal x-ray findings, they are relatively rare. Moreover, there's no specific link between CKD and an increased risk of sarcoma. In addition, the gradual onset of symptoms over weeks is more consistent with a metabolic or inflammatory process rather than a malignant one.

Osteomyelitis could cause similar symptoms but it's typically associated with acute onset of severe pain, fever and other systemic signs of infection which are not mentioned in the scenario. Furthermore, the x-ray changes seen in osteomyelitis usually show areas of bone destruction and possible abscess formation rather than generalised alterations in bone density.

The option **Fibrous dysplasia** is also incorrect. Fibrous dysplasia is a benign condition where normal bone and marrow are replaced by fibrous tissue leading to abnormal growth or swelling of bone. However, it does not have any correlation with CKD nor does it typically present with progressive pain.

Lastly, **Complication of phosphate binder therapy** is unlikely because phosphate binders such as calcium acetate or sevelamer are used to control high serum phosphate levels in patients with CKD. Although they can cause side effects like constipation or nausea, they do not directly cause bone pain or radiological changes seen on the x-ray.

Question 68 of 84

A 21-year-old male with end stage renal failure who had undergone a cadaveric renal transplant 8 weeks previously presented with haematuria and a decline in his graft function from a serum creatinine of 110 µmol/L to 320 µmol/L. He developed renal failure and sensorineural deafness in childhood. He was compliant with his current medications which included prednisolone, tacrolimus and cyclophosphamide. Three of his maternal uncles had developed renal failure in their teenage years.

On examination, he appeared well but pale with a scar in the left iliac fossa and a palpable, tender renal graft. His temperature was 37.2 degrees Celsius, his pulse was 85 beats per minute and regular, his blood pressure was 112/75 mmHg, his respiratory rate was 18 breaths per minute and his oxygen saturations were 97% on room air.

Investigations:

Urine dip: blood+++, protein++

Haemoglobin	95 g/L
White cell count	7.7 x10^9/L
Platelet Count	181x10^9/L
INR	0.9
Serum sodium	141mmol/L
Serum potassium	5.1mmol/L
Serum urea	20.4mmol/L
Serum creatinine	320micromol/L
CRP	24 mg/L

Renal transplant ultrasound scan: The renal transplant lies in the left iliac fossa. It is normal in size with increased cortical echo texture. There is no hydronephrosis. Doppler studies demonstrate good arterial and venous flow.

What is the most likely reason for the decline in graft function?

A. Cytomegalovirus infection
B. Urinary tract infection
C. Anti-glomerular basement membrane antibodies
D. Dehydration
E. Urinary calculi

C. Anti-glomerular basement membrane antibodies

The family history of renal disease in this patient suggests a familial cause for this patient's endstage renal failure. This, coupled with the history of sensorineural hearing loss, suggests that this patient suffers from Alport syndrome. This is a genetic disorder of type IV collagen which is normally inherited in an X-linked dominant fashion and is more severe in males. It classically causes haematuria, progressive loss of renal function, sensorineural deafness and ocular abnormalities. Following renal transplantation, these patients can mount an immune response to the type IV collagen found in the graft transplant as it is a novel antigen. This leads to the formation of anti-glomerular basement membrane antibodies, a rapidly progressive glomerulonephritis and a decline in graft function as seen in patients with Goodpasture's syndrome. As such, C is the most likely cause of the deterioration in this patient's renal function.

Cytomegalovirus (CMV) infection presents with a mononucleosis-type illness and a deterioration in graft function and is, therefore, unlikely in this case. Although bacterial infections are more common in immunosuppressed individuals, there is nothing in the clinical history to suggest urinary tract infection. Dehydration alone would not account for this patient's symptoms. There is no history of pain to suggest urinary calculi.

Question 69 of 84

A 19 year-old man presents with frank haematuria. He had just recovered from a severe cold and states that the symptoms of the cold have now resolved. He took paracetamol for the cold and was not prescribed any antibiotics. His past medical history includes hearing problems from a young age, for which he wears hearing aids and a recent corneal ulcer. His observations are stable and urine dipstick reveals protein ++ and blood +++ in his urine.

It is decided that he will undergo a renal biopsy.

What is the most likely finding under light microscopy?

A. Nothing
B. Rapidly progressive glomerulonephritis
C. Membranous nephritis
D. Mesangiocapillary glomerulonephritis
E. Acute tubular necrosis

A,. Nothing

This patient has Alport's syndrome - a combination of nephritis causing microscopic haematuria with episodic frank haematuria, high frequency sensorineural deafness and ocular problems which include corneal ulcers and bilateral lenticonus. Light microscopy is usually unremarkable and electron microscopy is usually required.

Question 70 of 84

A 64-year-old gentleman was seen in the renal patient clinic. He had a known history of polycystic kidneys and for the last nine months was undergoing haemodialysis. He complained of increasing shortness of breath on exertion over the last few weeks with a reduced appetite. He was assessed by his GP who undertook the following investigations:

Results (16 weeks ago):

Hb	96 g/l
Platelets	242 * 10 /l9
WBC	6.6 * 10 /l9
Ferritin	22 (NR 15-250 mcg/l)
B12	322 (NR 160-900 ng/l)
Folate	18 (NR 3-20 mcg/l)
Na +	136 mmol/l
K +	4.8 mmol/l
Urea	22.2 mmol/l
Creatinine	468 µmol/l

Upper GI endoscopy: hiatus hernia seen

Lower GI endoscopy: NAD He was subsequently commenced on ferrous sulphate 200mg TDS by his GP, but did not feel much better. His past medical history comprised diabetes mellitus, hypertension and hypercholesterolaemia for which he was prescribed aspirin 75mg OD, simvastatin 40mg ON, ramipril 5mg OD, amlodipine 5mg OD and gliclazide 80mg OD. Examination revealed the presence of a pale gentleman with a blood pressure of 132/78 mmHg, heart rate 82 bpm and respiratory rate of 18/min. Examination of his cardiovascular and respiratory systems revealed the presence of a JVP 3cm and an absence of pedal oedema with good air entry in all zones. Examination of his abdomen revealed the presence of two ballotable masses in the renal angle but otherwise no masses and auscultation was unremarkable. Investigations undertaken at the clinic revealed the following results:

Hb	101 g/l
Platelets	222 * 10 /l9
WBC	7.2 * 10 /l9
Ferritin	76 (NR 15-250 mcg/l)
Transferrin saturation	18%

B12	342 (NR 160-900 ng/l)
Folate	17 (NR 3-20 mcg/l)
Na +	136 mmol/l
K +	4.6 mmol/l
Urea	25.2 mmol/l
Creatinine	512 µmol/l
TSH	1.2 mu/l

What is the single next best management step?

A. Continue oral iron therapy and recheck FBC, ferritin and transferrin saturation in four weeks
B. Commence intravenous iron therapy
C. Arrange referral to gastroenterology and haematology for further investigation
D. Commence erythropoietin therapy
E. Arrange packed red cell transfusion

B. Commence intravenous iron therapy

Anaemia in CKD: correct iron deficiency before starting erythropoiesis-stimulating agents

This gentleman has developed anaemia, a well recognised complication of chronic kidney disease. There are several causes of anaemia, including erythropoietin deficiency, iron deficiency anaemia, chronic inflammation, malignancy as well as the usual causes of anaemia, which require consideration. His iron stores remain deplete despite oral iron therapy, as indicated by a ferritin of less than 100 and a transferrin saturation of less than 20. In this instance he therefore requires repletion of his iron stores prior to commencing erythropoietin therapy. Intravenous iron therapy is thus the most appropriate treatment option; there is no indication for a packed red cell transfusion here. See guidelines by The Renal Association for more information.

Question 71 of 84

You are the medical doctor on an acute medical admissions unit. A 42-year old gentleman with type 1 diabetes and Alport's syndrome presents with an acute kidney injury following a renal transplant 2 weeks ago from his brother (still alive). He is on immunosuppressive therapy (tacrolimus and mycophenolate mofetil) and otherwise feels well. Examination is normal but his urine dipstick has 3+ blood and protein.

His blood tests are as follows:

Na +	141 mmol/l
K +	4.7 mmol/l
Urea	18.6 mmol/l
Creatinine	288 µmol/l (baseline 80 µmol/l)
eGFR	34 ml/min (baseline 82 ml/min)

What is the most likely cause of his presentation?

A. Acute graft rejection
B. Accelerated graft rejection
C. Renal vein thrombosis
D. CMV infection
E. Goodpasture's syndrome

E. Goodpasture's syndrome

Accelerated graft rejection happens within days. Patients with renal vein thrombosis and CMV infection would likely be symptomatic. Acute graft rejection would be the appropriate answer if Goodpasture's syndrome was not an option and the patient did not have Alport's syndrome. Alport's syndrome is associated with a defective type IV collagen synthesis, which form part of the basement membrane in the kidneys. The body therefore mounts an immune response towards the type IV collagen in the transplanted kidneys and patient's develop Goodpasture's syndrome.

Question 72 of 84

A 23-year-old woman with sickle cell anaemia and repeat crises associated with this is seen because she has noticed blood in her urine in the last 2 days. She also is having left sided loin pain that does not radiate. She reports it coming in waves. Her bowel habits are unchanged and she saw her GP at the onset of symptoms who suggested this was a urinary tract infection and started her on nitrofurantoin.

On examination, she is tender in the left loin . Her observations show a heart rate of 120 beats per minute, respiratory rate of 22/min and temperature of 37.5ºC. She starts to shake but remains conscious. She passes urine which is red with brown sediment within. She is investigated further.

Hb	98 g/l	Na +	137 mmol/l
Platelets	345* 10 /l 9	K +	4.2 mmol/l
WBC	13.2 * 10 /l 9	Urea	5.2 mmol/l
Neuts	9.6* 10 /l 9	Creatinine	107 µmol/l
Lymphs	2.2 * 10 /l 9	CRP	96 mg/l
Eosin	0.2 * 10 /l9		
Urine microscopy	red cell casts seen, sloughed tissue of medullary cell		
Ultrasound KUB	rings seen in medulla,		

What is the likely diagnosis?

A. Papillary necrosis
B. Urinary tract infection
C. Renal calculus
D. Renal tubular acidosis
E. IgA nephropathy

A. Papillary necrosis

Haematuria in sickle cell anaemia -> renal papillary necrosis

This lady has sickle cell anaemia and from the history may also have a urinary tract infection though she has become very unwell with it. There are features of pyelonephritis here with haemodynamic instability and loin pain but she also has frank haematuria which is rare. This is unlikely to be a renal calculus given the urine microscopy and USS KUB findings. IgA nephropathy would have a viral upper respiratory tract infection prodrome. Renal tubular acidosis does not present so acutely. This lady may have pyelonephritis but this is not an option. The sloughed renal medulla cells suggests infarction of the renal medulla and this occurs in renal papillary necrosis which is common with sickle cell anaemia and pyelonephritis.

Question 73 of 84

A 40 year old man attended his General Practitioner and reported ongoing frequency of urination for the past few weeks. The patient reported passing very dilute urine. There was no history of hesitancy of micturition or terminal dribbling.

The patient was also under investigation for suspected liver disease after presenting with mild jaundice 3 weeks previously. Some initial investigations had been completed but hepatology clinic review was still awaited. There was no other past medical history and the patient took no regular or illicit medications. The patient worked as an insurance agent, lived alone and drank only minimal amounts of alcohol.

A summary of initial investigations arranged by the General Practitioner and through endocrine clinic are given below.

Haemoglobin	11.5 g / dL
White cell count	6.5 * 10 /l9
Neutrophils	4.2 * 10 /l9
Platelets	156 * 10 /l9
Urea	9.3 mmol / L
Creatinine	145 micromol / L
Sodium	156 mmol / L
Potassium	3.9 mmol / L
Albumin	30 g / L
Alkaline phosphatase	170 U / L
ALT	150 U / L
Bilirubin	80 micromol / L
Ferritin	439 ng / mL
Serum immunoglobulins	No abnormality detected
Hepatitis viruses	No abnormality detected
Thyroid stimulating hormone	1.0 microU / L (reference 0.4-5.0)
T4 free serum	13.4 pmol / L (reference 8.5-15.2)
Plasma osmolality	390 mmol / L (reference 280-295)
Urine osmolality	80 mmol / L (reference 100-900)
Plasma osmolality (after desmopressin)	267 mmol / L (reference 280-295)
Urine osmolality (after desmopressin)	265 mmol / L (reference 100-900)
Random serum glucose	4.5 mmol / L

What is the cause of the patient's polydipsia?

A. Nephrogenic diabetes insipidus
B. Liver cirrhosis secondary to haemachromatosis
C. Primary polydipsia
D. Chronic renal failure
E. Cranial diabetes insipidus

E. Cranial diabetes insipidus

> Diabetes insipidus is characterised by a **high plasma** osmolality and a **low urine** osmolality

The patient has a high plasma osmolality and low urine osmolality consistent with diabetes insipidus. The osmolality values correct with desmopressin indicating a cranial aetiology. The likely cause of this is haemachromatosis implied by deranged liver function tests and raised ferritin. The other possible answers would not explain the observed osmolality values.

Question 74 of 84

An 18-year-old female presents with collapse. Her bloods show

Hb	112 g/l
Platelets	324 * 10 /l9
WBC	4.2 * 10 /l9
Na +	130 mmol/l
K +	2.4 mmol/l
Urea	8.9 mmol/l
Creatinine	109 µmol/l
CRP	0.5 mg/l

Her blood pressure is 77/38 mmHg and heart rate 121 beats per minute. Her only past medical history is anorexia and self-harm and she is minimally cooperative with any attempt at a history or examination.

Which of these is the most likely cause for her presentation?

A. Pseudohypoaldosteronism
B. Familial hyperaldosteronism type 1
C. Addison's disease
D. Furosemide abuse
E. Aldosterone-producing adenoma

D. Furosemide abuse

The most likely cause here is furosemide abuse as it explains her hypotension, tachycardia and acute kidney injury (dehydration/ fluids depletion) and hypokalaemia.

Addison's disease would explain the hyponatraemia and hypotension but is typically associated with hyperkalaemia. Similarly, pseudohypoaldosteronism would likely cause high potassium.

Excess aldosterone (either production by adenoma or familial) would cause hypokalaemia but is far more commonly associated with hypertension.

Question 75 of 84

A 58-year-old man is referred to metabolic medicine clinic by his urologist. The patient has been unfortunate enough to suffer from repeated episodes of renal stones over the past few years.

The initial presentation was of multiple attacks of right sided ureteric colic with presence of renal calculi demonstrated on ultrasound. At this initial presentation, the patient's stones had passed spontaneously. The patient reported that since his initial symptoms he had been very careful to maintain his hydration level, going so far as to carry a large water bottle with him at all times.

Two months previously, a new episode of left sided ureteric colic had started. Abdominal CT scanning had shown multiple left and right sided renal calculi with left sided hydronephrosis and hydroureter. The patient had undergone lithotripsy and an ureteric stent had been sited to relieve the obstruction. The stent had been removed after six weeks and a retained renal calculi sent to the laboratory for analysis.

The patient had an otherwise fairly limited past medical history, reporting only a previous diagnosis of hypercholesterolaemia and a previous appendicectomy. His only regular medication was simvastatin 40 mg daily. The patient lived with his wife and worked full time as a bus driver.

While the patient was currently free of symptoms, he was keen to consider any further interventions that would reduce his risk of suffering from recurrent attacks in the future. Results of investigations organised following clinic review are given below.

Urea	7.3 mmol / L
Creatinine	95 micromol / L
Sodium	137 mmol / L
Potassium	4.7 mmol / L
Parathyroid hormone	5.1 pmol/L (reference: 1.6-6.9)
Urinary calcium (24 hour collection)	11.6 mmol / 24 hours (reference: 2.5-8.6)
Urinary sodium (24 hour collection)	156 mmol / 24 hours (reference 40-220)
Urinary uric acid (24hour collection)	2.5 mmol / 24 hours (reference: < 4.8)
Biochemical analysis of calculi	calcium phosphate

What it the correct management to reduce the patient's risk of recurrent renal stones?

A. Chlorthalidone
B. Allopurinol
C. Acetohydroxamic acid
D. Bumetanide
E. Sodium bicarbonate

A. Chlorthalidone

In a patient with hypercalciuria and renal stones, calcium excretion and stone formation can be decreased by the use of thiazide diuretics

The patient has recurrent symptoms secondary to renal calculi with biochemical analysis indicating calcium phosphate stones. Twenty-four hour urine collection demonstrates hypercalciuria but normal urinary excretion of sodium and uric acid. In such patients, thiazide diuretics such as chlorthalidone have been shown to offer a 47 % relative risk reduction in stone recurrence and therefore this is the appropriate treatment option.

Allopurinol has a role in the treatment of the 15-20 % of calcium stone formers who have associated hyperuricosuria. Sodium bicarbonate is used as a second line option in the treatment of distal renal tubular acidosis. Acetohydroxamic acid is a strong urease inhibitor used in certain patients to reduce the risk of recurrent struvite stones. The loop diuretic bumetanide does not have a role in the medical management of renal stones.

Question 76 of 84

A 54-year-old woman has been admitted to the hospital but the renal team over concerns about possible lupus nephritis after recent blood tests showed an acute kidney injury. Her past medical history includes systemic lupus erythematosus (SLE), two prior transient ischaemic attacks, hypertension, and obesity. She takes hydroxychloroquine, clopidogrel and ramipril regularly.

The renal team is planning to perform a renal biopsy to look for evidence of lupus nephritis and has requested an ultrasound prior to the procedure.

Renal ultrasound	The right renal pelvis appears dilated raising concerns for hydronephrosis. There is no visible obstruction or underlying collection. Both kidneys appear atrophic.

What is an absolute contraindication to the procedure?

A. Antiplatelet therapy
B. Hydronephrosis
C. Hypertension
D. Obesity
E. Renal atrophy

B. Hydronephrosis

Before a renal biopsy is performed, patients should have a renal tract ultrasound. The presence of polycystic kidneys, obstruction of the urinary tract, or hydronephrosis are regarded as absolute contraindications to biopsy

Of the five options above, only **hydronephrosis** is an absolute contraindication to renal biopsy. Other absolute contraindications include polycystic kidneys, urinary tract obstruction, uncontrolled hypertension, significant renal malignancy, and significant bleeding disorders.

Antiplatelet therapy is a relative contraindication to biopsy. Ideally, antiplatelets should be discontinued prior to biopsy (dependent on the antiplatelet used, e.g. 24 hours for aspirin, 5 days for clopidogrel), however, biopsies can still take place despite antiplatelet therapy is the benefits outweigh the risks of bleeding.

Hypertension is neither a relative contraindication nor an absolute contraindication. It is uncontrolled hypertension that is an absolute contraindication due to the increased risk of severe bleeding.

Obesity is a relative contraindication due to the difficulty in performing the biopsy.

Renal atrophy is also a relative contraindication to renal biopsy. A biopsy can go ahead if the benefits outweigh the risk of impaired renal function.

Question 77 of 84

A 65-year-old woman presents with tiredness and a rash which have worsened over the past week. Prior to this, she had an episode of diarrhoea and colicky abdominal pain which was treated as gastroenteritis by her general practitioner. She also states that she has been passing tiny volumes of urine. She has no significant past medical history. Her observations are as follows: heart rate 100 beats per minute, blood pressure 110/65 mmHg, temperature 37.5ºC, and SpO2 95% (on air).

Blood results are as follows:

Hb	85 g/L	Male: (135-180)Female: (115 - 160)
Platelets	15 * 10 /L 9	(150 - 400)
WBC	14.8 * 10 /L 9	(4.0 - 11.0)

Urea	24.7 mmol/L	(2.0 - 7.0)
Creatinine	496 µmol/L	(55 - 120)

Prothrombin time (PT)	14 secs	(10-14 secs)
Activated partial thromboplastin time (APTT)	32 secs	(25-35 secs)
Fibrinogen	6.8 g/L	(2 - 4)
D-Dimer	880 ng/mL	(< 400)
Folate	2.8 nmol/L	(> 3.0)
Reticulocytes	8.5 %	(0.5 - 1.5)
ADAMTS13 activity level	15 %	(>50)

A blood film is also performed:

Blood film	Significant red cell fragmentation

What is the most likely diagnosis?

A. Disseminated intravascular coagulation (DIC)
B. Haemolytic uraemic syndrome (HUS)
C. Immune thrombocytopenic purpura (ITP)
D. Megaloblastic anaemia
E. TTP

B. Haemolytic uraemic syndrome (HUS)

Normocytic anaemia, thrombocytopaenia and AKI following diarrhoeal illness - consider HUS

The anaemia, reticulocytosis, and presence of schistocytes on blood film are highly suggestive of microangiopathic haemolytic anaemia (MAHA).

Haemolytic uraemic syndrome (HUS) is correct. HUS is characterised by the triad of microangiopathic haemolytic anaemia (MAHA), thrombocytopenia, and acute kidney injury. The patient thus fulfils all these criteria.

Disseminated intravascular coagulation (DIC) is incorrect. The coagulation profile makes DIC extremely unlikely. DIC classically presents with prolonged PT, prolonged APTT, extremely high Ddimers, and a low fibrinogen.

Immune thrombocytopenic purpura (ITP) is incorrect. ITP usually presents with an isolated thrombocytopenia. The other features in this case (e.g. renal failure) make this a less likely diagnosis.

Megaloblastic anaemia is incorrect. Although the patient is folic acid deficient (likely as a consequence of red cell haemolysis and increased red cell turnover) there are no other features of megaloblastic anaemia (e.g. hypersegmented neutrophils on blood film). Furthermore, renal failure would not be expected in this condition.

Thrombotic thrombocytopenic purpura (TTP) is incorrect. TTP is classically defined by the pentad of fever, thrombocytopenia, MAHA, neurological symptoms (i.e., headaches, altered mental status, seizures, and visual disturbance), and renal failure. In contrast to HUS, the degree of renal impairment is usually mild. ADAMTS13 levels are usually < 10% in TTP. In this case, the ADAMTS13 level > 10%, the presence of oliguric renal failure, and the lack of fever or neurological disturbance favour the diagnosis of HUS.

Question 78 of 84

A 71-year-old male presents with a fractured neck of femur following a fall while getting out of the car. They are successfully managed with a dynamic hip screw, and are recovering on the ward.

He has a history of hypertension and chronic kidney disease (CKD), taking regular ramipril and amlodipine. He is normally independent at home, living with his wife. He currently smokes 15 cigarettes per day, and does not drink alcohol.

His FRAX score is calculated as 11% 10 year fracture risk, and he is investigated for secondary causes of fragility fractures. Results are below:

DEXA scan	T-score: -3.1

Calcium	2.0 mmol/L	(2.1-2.6)
Phosphate	1.8 mmol/L	(0.8-1.4)
Parathyroid Hormone	89ng/L	10-65 ng/L
Vitamin D	10 nmol/L	25-100 nmol/L
Free thyroxine (T4)	12.1 pmol/L	(9.0 - 18)

What is the most likely cause of his fragility fracture?

A. Primary hyperparathyroidism
B. Idiopathic osteoporosis
C. Secondary hyperparathyroidism
D. Primary osteomalacia
E. Tertiary hyperparathyroidism

C. Secondary hyperparathyroidism

CKD induced secondary hyperparathyroidism is a common contributing factor to fragility fractures, and all patients presenting with fragility fractures should be investigated for underlying causes

Renal dysfunction from CKD leads to a deficiency of the active form of vitamin D, which leads to hypocalcaemia. Parathyroid hormone (PTH) levels rise in response to this, giving secondary hyperparathyroidism, which leads to reduced bone mineral density, placing patients at an increased fracture risk.

Primary hyperparathyroidism causes elevated calcium levels.

Idiopathic osteoporosis is less common in men, and would not account for the elevated PTH levels.

Primary osteomalacia usually gives a low phosphate (although osteomalacia secondary to CKD may have high phosphate).

Tertiary hyperparathyroidism has a normal-high calcium level, in conjunction with an inappropriately high PTH level.

Question 79 of 84

A 40-year-old man with chronic kidney disease stage 5 secondary to autosomal-dominant polycystic kidney disease is seen in general nephrology clinic. He reports worsening fatigue and denies any alteration in bowel habit or weight loss. Examination demonstrates a blood pressure of 120/56mmHg, heart rate 56bpm, respiratory rate 18/min, Sats 99% on air and he is apyrexial. Abdomen is soft on non-tender with bilateral palpable kidneys and a palpable irregular liver edge. He is euvolemic.

Hb	78 g/l
Platelets	167 * 10 /l9
WBC	7 * 10 /l9
Ferritin	95 ng/mL

B12 and folate are within normal range.

What is the most appropriate next step?

A. Faecal occult blood testing
B. Blood transfusion
C. Referral for OGD
D. Iron transfusion
E. Erythropoietin

D. Iron transfusion

Anaemia in CKD: correct iron deficiency before starting erythropoiesis-stimulating agents

This gentleman has chronic kidney disease. In this situation, with a ferritin of <200, then ensuring he is iron replete is the first step, prior to consideration fo EPO-stimulating agents. A blood transfusion would be avoided if possible in order to avoid antigen-sensitisation should he become eligible for transplant.

Should his iron levels not respond to transfusion, then further investigation for other causes of blood loss - such as gastrointestinal - need to be considered. OGD would be appropriate; faecal occult blood testing is used only in the context of the screening programme and is not useful in the investigation of iron deficiency anaemia.

Question 80 of 84

A 59 year-old woman presents for her annual diabetic check-up. She has had type 2 diabetes mellitus for 15 years and takes only metformin for the diabetes. Her HbA1c was revealed as being 40 mmol/mol on her last diabetes check-up 1 week ago. Her past medical history includes peripheral vascular disease and migraine. She has a smoking history of 20 pack years and drinks 5- 10 units of alcohol per week. On examination, her pulse rate is 86 beats per minute and her blood pressure is 126/70 mmHg and is otherwise unremarkable.

Blood tests reveal:

Na +	140 mmol/l
K +	3.4 mmol/l
Urea	7.0 mmol/l
Creatinine	105 µmol/l

Urinalysis reveals:

Protein	Negative
Glucose	Negative
Leucocytes	Negative
Nitrites	Negative

24-hour urine sample demonstrates 190 mg of albumin in the urine.

What is the most appropriate next management with regards to her renal function?

A. Start a beta blocker
B. Start an angiotensin II receptor blocker
C. Optimise the patient's glycaemic control
D. Start an ACE inhibitor
E. Start a thiazide diuretic

D. Start an ACE inhibitor

All diabetic patients with a urinary ACR of 3 mg/mmol or more should be started on an ACE inhibitor or angiotensin-II receptor antagonist

ACE inhibitors are the most appropriate management option prognostically when a diabetic patient presents with microalbuminuria and have been shown to reduce the progression to end stage renal disease. Optimising the patient's glycaemic control, while important in trying to prevent microvascular damage, is limited as their HbA1c is already at a satisfactory level and it important to avoid putting the patient at risk of hypoglycaemia.

Question 81 of 84

A 45-year-old woman attended the hospital with facial swelling and shortness of breath.

Her past medical history includes vesicoureteric reflux, rheumatoid arthritis for which she takes methotrexate, and alcohol excess.

On examination, she had pitting oedema up to her thighs, generalised facial swelling, and fine crepitations to both lung bases. There are needle track marks in both antecubital fossae and a patch of cellulitis 2x5cm around the umbilicus.

Heart rate 102 bpm, BP 174/81mmHg, SpO2 94% on room air, respiratory rate 20/min, T 37.1ºC A urine dip shows protein +++

Na +	144 mmol/L	(135 - 145)
K +	5.4 mmol/L	(3.5 - 5.0)
Urea	18.5 mmol/L	(2.0 - 7.0)
Creatinine	301 µmol/L	(55 - 120)
INR	1.6	(<1.5)

Ultrasound renal tract: mild, bilateral hydronephrosis stable compared to previous, otherwise normal appearances.

Despite fluid resuscitation, her renal function improves only minimally. You want to consider renal biopsy to investigate further.

Why would this patient not be a candidate for this investigation?

A. Current IV drug user
B. INR of 1.6
C. Methotrexate use
D. Presence of hydronephrosis on ultrasound
E. Presence of peri-umbilical cellulitis

D. Presence of hydronephrosis on ultrasound

Before a renal biopsy is performed, patients should have a renal tract ultrasound. The presence of polycystic kidneys, obstruction of the urinary tract, or hydronephrosis are regarded as absolute contraindications to biopsy

Presence of hydronephrosis on ultrasound: This patient would not be a candidate for a renal biopsy due to the presence of hydronephrosis on her ultrasound. This is likely a result of her longstanding vesicoureteric reflux.

Current IV drug user: is incorrect. IV drug use would not preclude someone from having a renal biopsy.

Methotrexate use: is incorrect. Methotrexate is not a contraindication for renal biopsy, though these patients need to be informed about the higher risk of infection due to the immunosuppressive effect of methotrexate. **INR of 1.6**: is incorrect. Renal biopsy can generally be completed safely with INR levels of 1.8 and below.

Presence of peri-umbilical cellulitis: is incorrect. If the cellulitis was covering the site at which the needle needed to pass through the skin this would be an important consideration, however, renal biopsies are generally approached from the posterior flank.

Question 82 of 84

A 64-year-old male Jehovah's witness was brought into hospital two days ago by his wife after increasing generalised fatigue and malaise over the past 4 weeks. Today, he has been unable to get out of bed. His past medical history includes stage 5 chronic kidney disease, hypertension and type 2 diabetes. He continues to pass urine without renal replacement therapy with a baseline creatinine of 260 µmol/l. On examination, he is warm peripherally with conjunctival pallor. His heart sounds are unremarkable, the chest is clear and abdomen soft and non-tender. He has passed 800mls of urine in the last 24 hours. His blood tests are as follows:

Hb	80 g/l
Platelets	201 * 10 /l9
WBC	6.7 * 10 /l9
Ferritin	4 ng/ml
Transferrin saturation	19%
Na +	145 mmol/l
K +	4.9 mmol/l
Urea	17.7 mmol/l
Creatinine	276 µmol/l

What is the most appropriate management?

A. Intravenous blood transfusion
B. Haemofiltration
C. Intravenous iron supplement
D. Intravenous erythropoietin
E. List for renal transplant

C. Intravenous iron supplement

Anaemia in CKD: correct iron deficiency before starting erythropoiesis-stimulating agents

Non-specific symptoms such as malaise and fatigue are common presentations of iron deficient patients with renal disease. This is confirmed biochemically with low ferritin (normal >250 ng/ml) and transferring saturation (TAST), calculated by plasma iron divided by total iron binding capacity (normal >20%). This patient has no acute indications for renal replacement therapy. In fact, he has no signs of acute kidney injury, defined as serum creatinine greater than 1.5 times of baseline and oliguria for 6 hours. In this scenario, there is the added complication of receiving blood products as a result of the patient being a Jehovah's witness. The most appropriate management would be to correct iron stores, which from experience can produce dramatic clinical improvements in renal transplant patients even if ferritin and TSAT are close to normal ranges.

Erythropoietin (EPO) is not appropriate until iron replacement has completed, desaturation of iron stores is one of the most common causes of EPO resistance.

Question 83 of 84

A 22-year-old male was admitted with severe abdominal pain. The pain was in his left flank area going down into his groin. It was constant and he was not able to lie still with the pain. With the pain he felt nauseated. He did not notice any urinary symptoms. He had a similar episode six months ago which resolved with conservative management. He stated since the episode he increased his fluid intake and instituted a trial of dietary change. He was otherwise fit and well, except for a past medical history of asthma for which he used Clenil modulite 200mcg BD and salmeterol 100mcg BD. He smoked 20 cigarettes per day and consumed 20 units of alcohol per week mainly at the weekends.

On examination, he was in obvious discomfort and restless. His blood pressure was 118/72 mmHg, his heart rate was 88 and his temperature was 36.9 degrees celsius. Examination of the cardiovascular system revealed the presence of normal heart sounds with warm well perfused peripheries. Other than tachypnoea, examination of his respiratory system was unremarkable. Examination of his gastrointestinal system revealed the presence of left renal angle tenderness but otherwise a soft non-tender abdomen with no evidence of guarding or rigidity. Examination of his neurological system was unremarkable.

A number of investigations were performed:

Na +	142 mmol/l
K +	4.0 mmol/l
Urea	4.2 mmol/l
Creatinine	77 µmol/l
CRP	12 mg/l
Adjusted calcium	2.40 mmol/l

Urinalysis: blood +++, nil else

KUB x-ray: single solitary area of calcification within left kidney

The patient was treated conservatively with an intravenous fluid infusion, non-steroidal antiinflammatory drugs and opioid analgesia and within 48 hours recovered fully.

He was subsequently seen in the renal clinic where he underwent a number of investigations:

Urea and electrolytes	normal
Urate	0.39 (NR 0.12-0.42 mmol/l)
Vitamin D	62 (NR 30 100 ng/ml)
Parathyroid hormone 48	(NR 11-54 pg/ml)
Urine 24 hr collection	Calcium 525 (NR 25 - 300mg/24hrs) Oxalate 21 (NR 7 - 44 mg/24hrs) Citrate 588 (NR 320 1240mg/24hrs) Sodium 137 (NR 40 220mg/24hrs)

Which therapeutic option will most likely be of benefit in the prevention of future episodes?

A. Calcium carbonate
B. Bendroflumethiazide
C. Allopurinol
D. Captopril
E. Furosemide

B. Bendroflumethiazide

In a patient with hypercalciuria and renal stones, calcium excretion and stone formation can be decreased by the use of thiazide diuretics

This patient has had two episodes of renal colic secondary to renal stones (note that a normal urinalysis does not exclude renal calculi). His abdominal x-ray shows calcification, hence indicating that the stone composition most likely contains a significant quantity of calcium. The most common types of stone contain either calcium phosphate or calcium oxalate. Thiazide diuretics are useful in the prevention of stones due to hypercalciuria (such as eg calcium phosphate stones); potassium citrate may be of use of use for calcium oxalate stones. Allopurinol is of use for uric acid stones, which tend to be radiolucent on plain x-ray investigations.

Question 84 of 84

A 75-year-old female patient with chronic kidney disease (CKD) stage 4 secondary to diabetic nephropathy was reviewed in clinic. She complained of worsening fatigue and exertional breathlessness over the preceding two months. She denied any bleeding or gastrointestinal symptoms. Her medications included ramipril, bisoprolol, atorvastatin and gliclazide.

On examination she was euvolaemic and appeared pale. Cardiovascular, respiratory and abdominal examinations were unremarkable.

Her blood results are shown below. A myeloma screen was also performed, which was negative.

Hb	96 g/L	Male: (135-180)Female: (115 - 160)
Platelets	350 * 10 /L ^{9}	(150 - 400)
WBC	6.5 * 10 /L ^{9}	(4.0 - 11.0)
Na^+	138 mmol/L	(135 - 145)
K^+	5.0 mmol/L	(3.5 - 5.0)
Urea	11 mmol/L	(2.0 - 7.0)
Creatinine	270 µmol/L	(55 - 120)
CRP	3 mg/L	(< 5)
Ferritin	35 ng/mL	(20 - 230)
Transferrin saturations	16 %	(20 - 40)
Vitamin B12	380 ng/L	(200 - 900)
Folate	4.5 nmol/L	(> 3.0)
Reticulocytes	1.2 %	(0.5 - 1.5)

What is the most appropriate next step in management?

A. Start erythropoietin-stimulating agent (ESA) therapy
B. Blood transfusion
C. 3 month trial of oral iron
D. Bone marrow biopsy
E. Intravenous iron

C. 3 month trial of oral iron

Anaemia in CKD: correct iron deficiency before starting erythropoiesis-stimulating agents

This patient has anaemia of chronic kidney disease and iron deficiency. According to NICE guidelines, iron therapy should be offered to people with anaemia of CKD who are iron deficient and who are not receiving ESA therapy, before discussing ESA therapy.

NICE guidelines also state that for CKD patients who are not receiving haemodialysis, a trial of oral iron should be considered before offering intravenous iron therapy.

Blood transfusion would not be appropriate in this situation, as the patient is stable and not bleeding.

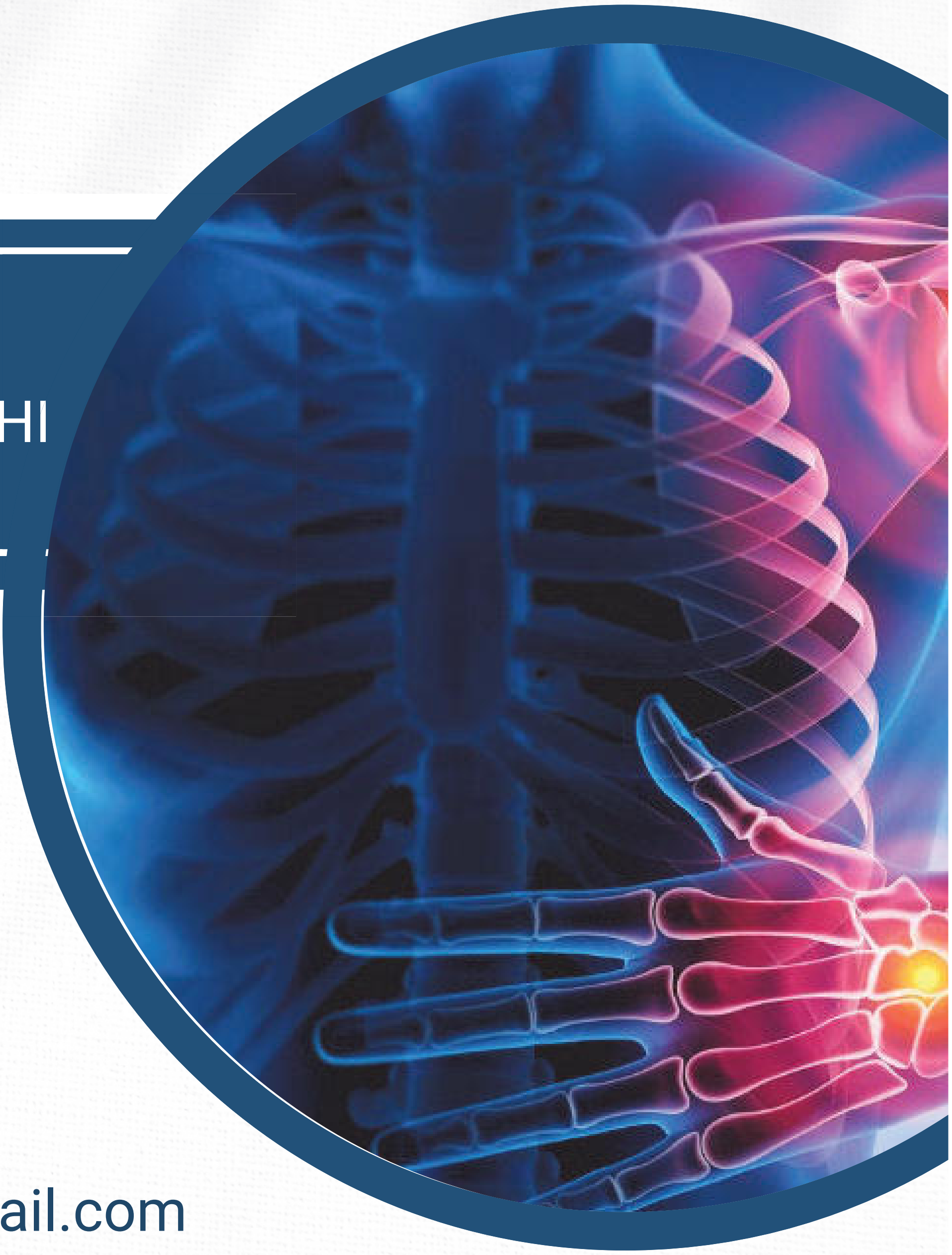
A bone marrow biopsy wouldn't be indicated at this stage, particularly as the myeloma screen was negative, but might be considered if the patient failed to respond to iron and ESA therapy.

Part 2

Alrajhi Jad

PASSMEDICINE
RHEUMATOLOGY

Internal medicine



EDITED BY
DR. JAMIL ALRAJHI

2024

773-555-493
jameelalrajhi@gmail.com

Question 1 of 178

A 65-year-old woman is referred to the rheumatology outpatient department by her general practitioner complaining of bilateral knee pain, worse on mobilization. She has a past medical history of hypertension. She takes amlodipine. She has smoked five cigarettes per day for 20 years and she does not drink alcohol. She works as a cleaner.

On examination, the knees are mildly swollen bilaterally and there is patellofemoral crepitus. There is no joint swelling or tenderness elsewhere. Cardiorespiratory examinations are unremarkable.

What is the most appropriate management?

A. Codeine phosphate
B. Local muscle strengthening exercises
C. Methotrexate
D. Oral NSAID
E. Prednisolone

B. Local muscle strengthening exercises

Local muscle strengthening exercises and general aerobic fitness is an important component of knee and hip osteoarthritis

Local muscle strengthening exercises and general aerobic fitness is the correct answer. The patient presents with bilateral knee pain with mild swelling, worse on mobilization and there is clinical evidence of crepitus. These symptoms and signs are consistent with a diagnosis of osteoarthritis of the knees. The initial management of osteoarthritis of the knees consists of advice regarding local muscle strengthening exercises and general aerobic fitness +/- paracetamol and topical ibuprofen.

Codeine phosphate is incorrect. This is a potential option if there is ongoing pain despite paracetamol, topical ibuprofen and oral ibuprofen but would not be first-choice.

Methotrexate is incorrect. The pain of rheumatoid arthritis tends to be worse at rest rather than on activity and there is no mention of diurnal variation or early morning stiffness in this case. This is a medication used in maintenance of remission in rheumatoid arthritis. Smoking is a risk factor for this condition.

Prednisolone is incorrect. This patient has osteoarthritis, not rheumatoid arthritis and therefore there is no place for oral steroids.

Oral NSAID is incorrect. This is an option for the management of knee osteoarthritis after failure of exercises, oral paracetamol and topical ibuprofen.

Question 2 of 178

A 68-year-old woman presents to a care of the elderly clinic. This is her first attendance following recent frequent falls at home. Her most recent fall was two weeks ago during which time she had a CT head/c-spine/right hip which was negative for any acute injury.

Her only symptom between falls is a pain in her right hip which has been worsening over the past year. This is worse on activity and in the evenings. She is tender to deep palpation and has painful internal and external rotation on examination. Otherwise, she reports that she is well in herself and her only past medical history includes obesity and type 2 diabetes mellitus. She has been advised by her general practitioner to lose weight.

Which of the following is the most appropriate additional advice for her hip pain?

A. Elevate lower limbs when seated
B. Muscle strengthening exercises and aerobic fitness
C. Reduce alcohol intake
D. Regular rest throughout the day
E. Smoking cessation

Muscle strengthening exercises and aerobic fitness

Local muscle strengthening exercises and general aerobic fitness is an important component of knee and hip osteoarthritis

This woman has osteoarthritis of the right hip. This is evidenced by the chronicity and nature of the pain. These factors, as well as her recent negative imaging, make acute injury unlikely. As per the latest NICE guidance, all patients diagnosed with osteoarthritis of the knee or hip should be offered help with weight loss, given advice about local muscle strengthening exercises and general aerobic fitness.

Elevating limbs is helpful in acute injury to reduce acute swelling but will not affect this woman's osteoarthritis.

Reducing alcohol intake is a preventative measure for gout however would not be effective in this case.

Regular rest throughout the day may temporarily prevent symptoms but will be debilitating and not result in any lasting improvement.

Smoking cessation is useful in rheumatoid arthritis however the history here is more classical of osteoarthritis

Question 3 of 178

A 28-year-female presents with increasing fatigue. She also complains of joint pains affecting several joints. There is associated joint stiffness lasting about 20-30 mins. She also gets a skin rash, especially in sun-exposed areas. There is no significant past medical history and she is not using any regular medications. She works as an office clerk and the joint pains are making it difficult for her to continue her work normally. Following are the results of her investigations:

Hb	9.3 g/l	Na	136 mmol/l	Bilirubin	98 µmol/l
Platelets	99 * 10 /l	K	4.2 mmol/l	ALP	89 u/l
WBC	7.3	Urea	4.5 mmol/l	ALT	23 u/l
Neuts	3.4 * 10 /l	Creatinine	87 µmol/l	γGT	43 u/l
Lymphs	2.7 * 10 /l	CRP	<1 mg/l	Albumin	32 g/l
Eosin	0.2 * 10 /l	C3, C4	low		

Chest x-ray and ECG were normal.

Urine exam showed protein +++.

Which of the following is a feature of the underlying condition in this patient?

A. ANA usually negative

B. Absence of a relapsing and remitting course

C. Lower risk of thrombo-embolism

D. Normal CRP

E. Anti-CCP antibody level is highly specific

D. Normal CRP

A raised CRP in a patient with known SLE may indicate an underlying infection

Systemic Lupus Erythematosus (SLE) is the most likely diagnosis in this patient as there are several pointers towards it including female sex, joint pains, photosensitive rash, haemolytic anaemia, thrombocytopenia, normal CRP, low complement level, and proteinuria.

ANA is positive in more than 90-95% of patients with SLE.

with a negative ANA.

SLE is a relapsing and remitting disease.

SLE is associated with an increased risk of thrombosis.

CRP is normal in SLE unless there is a secondary infection.

Anti-CCP has high specificity for rheumatoid arthritis rather than SLE.

Question 4 of 178

A 28-year-old woman presents to the medical take with a macular rash, and arthralgia, which she reports have been ongoing for the past three weeks. She had initially attributed the symptoms to a viral infection, however, when her symptoms failed to improve after a week and she continued to experience fevers of > 39ºC she visited her GP, who referred her to the medical team for further investigations. She denied any early morning stiffness, gastrointestinal upset, dysuria or visual symptoms and prior to the onset of her symptoms was otherwise in good health.

She is a non-smoker, drinks occasional alcohol and has travelled to France and to Spain in the last year. She reports no family history of note and has no risk factors for HIV or tuberculosis aside from previous travel to India 5 years previously. She works as a financial analyst and managed to return to work after 1 week of sick leave, although she has not been able to pursue her usual sporting activities.

On examination, she has a widespread macular rash with minimal signs of excoriation. She has an erythematous oropharynx with no signs of exudate and no significant lymphadenopathy. Cardiovascular, respiratory, abdominal and neurological examinations are unremarkable. Examination of the joints demonstrates no signs of active synovitis and in spite of tenderness in the shoulders and knees, there are no areas of restricted active or passive range of movement.

Her blood tests are detailed below:

Hb	130 g/L	Male: (135-180)Female: (115 - 160)
Platelets	550 * 10 /L 9	(150 - 400)
WBC	15.4 * 10 /L 9	(4.0 - 11.0)
Neuts	13.1 * 10 /L 9	(2.0 - 7.0)
Lymphs	1.6 * 10 /L 9	(1.0 - 3.5)
Mono	0.5 * 10 /L 9	(0.2 - 0.8)
Eosin	0.2 * 10 /L 9	(0.0 - 0.4)
Na +	140 mmol/L	(135 - 145)
K +	4.0 mmol/L	(3.5 - 5.0)
Urea	4.0 mmol/L	(2.0 - 7.0)
Creatinine	70 µmol/L	(55 - 120)
Bilirubin	15 µmol/L	(3 - 17)
ALP	70 u/L	(30 - 100)
ALT	35 u/L	(3 - 40)
ANA	1:20	(< 1:40)

Rheumatoid factor	5 IU/ml	(<14)
CRP	81 mg/L	(< 5)
INR	1.0	(0.9 - 1.3)

A full autoimmune screen along with Lyme serology, HIV and hepatitis screen are all unremarkable and a CT chest/abdomen/pelvis reveals no significant abnormalities. An echocardiogram shows no signs of infective endocarditis and 3 consecutive blood cultures performed off antibiotics are negative. A blood film confirms neutrophilia.

Given the likely diagnosis, what would be the first-line treatment for this patient?

A. Anakinra

B. Methotrexate

C. Methylprednisolone

D. Non-steroidal anti-inflammatory drugs (NSAIDs)

E. Prednisolone

D. Non-steroidal anti-inflammatory drugs (NSAIDs)

NSAIDs are the first line treatment for Still's disease, not steroids

This patient has adult-onset Still's disease as suggested by the Yamaguchi criteria - this patient has four of the major criteria (fever >39 °C, for over 1 week, arthralgia or arthritis for more than two weeks, rash, leukocytosis with >80% polymorphonuclear cells) and one of the minor criteria (negative rheumatoid factor and negative ANA).

Adult-onset Still's disease is effectively a diagnosis of exclusion and malignancy, autoimmune and infection must be ruled out.

This patient's presentation would be described as mild disease as she has no evidence of organ involvement and has been able to continue with her daily life to a large extent. Therefore, NSAIDs should be considered first-line. Should she see no improvement or limited improvement after 10 days - 2 weeks of regular NSAID therapy, oral steroids would be considered.

In patients with moderate disease, i.e. evidence of organ involvement and/or very limited by their symptoms, steroids would be considered first-line.

In patients with severe or life-threatening disease i.e. significant organ dysfunction should be commenced on IV steroids.

In patients who require ongoing steroids to control their symptoms or in whom steroids do not control symptoms, disease-modifying agents such as anakinra or methotrexate should be considered.

Question 5 of 178

A 47-year-old lady presented with a three-week history of pain in her fingers. She had noticed her hands were getting extremely cold when she went outside and turned a 'funny colour'. When she came back inside her hands were very painful as they began to warm up. She had managed in the past by wearing gloves outside but now had ulcers on her fingertips which she had never experienced before. She also complained of epigastric pain and had longstanding shortness of breath.

Her past medical history included pulmonary fibrosis and hypertension. Her medications included propranolol, amlodipine, simvastatin and omeprazole.

On examination the skin over her hands was dry and shiny and there was severe digital ulceration on three fingertips of the left hand. There was no exudate or erythema. The fingertips were dusky in colour and extremely tender. The skin over the upper arms and chest appeared normal. On auscultation of the lungs there were fine bibasal inspiratory crepitations which did not alter in character upon coughing. Heart sounds were normal with no added murmurs. There was a left ventricular heave

Which of the following is the most appropriate management plan for this lady?

A. Start flucloxacillin and stop all anti-hypertensive medications
B. Educate this lady about the use of gloves and hand-warmers and increase her amlodipinedose
C. Stop amlodipine and refer for an urgent dermatology assessment
D. Stop propranolol and admit for an iloprost infusion
E. EStart high dose oral prednisolone

D. Stop propranolol and admit for an iloprost infusion

Epoprostenol (amongst other prostaglandins) can be used in the treatment of Raynaud's phenomenon

This patient has secondary Raynauds associated with an underlying diagnosis of Limited Systemic Sclerosis. Her disease is 'limited' as opposed to 'diffuse' as she does not have skin changes

Question 6 of 178

An 85-year-old man successfully completed treatment for community-acquired pneumonia. He has been medically stable for discharge but unfortunately is still an inpatient as he is awaiting a package of care to be sourced. His past medical history includes hypertension, ischaemic heart disease, type 2 diabetes and advanced chronic kidney disease (not for renal replacement therapy).

He complains of severe pain in his left foot. On examination, you note the metatarsophalangeal joint on the hallux is swollen, erythematous and hot to touch. No abnormality is detected on his right foot.

He is afebrile and scores 0 on his early warning score. His latest blood results are shown below:

Hb	120 g/L	Male: (135-180)
Platelets	454 * 10 /L	(150 - 400)
WBC	10.2 * 10 /L	(4.0 - 11.0)
Na	137 mmol/L	(135 - 145)
K	4.9 mmol/L	(3.5 - 5.0)
Urea	8.7 mmol/L	(2.0 - 7.0)
Creatinine	525 µmol/L	(55 - 120)
CRP	12 mg/L	(< 5)

Given the likely diagnosis and his co-morbidities, what would be the most appropriate treatment to commence?

A. Prednisolone
B. Colchicine
C. Ibuprofen
D. Febuxostat
E. Allopurinol

A. Prednisolone

For gout, if NSAIDs and colchicine are contraindicated or not tolerated the next option is a steroid

The patient is suffering from an acute attack of gout. The classical presentation is the 1st metatarsophalangeal joint being affected.

Whilst colchicine and NSAIDs such as ibuprofen are advocated by NICE as 1st line treatment for acute gout, they are contraindicated in this patient due to the severity of his CKD.

As a result, oral steroids such as prednisolone should be used.

Allopurinol is the 1st line treatment for the prevention of gout but has no benefit in an acute attack. However, if the patient is already on allopurinol, this can be continued throughout the acute flare.

Like allopurinol, febuxostat is a urate-lowering therapy used for the prevention of gout. It is used as a second-line treatment and is particularly useful in renal impairment where allopurinol is contraindicated.

Question 7 of 178

A 65-year-old man presents to an outpatient respiratory clinic with a 3-month history of weight loss, cough, weakness and intermittent haemoptysis. His past medical history includes type 2 diabetes and hypertension. He is on regular amlodipine, ramipril and metformin. He has smoked 15 cigarettes daily for approximately 40 years. He denies alcohol or recreational drug use.

On clinical examination, he appears underweight. His observations demonstrate a heart rate of 87 beats per minute, blood pressure 145/82 mmHg, respiratory rate 15/minute, oxygen saturations of 97% on room air and temperature of 36.7ºC. Chest auscultation reveals a monophonic wheeze in the left upper lobe. His heart sounds are normal and there are no murmurs or peripheral oedema. There is no evidence of lymphadenopathy or organomegaly. His fingers are clubbed. Power is 3+/5 proximally in the upper and lower limbs. He finds it difficult to get up off his chair. Sensation is preserved, reflexes are normal and plantar reflexes are downgoing. There is no rash.

Blood tests:

Hb	111 g/L	Male: (135-180)Female: (115 - 160)
Platelets	444 * 10 /L	(150 - 400)
WBC	8.4 * 10 /L	(4.0 - 11.0)
Na	129 mmol/L	(135 - 145)
K	4 mmol/L	(3.5 - 5.0)
Urea	8.1 mmol/L	(2.0 - 7.0)

Creatinine	111 µmol/L	(55 - 120)
CRP	8 mg/L	(< 5)
Creatine kinase	5891 U/L	(40-320)
TSH	5.9 miU/L	(0.2 - 5.5)
Free T4	11.1pmol/L	(10 - 24.5)

A chest x-ray demonstrates a coin lesion in the upper zone of the left lung.

What is the most appropriate initial treatment

A. IV sodium bicarbonate
B. Intravenous fluids
C. Intravenous immunoglobulin
D. Levothyroxine
E. Prednisolone

E. Prednisolone

Malignancy + raised CK → ?polymyositis

Prednisolone is correct. The history suggests a new diagnosis of lung cancer complicated by a cancer-associated myositis, given the significantly elevated creatine kinase and proximal weakness. The dose of prednisolone used to treat myositis is usually in the region of 1mg/kg with a slow taper over months. The co-existing malignancy will also need to be treated by usual means e.g. surgery, chemotherapy or radiotherapy depending on stage and grade.

IV sodium bicarbonate is incorrect. This is sometimes used to prevent acute kidney injury and treat metabolic acidosis in cases of rhabdomyolysis. However, given the clinical context of malignancy, raised creatine kinase and subacute weakness, this is not the correct answer.

Levothyroxine is not the right answer. The patient's thyroid function tests demonstrate mild subclinical hypothyroidism. Severe hypothyroidism can sometimes masquerade as a myositis but these results are not dramatic enough to give this picture.

Intravenous fluids are incorrect. This would be a sensible treatment if the diagnosis was rhabdomyolysis.

Intravenous immunoglobulin is not the right answer. This is a second-line treatment for those with refractory myositis.

Question 8 of 178

A 22-year-old woman presents to the emergency department with two weeks of sore throat, fever, rash and joint pains. She has no past medical history and does not take any regular medications. She does not smoke or drink alcohol. She has a family history of rheumatoid arthritis.

Observations:

- Heart rate 89 beats per minute
- Respiratory rate 19/minute
- Oxygen saturations 96% on room air
- Temperature 39.1
- Blood pressure 124/77 mmHg

On examination, there is synovitis affecting the wrists bilaterally. There is a salmon-pink rash on her chest. There is evidence of pharyngitis. The examination is otherwise normal.

Blood tests:

Hb	138 g/L	Male: (135-180)Female: (115 - 160)
Platelets	422 * 10 /L $_9$	(150 - 400)
WBC	17.1 * 10 /L $_9$	(4.0 - 11.0)
Na $_+$	136 mmol/L	(135 - 145)
K $_+$	4.2 mmol/L	(3.5 - 5.0)
Urea	5.4 mmol/L	(2.0 - 7.0)
Creatinine	101 µmol/L	(55 - 120)
Bilirubin	12 µmol/L	(3 - 17)
ALP	89 u/L	(30 - 100)
ALT	64 u/L	(3 - 40)
Î³GT	82 u/L	(8 - 60)
Albumin	36 g/L	(35 - 50)
CRP	52 mg/L	(<5)
Ferritin	2240 ng/mL	(20-250)

Further testing:

Rheumatoid factor	negative
Antinuclear antibody	negative
Blood cultures	negative

Plain radiography of the chest is unremarkable.

What is the most appropriate management?

A. Anakinra
B. Corticosteroids
C. Methotrexate
D. NSAIDs
E. Antibiotics

D. NSAIDs

NSAIDs are the first line treatment for Still's disease, not steroids

NSAIDs is the correct answer. This patient has Still's disease as evidenced by fever, characteristic rash, arthritis, deranged LFTs, hyperferritinaemia and negative testing for (antinuclear antibody) ANA and rheumatoid factor. She has no life-threatening organ dysfunction and therefore the initial management is with non-steroidal anti-inflammatory medications.

Corticosteroids is incorrect. This class of medication can be used if the condition is refractory to NSAID treatment or if there is more severe disease manifestations and organ dysfunction.

Methotrexate is incorrect. If the patient needs maintenance disease-modifying therapy, then this would be an option. However, it is not used to induce remission.

Antibiotics are incorrect. While the patient has a fever and raised inflammatory markers, the clinical picture is that of Still's disease, rather than infection.

Anakinra is incorrect. This is an interleukin (IL)-1 inhibitor used in the treatment of a variety of inflammatory conditions including

Still's disease. It is used for severe cases that are refractory to initial treatment.

Question 9 of 178

A 62-year-old lady with idiopathic Raynaud's comes to clinic concerned regarding painful, 'blue' fingers and areas that appear to be consistent with the beginning of ulceration. She has tried numerous over the counter medications, heating techniques and calcium channel blockers with no improvement. She is also known to suffer from hypertension for which she is on Losartan.

On examination you note prominent acrocyanosis with areas of skin discolouration and moderate digital ulceration. There is no sclerodactyly or telangiectasiae. Her blood pressure is: 105/60 mmHg.

What medication could be offered to this lady to help with her symptoms?

A. Epoprostenol
B. Diltiazem
C. Doxazosin
D. Sertraline
E. Amitriptyline

A. Epoprostenol

Epoprostenol (amongst other prostaglandins) can be used in the treatment of Raynaud's phenomenon

In Raynaud's phenomenon (whether idiopathic or not) optimisation of vasodilation is important. This is a lady however who has not tolerated the use of CCB. Given the presence of digital ulceration, sildenafil is the first line option for treatment. As this is not offered as a choice the correct decision would be the administration of IV prostanoids in the form of epoprostenol.

Question 10 of 178

A 69-year-old man attends the medical unit with an acutely hot and swollen joint. The swelling is limited to the first metatarsophalangeal joint **(MTPJ)** and is causing him severe discomfort. He states that he has had this problem before and had it successfully treated, but the medicine used caused him to have severe diarrhoea.

On examination, their first MTPJ is swollen, red and very tender to touch. There is limited movement and when they try to walk, it causes severe pain.

Their past medical history includes chronic kidney disease, gout, osteoarthritis and angina. Blood tests taken at the time of admission:

Hb	140 g/L	Male: (135-180)Female: (115 - 160)
Platelets	300* 10 /L	(150 - 400)
WBC	10.4 * 10 /L	(4.0 - 11.0)
Na	138 mmol/L	(135 - 145)
K	4.8 mmol/L	(3.5 - 5.0)
Urea	14 mmol/L	(2.0 - 7.0)
Creatinine	230 µmol/L	(55 - 120)
CRP	32 mg/L	(< 5)

You suspect the diagnosis is an acute flare of gout. What is the most appropriate treatment?

A. Colchicine
B. Febuxostat
C. IM steroid
D. Ibuprofen
E. Oral prednisolone

E. Oral prednisolone

For gout, if NSAIDs and colchicine are contraindicated or not tolerated the next option is a steroid

The answer is oral prednisolone as the most appropriate treatment for this man. He has gout affecting one small joint with relative contraindications to NSAIDS and colchicine (asthma, previous intolerance of colchicine and renal disease).

Febuxostat is not an appropriate choice as this a gout prophylaxis medication.

An alternative treatment would be intra-articular injection of steroid in this case but not an IM steroid injection.

Question 11 of 178

A 25-year-old woman is admitted to the hospital with recurrent fevers. She also complains of joint pain. There is no recent history of foreign travel, and she denies any recreational drug use. Observations include a temperature of 39.2ºC, pulse rate of 92, blood pressure of 124/72 mmHg, respiratory rate of 18/min, and oxygen saturation of 97% on room air. On examination, there is a salmon-pink rash on her trunk that feels bumpy to the touch. There is also mild cervical lymphadenopathy. Blood results are as follows:

Hb	129 g/L	Male: (135 - 180)Female: (115 - 160)
Platelets	244 * 10 /L	(150 - 400)
WBC	12.2 * 10 /L	(4.0 - 11.0)
Bilirubin	15 µmol/L	(3 - 17)
ALP	120 u/L	(30 - 100)
ALT	50 u/L	(3 - 40)
Î³GT	72 u/L	(8 - 60)
Albumin	38 g/L	(35 - 50)

ESR	35 mm/hr	Men: (<22) Women: (<17)
Prothrombin time (PT)	12 secs	(10-14 secs)
Activated partial thromboplastin time (APTT)	27 secs	(25-35 secs)
Fibrinogen	2 g/L	(2 - 4)
Ferritin	2100 ng/mL	(20 - 230)
Vitamin B12	450 ng/L	(200 - 900)

Folate	4.2 nmol/L	(>3.0)
Reticulocytes	0.7%	(0.5 - 1.5)

What is the most appropriate first-line treatment given the probable diagnosis?

A. Azathioprine
B. Ciprofloxacin
C. Ibuprofen
D. Prednisolone
E. Rituximab

C. Ibuprofen

NSAIDs are the first line treatment for Still's disease, not steroids

This patient is displaying the classic triad of fevers, joint pain, and a salmon-colored bumpy rash. The diagnosis of adult-onset Still's disease (AOSD) is considered a diagnosis of exclusion and is normally a clinical one. The majority of patients will have a very high ferritin level and a high ESR. Derangement in liver function tests is also common. First-line treatment involves NSAIDs such as ibuprofen or naproxen.

Immunosuppressive therapy can then be used to control the disease and induce remission in severe disease. Options include steroids such as prednisolone.

Ciprofloxacin is an antibiotic that can be used to treat several infections including typhoid fever, which can also present with a salmon-pink rash. Typhoid fever is incredibly unlikely given the negative travel history.

Azathioprine is another immunosuppressive commonly used in rheumatoid arthritis, granulomatosis with polyangiitis, and other conditions. It can also be given post-kidney transplant to prevent rejection.

Rituximab is a monoclonal antibody used to treat many autoimmune diseases. It has been used in drug-resistant AOSD.

Question 12 of 178

A 47-year-old man has asked for a review of his right elbow due to pain and a restriction in the range of movement. He denies trauma or a recent injury.

There is no past medical history and he works as a plumber, does not smoke, drinks 35 units of alcohol per week, and is not keen on exercise.

On examination of his elbow, there is no evidence of effusion or swelling. Pain is exacerbated during wrist extension and supination whilst the elbow is extended. There is no motor or sensory deficit.

What is the likely diagnosis?

A. Cubital tunnel syndrome
B. Lateral epicondylitis
C. Medial epicondylitis
D. Olecranon bursitis
E. Triceps tendonitis

B. Lateral epicondylitis

Lateral epicondylitis: worse on resisted wrist extension/suppination whilst elbow extended

Examination findings of pain or weakness being exacerbated when the wrist is extended or supinated during elbow extension are indicative of **lateral epicondylitis**. Also known as tennis elbow, lateral epicondylitis can be caused or exacerbated by repetitive occupational or recreational movements. His career as a plumber is likely to be the cause.

Cubital tunnel syndrome is caused by ulnar nerve entrapment and commonly causes medial elbow pain with paraesthesia in the ulnar nerve distribution making this answer incorrect.

Medial epicondylitis, or golfer's elbow, is also secondary to repetitive occupational or recreational activity. Pain is exacerbated with resisted wrist flexion and pronation, the opposite of lateral epicondylitis.

Olecranon bursitis typically presents as a boggy, non-tender swelling over the olecranon following trauma. This is incorrect as clinical examination showed no evidence of swelling or effusion and there was no history of trauma.

Triceps tendonitis presents as pain in the posterior aspect of the elbow which is exacerbated on resisted extension. There is rarely worsening pain with supination making this answer incorrect.

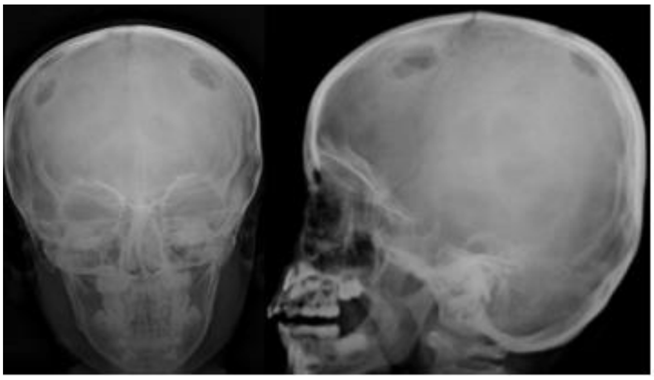
Question 13 of 178

A 7-year-old girl who has recently emigrated from Turkey is brought to the Emergency Department with pain on walking and multiple swellings over her head. She also complains of persistent headaches which are now quite distracting and causing her to miss school. This symptoms have been getting gradually worse for the past few weeks.

She has a past medical history of eczema and asthma which is well controlled with a salbutamol inhaler as required. There is no family history of similar problems.

On examination a number of soft tissue swellings are noted on the scalp. She also has non-specific tenderness over the proximal part of the left femur.

A skull x-ray is requested:



What is the most likely diagnosis?

A. Multiple myeloma
B. Langerhans cell histiocytosis
C. Neutrofibromatosis
D. Systemic mastocytosis
E. Wiskott-Aldrich syndrome

B. Langerhans cell histiocytosis

The age of the patient combined with 'punched out' osteolytic skull lesions make a diagnosis of Langerhans cell histiocytosis likely.

Systemic mastocytosis results from a neoplastic proliferation of mast cells and is associated with urticaria pigmentosa and flushing.

Question 14 of 178

62-year-old woman complains of knee pain. She has struggled with pain for several years. She finds that it gets worse through the day and is relieved by resting. She does not normally come and see doctors but the pain has gotten to the point where she would like additional treatment. She is not keen on surgery as of yet. She is known to have osteoarthritis. She has been taking paracetamol but not tried any other medication.

What is the most appropriate strategy to further help relieve her pain?

A. Codeine
B. Oral NSAIDs
C. Topical NSAIDs
D. Intra-articular steroid injection
E. Oral morphine

C. Topical NSAIDs

The correct answer is topical NSAIDs. This is a case of managing pain in osteoarthritis. As she has

knee osteoarthritis the first line of treatment can include paracetamol and topical NSAIDs. Topical

NSAIDs are only appropriate for osteoarthritis of the hands and knees. Second line treatment

includes oral NSAIDs, codeine, capsaicin cream and intra-articular corticosteroids.

Question 15 of 178

A 72-year-old man presents by blue light ambulance to the emergency department. He has been increasingly drowsy over the past 4 days and has not been out of bed for the past 48 hours. His wife reports two episodes of sweating and a high temperature during this period. He has been admitted three times in the last 9 months with urinary tract infections and is awaiting a transurethral resection of his prostate for benign prostatic hypertrophy. His other past medical history includes type 2 diabetes mellitus, diagnosed 4 years ago, and rheumatoid arthritis diagnosed 16 years ago. His wife tells you that he appears to be prone to infections over the past few years. He normally walks with a stick but his exercise tolerance has been decreasing since doctors told him that he had 'scarring of his lungs from his rheumatoid.' On examination, he is sleepy but easily rousable and orientated to time and place. He is cool peripherally, with dry mucous membranes and JVP +1cm above the angle of Louis. Blood pressure measures 82/55mmHg, heart rate is 105/minute. You note conjunctival pallor, bilateral ulnar deviation of his hands, an inflamed second MTP joint and nodules beneath both elbows. Auscultation of the chest demonstrates bibasal inspiratory fine crackles. Abdominal examination demonstrates a 2cm liver edge and a 13cm spleen. Neurological examination is unremarkable. Routine blood tests and blood cultures are taken.

Na	141 mmol/l
K	3.9 mmol/l
Urea	7.0 mmol/l
Creatinine	81 µmol/l

What is the most appropriate treatment?

A. Oral antibiotics as per local guidelines for mild-moderate community acquired pneumonia
B. Intravenous antibiotics as per local guidelines for severe community acquired pneumonia
C. Intravenous antibiotics as per local guidelines for urosepsis
D. Intravenous antibiotics as per local guidelines for intra-abdominal sepsis
E. Intravenous antibiotic as per local guidelines for neutropenic sepsis

E. Intravenous antibiotic as per local guidelines for neutropenic sepsis

The patient is known to have rheumatoid arthritis with multiple extra-articular features. He is also clinically septic and intravascularly dehydrated. The proneness to infections must raise suspicions of neutropenia, combining with RA with extra-articular features, splenomegaly to produce the classic triad of Felty's syndrome. Although he has had multiple episodes of previous UTIs, the history is unclear here. Neutropenic sepsis without a clear source of infection must be treated with intravenous

antibiotics as per local guidelines.

Question 16 of 178

A 62-year-old lady is seen in the rheumatology clinic. She was diagnosed with rheumatoid arthritis 16 years ago. Her symptoms were relatively well controlled with a combination of methotrexate 20mg once per week, folic acid 5mg once per week and azathioprine 100mg once per day until the last few months when she complained of increasing joint pain with stiffness. Since then her methotrexate dose was gradually titrated to the current dose of 25mg per week. She reported that her joints were less painful and stiff in the morning. Unfortunately, she was also complained of increasing tiredness with an increasing quantity of respiratory tract infections, requiring antibiotics twice in the last six months. She also noted that she bruised more easily of late.

Examination revealed a slender 62-year-old systemically well lady. She was haemodynamically normal and afebrile. Cardiovascular and respiratory examinations were unremarkable, and abdominal examination revealed a mass arising from the left upper quadrant.

Clinical examination of her joints revealed no evidence of synovitis or swelling.

Routine blood investigations prior to attending clinic were as follows:

Hb	115 g/l
MCV	84 fl
Platelets	82 * 10 /l
WBC	3.5 * 10 /l
Neutrophils	1.6 * 10 /l
Lymphocytes	1.0 * 10 /l
Eosinophils	0.9 * 10 /l

Bilirubin	12 µmol/l
ALP	99 u/l
ALT	13 u/l
Albumin	39 g/l

What is the single most likely cause of the clinical and haematological abnormalities?

A. Myelodysplastic syndrome
B. Chronic lymphocytic leukaemia
C. Marrow aplasia secondary to drug therapy
D. Felty's syndrome
E. Myelodysplasia

D. Felty's syndrome

Felty's syndrome is a complication of Rheumatoid Arthritis (RA). It consists of a combination of rheumatoid arthritis, neutropaenia and splenomegaly, and tends to affect RA of longstanding duration. The main differential diagnosis is drug-induced marrow aplasia; however, this would not easily account for the presence of splenomegaly.

Question 17 of 178

A 74-year-old female was admitted to the medical ward initially for the treatment of a CURB = 4 community-acquired pneumonia. She is now awaiting discharge but since her illness, she has not returned to her pre-morbid state. Her past medical history includes two previous myocardial infarctions, hypertension, type 2 diabetes mellitus, duodenal ulcer and obesity. In addition, the physiotherapists report significant right knee pain to be contributing to poor mobility. On questioning, the patient reports that the pain is chronic and has been progressively worsening for about 3 years.

Her GP had sent her for two X-rays previously that demonstrated cartilage loss and osteophyte formation, with a reduction in joint space. On examination, you note significant crepitus in the right knee, with reduced range of movements in flexion and extension. You also note bony outgrowths in the proximal interphalangeal joints of her second and third digits of her right hand. She had successfully lost 9kg in weight and had previously taken 1g paracetamol four times a day regularly but neither measure seemed to help her pain.

What is the most appropriate next step?

A. Increase 500mg paracetamol as required
B. Oral ibuprofen
C. Topical diclofenac
D. Oramorph as required
E. Glucosamine

C. Topical diclofenac

The patient describes osteoarthritic symptoms that have persisted despite non-pharmacological therapies (weight loss) and paracetamol on a regular basis. Her past medical history of a duodenal ulcer should make you wary of oral NSAIDs while her previous MIs indicate selective COX-2 inhibitors should be used with caution. Opioids are not the second line therapies for osteoarthritis. The evidence for glucosamine is limited and is not recommended for use in the NHS. Topical NSAIDs such as diclofenac or topical capsaicin as an adjunct are reasonable options in this setting. A 3rd line possibility if topical NSAIDs are not efficacious and in this case, where oral NSAIDs are contraindicated, are intraarticular steroid injections, which have been demonstrated to produce significant symptomatic improvements in the knee joint when compared to placebo (although evidence is weaker for other joints). Interestingly, an inflammatory element is not required fo

Question 18 of 178

A 32-year-old man presents with a several-month history of recurrent ulcers. He experiences oral ulcers regularly, multiple at a time, which resolve after a week. He has also been experiencing genital ulcers. These are not painful. His past medical history includes two recent attendances to the eye casualty department; both times he was diagnosed as having uveitis.

Examination today is unremarkable except for a number of oral ulcers inside the mouth and some genital ulcers.

What would support the likely diagnosis?

A. Antinuclear antibodies
B. Culture and serology of ulcers
C. HLA-B27
D. Pathergy test
E. Rheumatoid factor

D. Pathergy test

A positive pathergy test is suggestive of Behcet's syndrome

The history and examination here are suggestive of a diagnosis of Behcet's syndrome. This is a vasculitis that causes mucocutaneous, ophthalmological, vascular, gastrointestinal, and neurological manifestations. It is typically seen in patients aged 20-30 years, particularly in men. The classic presentation is that of oral and genital ulcers, with previous or current uveitis. There is no single test to accurately diagnose the condition; it is a clinical diagnosis. However, of the options listed, a **pathergy** test can support the diagnosis. This involves pricking the skin with a sterile needle, usually in the forearm. If the lesion becomes indurated within 48 hours, this is supportive of a diagnosis of Behcet's. The test is positive in approximately 60% of patients.

Antinuclear antibodies are not expected to be positive in Behcet's syndrome and so would not support the diagnosis. A positive result should prompt investigation of other autoimmune causes of the symptoms.

Culture and serology of the ulcers would not support a diagnosis of Behcet's. They would be expected to be negative; any positive results should prompt reconsideration of the diagnosis.

HLA-B27 would not be expected to be positive in Behcet's. A positive result would point towards seronegative spondyloarthropathies and would warrant further investigation.

Rheumatoid factor is not expected to be positive with Behcet's syndrome. If joint symptoms are also present, it can be useful to rule out rheumatoid arthritis but does not explicitly support the diagnosis of Behcet's.

Question 19 of 178

A 35-year-old man presents to his general practitioner with right elbow pain that has been present for 2 days and is exacerbated by movement. No other joints are affected and he denies stiffness. There is no past medical history of note and he is normally fit and well and plays sports regularly.

On examination, there is tenderness over the right elbow that is more prominent over the lateral aspect. There is no effusion or overlying skin changes.

What other examination finding best supports the likely diagnosis?

A. Pain on resisted finger abduction
B. Pain on resisted forearm extension
C. Pain on resisted forearm flexion
D. Pain on resisted wrist extension
E. Pain on resisted wrist flexion

A. Pain on resisted wrist extension

Lateral epicondylitis: worse on resisted wrist extension/suppination whilst elbow extended

This patient has lateral epicondylitis (tennis elbow) given the history of lateral elbow pain in a young sports player. The lateral epicondyle is an attachment point for the extensors of the wrist joint. Therefore, **pain on resisted wrist extension** is a diagnostic indicator.

Pain on resisted finger abduction tests the intrinsic hand muscles that would not be implicated in lateral epicondylitis.

Pain on resisted forearm extension tests the triceps muscles that attach from the scapula to the olecranon process of the elbow. Injury to the triceps muscle would not cause lateral epicondylitis.

Pain on resisted forearm flexion tests the biceps brachii and brachialis muscles that play no role in lateral epicondylitis.

Pain on resisted wrist flexion is a sign of medial epicondylitis (golfer's elbow) which is associate with pain over the medial epicondyle secondary to a strain of the flexor compartment of the forearm.

Question 20 of 178

A 32-year-old gentleman presented to his GP with an 8-week history of debilitating pain in his oral cavity and in his groin area. He had presented on numerous occasions to his GP with episodes of tiredness and non-specific malaise which each time was put down to non-specific viral illness. Eight months ago he was investigated by a gastroenterologist having presented with bloody diarrhoea and abdominal pain. He was diagnosed with a non-specific colitis of unknown origin which resolved spontaneously. He also suffered a solitary DVT of his left leg 6 years ago which was treated with oral anticoagulation. He smoked 20 cigarettes per day and consumed 20 units of alcohol per week. He was on no regular medication. Upon specific questioning, he denied joint pain or swelling. He also denied the presence of back pain. He was unaware of any family history as he was adopted from birth.

On examination, he appeared pale. His heart rate was 88 and blood pressure 118/78 mmHg. Examination of his cardiovascular system was unremarkable. Examination of his abdomen was likewise unremarkable. Examination of his oral mucosa revealed the presence of multiple aphthous ulceration. Examination of his external genitalia likewise revealed the presence of multiple shallow ulcers within his groin region. Examination of his joints was unremarkable.

Initial investigations revealed the following results:

Hb	139 g/l
Platelets	333 * 10 /l
WBC	5.1 * 10 /l
ESR	22 mm/hr
CRP	28 mg/l
Rheumatoid factor	negative
Anti CCP	negative
ANA	negative
HLA B27	positive

What is the most likely underlying diagnosis?

A. Seronegative arthritis
B. Disseminated gonococcal infection
C. Crohn's disease
D. Coeliac disease
E. Behcet's syndrome

E. Behcet's syndrome

This gentleman presents with a combination of malaise, oral and genital ulceration, colitis and iritis. Of the above options, Behcets syndrome is the only option that unifies this combination. Note that HLA B27 is positive in 10 % of the population in the absence of seronegative arthritis. Crohn's disease may present with a colitis and aphthous ulceration as well as iritis, but it would be difficult to account for the past deep vein thrombosis

Question 21 of 178

A 54-year-old woman attends with shortness of breath and a non-productive cough. She has a past medical history of rheumatoid arthritis. Her drug history is as follows: methotrexate, folic acid, and omeprazole. Observations are as follows: heart rate 95 beats per minute, respiratory rate 20 breaths per minute, blood pressure 140/85 mmHg, temperature 37.1ºC, and SpO2 94% on air.

Blood results are as follows:

Hb	125 g/L	Male: (135-180)Female: (115 - 160)
Platelets	420 * 10 /L	(150 - 400)
WBC	14.2 * 10 /L	(4.0 - 11.0)

A high-resolution CT (HRCT) is performed:

HRCT	Symmetrical and diffusely distributed ground-glass opacities with a basal predominance

What treatment is indicated?

A. Co-amoxiclav and clarithromycin
B. Co-trimoxazole
C. Co-trimoxazole and prednisolone
D. Prednisolone
E. Prednisolone and folinic acid

D. Prednisolone

Methotrexate may cause pneumonitis - typically presents with cough, dyspnoea and fever

Prednisolone is correct. Steroids are the cornerstone of treatment for methotrexate pneumonitis.

Co-amoxiclav and clarithromycin is incorrect. This combination of antibiotics can be used to treat severe community-acquired pneumonia. The radiological appearances, in this case, are more suggestive of pneumonitis rather than pneumonia.

Co-trimoxazole is incorrect. This drug can be used to treat pneumocystis pneumonia (PCP) which is a serious infection caused by the fungus *Pneumocystis jirovecii*. Although the patient is on lowdose methotrexate, this is not particularly immunosuppressive, making PCP a less likely diagnosis.

Co-trimoxazole and prednisolone is incorrect. The addition of steroids to co-trimoxazole can be used for severe cases of PCP characterised by a PaO2 < 8 kPa.

Prednisolone and folinic acid is incorrect. The clinical and radiological features are in keeping with methotrexate pneumonitis which should be managed with high-dose steroids. Indications for folinic acid include bone marrow toxicity which is not present in this case.

Question 22 of 178

A 22-year-old woman presents to her general practitioner with a longstanding history of fatigue and generalised body aches. She was formerly an avid sportsperson, but over the years has become increasingly tired and has twisted her ankles on several occasions. Her past medical history includes recurrent dislocation of her right shoulder.

On examination, she has soft, elastic skin. She demonstrates joint hypermobility in multiple joints.

What ocular feature is also associated with this condition?

A. Angioid streaks
B. Cataracts
C. Hypermetropia
D. Uveitis
E. Yellow sclera

A. Angioid streaks

Ehlers-Danlos syndrome: angioid retinal streaks is a feature

The history and examination are suggestive of Ehlers-Danlos syndrome (EDS), an inherited connective tissue disorder. Typical features include joint and skin hypermobility. Patients may have overlapping features of chronic fatigue syndrome and have a history of recurrent, easy dislocation of joints. Of the ocular features listed here, the only one with a clear association with EDS is **angioid streaks**. These appear as bilateral, narrow, irregular lines deep to the retina, appearing to radiate from the optic disc. These are usually asymptomatic but can lead to choroidal neovascularisation.

There is no clear association between **cataracts** and EDS. There are some conflicting reports on the incidence of cataracts in patients with EDS, but angioid retinal streaks have a much clearer association with the condition.

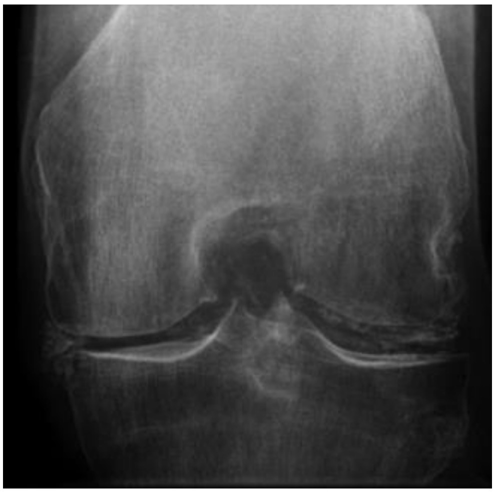
Hypermetropia is not associated with EDS. Rather, patients with EDS appear to have a higher prevalence of myopia than the general population.

Uveitis is not associated with EDS. It is linked to several autoimmune and rheumatological conditions.

Blue sclera, rather than **yellow sclera**, are associated with EDS. This is thought to be due to the loss of collagen in the eye.

Question 23 of 178

The radiograph below was taken from a patient who presented with pain, swelling and erythema of the right knee.



What is the diagnosis?

A. Osteoarthritis
B. Rheumatoid arthritis

C. Pseudogout
D. Gout
E. Tibial plateau fracture

C. Pseudogout

Chondrocalcinosis helps to distinguish pseudogout from gout

The radiograph demonstrates chondrocalcinosis (visible calcification of cartilage), a sign pathognomonic of pseudogout.

Question 24 of 178

A 34-year-old man with no fixed abode attends the emergency department with severe pain in his testicles and joint aches and pains. He has also noticed his left arm has been clumsy and that he has been dragging his left foot.

On examination, his scrotum is swollen and the right testicle is enlarged and extremely tender to touch. Triceps and brachioradialis reflexes are reduced, there is weakness in wrist extension and thumb abduction, with reduced sensation over the posterior forearm, and he has left-sided foot drop. He has purple, mesh-like discolouration of the skin on his lower limbs.

Chest x-ray: clear lung fields

What of the following is most in keeping with the likely diagnosis?

A. Eosinophilia
B. Positive borrelia burgdorferi serology
C. Positive cryoglobulins
D. Positive hepatitis B serology
E. Testicular biopsy showing non-caseating granulomas

D. Positive hepatitis B serology

Systemic vasculitic symptoms in the presence of hepatitis B signs and in the absence of pulmonary symptoms/signs suggests a diagnosis polyarteritis nodosa

The patient has orchitis, arthralgia, livedo reticularis, and mononeuritis multiplex. The most likely diagnosis is polyarteritis nodosa. This is strongly associated with hepatitis B, which is more commonly seen in people with no fixed abode.

Positive hepatitis B serology is correct. 30% of patients with polyarteritis nodosa have positive hepatitis B serology.

Testicular biopsy showing non-caseating granuloma is incorrect. Polyarteritis nodosa is a necrotising vasculitis which does not lead to granuloma formation. Non-caseating granulomas may be seen in other inflammatory conditions such as sarcoidosis and Crohn's. The symptoms

here are not in keeping with a diagnosis of sarcoidosis, there is no lymphadenopathy, no respiratory symptoms, and a chest x-ray has been reported as normal.

Positive *borrelia* burgdorferi serology is incorrect. Lyme disease may be a differential diagnosis, and it would be reasonable to test for it if there was diagnostic uncertainty, but there is no history of tick exposure and there is no characteristic erythema migrans rash.

Eosinophilia is incorrect. Eosinophilia is associated with Eosinophilic granulomatosis with polyangiitis. Symptoms usually include respiratory and nasal problems, but it can be associated with peripheral neuropathy and arthralgia as seen here. It would be extremely unusual for EGPA to cause orchitis, and so it is not the most likely diagnosis here.

Positive cryoglobulins is incorrect. Cryoglobulinaemia is a condition where cryoglobulin proteins clump at lower temperatures, this is usually in the extremities such as fingers and toes. There is usually purpura (which is not present in this case), joint pain, hyperviscosity symptoms, and weakness. Sensory abnormalities are not typical. When it presents with cryoglobulinaemic vasculitis, it is a cutaneous small-vessel vasculitis and so commonly presents with infarctions and necrosis of tissues such as the skin, this is not a feature described in the stem.

Question 25 of 178

A 75-year-old woman presents to the emergency department with a painful swollen wrist. There is no history of trauma. She has recently completed antibiotic treatment for a urinary tract infection. She has a background of gastro-oesophageal reflux disease and is on long-term omeprazole.

Observations:

- Heart rate 88 beats per minute
- Blood pressure 132/77 mmHg
- Respiratory rate 18/minute
- Oxygen saturation 97% on room air
- Temperature 37.3°C

On examination, there is a warm, tender swollen left wrist with a restricted range of motion. Blood tests:

Blood tests:

Hb	121 g/L	Male: (135-180)Female: (115 - 160)
Platelets	189 * 10 /L	(150 - 400)
WBC	8.2 * 10 /L	(4.0 - 11.0)
Na	138 mmol/L	(135 - 145)
K	4.2 mmol/L	(3.5 - 5.0)
Urea	8.9 mmol/L	(2.0 - 7.0)
Creatinine	124 µmol/L	(55 - 120)
CRP	41 mg/L	(< 5)
Magnesium	0.54 mmol/L	(0.7-1.00)

What is most likely to be found on a joint aspirate in this case?

A. Escherichia coli

B. Methicillin-resistant Staphylococcus aureus (MRSA (

C. Staphylococcus aureus

D. Negatively birefringent needle-shaped crystals

E. Positively birefringent rhomboid-shaped crystals

E. Positively birefringent rhomboid-shaped crystals

Pseudogout - weakly positively birefringent rhomboid-shaped crystals

Positively birefringent rhomboid-shaped crystals is correct. This elderly woman presents with monoarthritis of the wrist in close proximity to an infective episode. The most common explanation for this is an episode of pseudogout. Hypomagnesemia (likely caused by omeprazole use) is a risk factor for this condition. An infection should be excluded but is not the most likely explanation. It is possible to have septic arthritis with a normal WCC and temperature. However, the absence of neutrophilia and significant pyrexia does point away from the diagnosis. Pseudogout is characterized by positively birefringent rhomboid-shaped crystals in the synovial fluid.

Methicillin-resistant Staphylococcus aureus (MRSA) is incorrect. This can cause septic arthritis but is an unlikely community-acquired cause of septic arthritis. The CRP may be elevated in both. It would be more likely to occur in a hospital or after an intervention/procedure.

Escherichia coli is incorrect. Intercurrent urinary tract infections increase the probability that septic arthritis may be caused by a gram-negative rather than a gram-positive organism. This is still a rarer event than a flare of pseudogout, however.

Staphylococcus aureus is incorrect. This is the most common organism causing septic arthritis. However, as previously mentioned, a more common cause of monoarthritis of the wrist in this age group, in this context, is pseudogout.

Negatively birefringent needle-shaped crystals is incorrect. This finding is typical of the monosodium urate crystals of gout. A monoarthritis of the wrist is more likely to be pseudogout than gout.

Question 26 of 178

A 34-year-old patient presents to her general practitioner complaining of a rash in both ears. She has no past medical history. She does not take any regular medications. She frequently goes on holiday to hot-weather climates.

On examination, there are red plaques with adherent scales in the concha of both ears.

A skin biopsy demonstrates an interface dermatitis.

Given the likely diagnosis, what is the appropriate treatment?

A. Intravenous belimumab
B. Oral hydroxychloroquine
C. Oral steroids
D. Topical imiquimod
E. Topical steroids

E. Topical steroids

Discoid lupus erythematous - topical steroids → oral hydroxychloroquine

Topical steroids is the right answer. The patient presents with an erythematous rash with an adherent scale in both ears. This is a classic location for discoid lupus erythematosus (DLE) and is known as 'Schuster's sign' when it affects the conchal bowl of both ears. A biopsy demonstrating interface dermatitis is typical. The first-line treatment of discoid lupus is topical steroids.

Intravenous belimumab is incorrect. This is indicated as add-on therapy for those patients with systemic lupus erythematosus with severe disease not responding to conventional therapy. It is particularly indicated for those with active musculoskeletal and cutaneous (which may involve DLE) disease. It is not indicated for isolated DLE as a first-line therapy.

Oral hydroxychloroquine is incorrect. Topical steroids should be tried in the first instance. If this strategy is ineffective then oral hydroxychloroquine can be commenced. It should be noted that if discoid lupus was a manifestation of systemic lupus erythematosus then hydroxychloroquine should be commenced. Discoid lupus erythematosus can exist as an isolated skin disease with a negative antinuclear antibody (i.e. not as part of SLE) and in this setting, topical steroids alone are a reasonable first option.

Oral steroids is incorrect. Oral corticosteroids are associated with significant toxicity with longterm use and therefore are reserved only for severe or refractory cases.

Topical imiquimod is incorrect. This is an immunomodulator that is used in the treatment of genital warts, superficial basal cell carcinomas, and actinic keratoses. The description of this bilateral auricular conchal rash is not typical of a basal cell carcinoma (BCC), which is usually described as a lesion having pearly rolled edges. Actinic (solar) keratoses can cause scaly red plaques although they are more typically yellow. They tend to occur as a consequence of sun damage over time and 34 would be quite a young age to develop this type of lesion.

Question 27 of 178

A 47-year-old lady presents to her General Practitioner complaining of episodes of joint swelling, morning stiffness, weight loss, myalgia and general malaise. She is wondering if she has hyperthyroidism, as both her grandmother and her mother required thyroidectomy when they were younger.

Examination reveals a temperature of 37.1ºC and a mild erythema around the nose, but sparing the nasolabial folds. There is no proximal myalgia and cardiovascular and respiratory examination is unremarkable. There is no goitre.

Blood investigations come back and the raised ESR prompts performance of an autoimmune profile:

Rheumatoid Factor	Positive
Anti-CCP	Negative
ANA	1:320
ANCA	Negative
Anti-DsDNA	Positive
C3/C4 levels	Normal
Anti-RNP	Positive

TSH	4.5 miU/l
T4	12 pmol/l

Given the above results what is the most likely diagnosis?

A. Palindromic Rheumatoid Arthritis
B. Polymyalgia rheumatica
C. Systemic lupus erythematosus
D. Hyperthyroidism
E. Mixed Connective Tissue Disorder

C. Systemic lupus erythematosus

Anti-RNP antibodies can be positive in systemic lupus erythematosus in nearly 40% of patients

This lady has an array of positive antibodies associated with rheumatological conditions. It is important however here to pay attention to the actual symptomatology displayed by the patient and her presenting complaints. The gender, age and history lean towards the diagnosis of a systemic autoimmune condition, with Systemic lupus erythematosus appearing as the most plausible explanation, given her examination findings. The fleeting joint swelling is common in SLE, alongside the non-specific symptoms and the characteristic butterfly rash. The possibility of palindromic rheumatoid arthritis could explain this fleeting arthralgia but does not account for the rest of the signs observed. Polymyalgia rheumatica would be rare in the patient's age and would not exhibit the findings seen. Mixed Connective Tissue Disorder possibility is raised by the presence of anti-RNP antibodies, but one should bear in mind the absence of other overlap features and that anti-RNP antibodies can be found in up to 40% of patients with SLE. Finally Hyperthyroidism is purely a red herring, with patients commonly presenting in primary care with a fixed belief based on prior experiences, whilst actually the pathological process is different.

Question 28 of 178

A 76-year-old lady is seen in rheumatology outpatient clinic, as she was previously diagnosed with Giant Cell Arteritis 4 years ago. Unfortunately since then she has remained on prednisolone 8 mg, and seems to be unable to go on a lower dose; previous attempts (x3) have resulted in excruciating temporal headaches, weakness and fatigue. The most recent attempt was accompanied with mild visual loss, which required her hospitalisation and caused her significant distress.

She is also on zoledronic acid for osteoporosis, as her most recent DEXA scan indicated a T-score on the neck of femur of -2.8. A recent spinal x-ray also revealed a number of vertebral fractures.

She comes wondering if steroid will continue to be her treatment, as she is quite concerned about their side effects, including diabetes (she tells you her sister recently lost 2 toes from diabetes) and weight gain.

What options exist in the management of this lady's giant cell arteritis?

A. Methotrexate
B. Continue with current dose of prednisolone
C. Attempt a reduction in prednisolone dosage
D. Etanercept
E. Ciclosporin

A. Methotrexate

Methotrexate can be used as a steroid-sparing agent in difficult to control, frequently relapsing giant cell arteritis

This lady has Giant Cell Arteritis that is difficult to manage, ridden with numerous relapses. Even though it is commonly accepted that low dose steroids can continue in elderly patients provided that no side effects have arisen, this is a lady with osteoporosis, who is also having family history of type II diabetes mellitus and is concerned herself about the long-time use of steroid. As per British Society of Rheumatology guidelines an appropriate alternative would include methotrexate, provided of course that she has normal liver function and all her pre-methotrexate testing is normal. Etanercept and ciclosporin, although used in the past in the treatment of difficult to manage GCA, studies have no shown any added benefit from the use of etanercept or ciclosporin in the management of such cases of GCA.

Question 29 of 178

A 38-year-old lady presents to her General Practitioner worried regarding a crusty patch of skin on her nose. The doctor proceeds to examine her and identifies a pearly nodule with shiny borders that is roughly 0.3 cm x 0.3 cm.

She is known to suffer from inflammatory bowel disease and is currently on Azathioprine. Despite advice from her gastroenterologist she is still smoking.

What risk factor in her history can be identified related to her presentation?

A. Female gender
B. Age above 30 years old
C. Inflammatory bowel disease
D. Smoking history
E. Azathioprine use

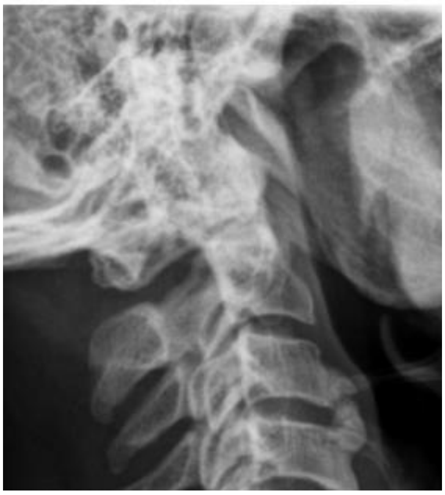
E. Azathioprine use

Azathioprine is associated with increased risk of non-melanoma skin cancer

The lesion described is that of basal cell carcinoma. The only risk factor associated with this is azathioprine

Question 30 of 178

A 78-year-old man has a cervical spine film after falling down the stairs at his house. He has no history of musculoskeletal problems, including no neck or arm pain. The cervical spine film is shown below:



What does the cervical spine film show?

A. Diffuse idiopathic skeletal hyperostosis
B. Multiple myeloma
C. Cervical rib
D. Spondylosis of the cervical spine
E. Ankylosing spondylitis

A. Diffuse idiopathic skeletal hyperostosis

The cervical film shows ossification of the anterior longitudinal ligament only sparing the segments C2/3.

Question 31 of 178

A 31-year-old woman has been referred to your clinic with multiple symptoms. She has reduced sensation in the 4th and 5th digits, extreme fatigue, pain in her hands, and weight loss.

On examination, she has active synovitis in multiple metacarpophalangeal joints and reduced sensation over the 4th and 5th digits of the right hand. She also has a rash over her chest and back with large, ring-shaped lesions that overlap. Her chest is clear and her heart sounds are normal.

Pre-clinic blood tests sent in the community are as below:

Hb	109 g/L	Male: (135-180)Female: (115 - 160)
Platelets	434 * 10 /L	(150 - 400)
WBC	7.4 * 10 /L	(4.0 - 11.0)
Neuts	6.0 * 10 /L	(2.0 - 7.0)
Lymphs	0.3 * 10 /L	(1.0 - 3.5)
Mono	0.7 * 10 /L	(0.2 - 0.8)
Eosin	0.4 * 10 /L	(0.0 - 0.4)
Na	135 mmol/L	(135 - 145)
K	3.6 mmol/L	(3.5 - 5.0)
Urea	3.5 mmol/L	(2.0 - 7.0)
Creatinine	60 µmol/L	(55 - 120)
CRP	43 mg/L	(< 5)

You send some more blood tests and the results are awaited.

Given the likely diagnosis, what would be the most appropriate treatment of choice for long-term management?

A. Azathioprine
B. Cyclophosphamide
C. Hydroxychloroquine
D. Methotrexate
E. Prednisolone with lansoprazole cover

C. Hydroxychloroquine

Hydroxychloroquine is the treatment of choice for SLE

The most likely diagnosis here is systemic lupus erythematosus. Known as the 'great mimic', SLE can present with a vast variety of symptoms. In this case, there is a description of a classic subacute cutaneous lupus rash, joint involvement, peripheral neuropathy, and extreme fatigue. The blood tests show anaemia, thrombophilia, and lymphopenia, all signs of active inflammatory disease. You would also expect a positive ANA.

Hydroxychloroquine is the correct answer. This is the treatment of choice for SLE.

Azathioprine is incorrect. Although this can be used in the management of SLE, it would not be the typical first-choice option. It may be considered if hydroxychloroquine is insufficient in managing non-renal manifestations of lupus after TPMT testing.

Cyclophosphamide is incorrect. This would not be the typical first-line option for long-term management of lupus particularly in this patient who is of childbearing age. It is not typically used long-term due to toxicity. In severe cases of lupus, cyclophosphamide is considered to gain rapid disease control.

Methotrexate is incorrect. Although this can be used in the management of SLE, it would not be the typical first-choice option. It may be considered if hydroxychloroquine is insufficient in managing non-renal manifestations of lupus.

Prednisolone with lansoprazole cover is incorrect. Prednisolone is associated with many complications including gastric ulceration, osteoporosis, and steroid-induced diabetes amongst others. It would not be an appropriate first-line long-term management strategy

Question 32 of 178

A 25-year-old Greek man attends the emergency department with a fever. He states that this has been occurring most evenings for the past two weeks. At the onset of the fever, he also develops a rash and pain in his elbows, knees and ankles.

His observations are as follows: heart rate 110 beats per minute, blood pressure 115/70 mmHg, respiratory rate 16 breaths per minute, temperature 39.2ºC, and SpO2 98% (on air). On examination, you note a maculopapular rash over his back and upper limbs. There is also pain and swelling of his wrists, knees and ankles. There is no evidence of lymphadenopathy or hepatosplenomegaly.

Blood results are as follows:

Hb	145 g/L	Male: (135-180)Female: (115 - 160)
Platelets	580 * 10 /L	(150 - 400)

WBC	14.8 * 10 /L	(4.0 - 11.0)
Na	138 mmol/L	(135 - 145)
K	3.8 mmol/L	(3.5 - 5.0)
Urea	6.8 mmol/L	(2.0 - 7.0)
Creatinine	68 µmol/L	(55 - 120)
CRP	68 mg/L	(< 5)
Ferritin	>20,000 µg/L	(30 - 400)

Bilirubin	10 µmol/L	(3 - 17)
ALP	88 u/L	(30 - 100)
ALT	24 u/L	(3 - 40)
Î³GT	42 u/L	(8 - 60)
Albumin	38 g/L	(35 - 50)

What is the most likely diagnosis?

A. Adult-onset Still's Disease
B. Familial Mediterranean fever (FMF (
C. Hemophagocytic lymphohistiocytosis (HLH)
D. Psoriatic arthritis
E. Rheumatoid arthritis

A. Adult-onset Still's Disease

Pyrexia in Still's disease has a characteristic pattern. It typically rises in the late afternoon/early evening in a daily pattern and accompanies a worsening of joint symptoms and rash

Adult-onset Still's Disease is correct. This condition can be extremely difficult to diagnose due to its rarity and clinical features which overlap with many other conditions. However, there are some clues which can point to this diagnosis including the typical daily fever which occurs in the late afternoon/early evening and accompanies a worsening of joint symptoms and 'salmon-pink' maculopapular rash. An extreme hyperferritinemia >20,000 µg/L is a further clue.

Familial Mediterranean fever (FMF) is incorrect. Familial Mediterranean fever is an inherited condition characterised by recurrent episodes of painful inflammation in the abdomen, chest, or joints. These episodes are often accompanied by fever and sometimes a rash or headache. The absence of abdominal pain makes this a less likely diagnosis. Approximately 90 percent of individuals with FMF experience abdominal symptoms that can range from mild bloating to peritonitis. Although this patient is from the Mediterranean, the overall features are more in keeping with adult-onset Still's Disease.

Hemophagocytic lymphohistiocytosis (HLH) is incorrect. HLH is a condition in which the body makes too many activated immune cells (macrophages and lymphocytes). Clinical features include fever, hepatosplenomegaly, cytopenias, hepatitis and neurological abnormalities. Although this remains within the differential diagnosis (especially with such a high ferritin level), the absence of cytopenias and organomegaly make this a less likely diagnosis.

Psoriatic arthritis is incorrect. The rash described in this case is not typical of psoriasis. Psoriasis is characterised by clearly defined, red and scaly plaques.

Rheumatoid arthritis is incorrect. Rheumatoid nodules, accelerated nodulosis, rheumatoid vasculitis, and neutrophilic dermatosis are some of the classic lesions found in rheumatoid arthritis. The maculopapular rash in this case is not typical of rheumatoid arthritis.

Question 33 of 178

A 52-year-old woman attends with progressive shortness of breath on exertion. On examination, you note fine bibasal inspiratory crepitations and thickening of the skin of her proximal limbs. Observations are as follows: heart rate 95 beats per minute, respiratory rate 22 breaths per minute, blood pressure 155/90 mmHg, temperature 37.5ºC, and oxygen saturation 94% on air.

A high-resolution CT (HRCT) is performed:

HRCT	Ground glass changes present in the lower lobes

What antibody is associated with the development of severe interstitial lung disease?

A. ANA
B. Anti-Jo-1
C. Anti-centromere antibodies
D. Anti-scl-70
E. Rheumatoid factor

D. Anti-scl-70

Anti-scl-70 antibodies are associated with a higher risk of severe interstitial lung disease in systemic sclerosis

Anti-scl-70 is correct. The clinical presentation is of diffuse cutaneous systemic sclerosis with interstitial lung disease (ILD). The most specific antibody for this subtype is anti Scl-70. This antibody is also associated with a higher risk of severe ILD.

ANA is incorrect. Although ANA is most often positive (>90%) it is not specific to diffuse cutaneous systemic sclerosis. Other conditions associated with a positive ANA include systemic lupus erythematosus (SLE), rheumatoid arthritis (RA), Sjogren's syndrome, Addison Disease, and autoimmune hepatitis. A positive ANA is not directly related to the development of ILD.

Anti-Jo-1 is incorrect. This antibody is relatively specific to polymyositis and dermatomyositis. In these conditions, there are higher rates of ILD associated with a positive anti-Jo-1. However, the clinical features in this case are more suggestive of systemic sclerosis.

Anti-centromere antibodies is incorrect. Anti-centromere antibodies are associated with limited cutaneous systemic sclerosis which is characterised by skin thickening distal to the elbows, distal to the knees, and/or face without trunk involvement. Interstitial lung disease is not common in the limited subtype.

Rheumatoid factor is incorrect. Rheumatoid factor is positive in approximately 30% of cases of systemic sclerosis, however, it is not specific to this condition. The development of ILD is not linked to a positive rheumatoid factor result.

Question 34 of 178

A 65-year-old man presents to the emergency department with visual loss in his right eye since he woke up. He states he has felt very run down over the last week with fatigue and a headache.

He is still a working farmer and has been struggling recently as two of his employees are off sick. On questioning, he complains of aching shoulders over the last 2 weeks, particularly in the morning, which he thinks may be from an increased workload.

On examination, vision in the right eye is reduced to finger counting. Vision in his left eye is 18/20.

What is the most likely fundoscopy finding on examination of his right eye?

A. Increased vertical cup-to-disc ratio
B. Multiple abnormal vessel formation with haemorrhage
C. Retinal detachment
D. Swollen pale disc and blurred margins
E. Venous tortuosity and flame-shaped haemorrhages

D. Swollen pale disc and blurred margins

Anterior ischemic optic neuropathy - fundoscopy typically shows a swollen pale disc and blurred margins

This patient has polymyalgia rheumatica (PMR), which classically presents with symmetrical pain and stiffness around the shoulders (most common), hips or neck. Symptoms are often worse in the morning. PMR is associated with temporal arteritis.

Symptoms of temporal arteritis (also known as giant cell arteritis) are constitutional upset (e.g. fatigue, fever), headache, jaw claudication and ocular involvement. This can cause permanent visual loss, of which the majority of cases are due to anterior ischaemic optic neuropathy. This occurs due to the arteritis reducing the perfusion to the optic nerve.

Swollen pale disc and blurred margins is the appearance of anterior ischemic optic neuropathy on fundoscopy, so this is the correct answer.

An **increased vertical cup-to-disc ratio** is classic for glaucoma. In addition, glaucoma would not explain the patient's other symptoms.

Multiple abnormal vessel formation with haemorrhage is typical of proliferative diabetic retinopathy where vascular endothelial growth factor (VEGF) drives the formation of new abnormal vessels.

Retinal detachment occurs following a retinal tear. This finding is not associated with temporal arteritis or anterior ischemic optic neuropathy.

Venous tortuosity and flame-shaped haemorrhages is associated with central retinal vein occlusion. Changes in temporal arteritis are largely arterial.

Question 35 of 178

An 81-year-old woman is being seen in an oncology clinic. She has been diagnosed with cancer of unknown primary with bone metastases and is currently awaiting further investigation. She has a past medical history of deep vein thrombosis and chronic kidney disease. Routine blood tests are performed and show the following:

Hb	97 g/L	(115 - 160)
Platelets	131 * 10 /L 9	(150 - 400)
WBC	4.6 * 10 /L 9	(4.0 - 11.0)
Na +	138 mmol/L	(135 - 145)
K +	4.2 mmol/L	(3.5 - 5.0)
Urea	15.9 mmol/L	(2.0 - 7.0)
Creatinine	161 µmol/L	(55 - 120)
eGFR	24 ml/min/1.73m2	(> 90)
CRP	31 mg/L	(< 5)
Vitamin D	28 ng/mL	(20-50)
Adjusted calcium	2.67 mmol/L	(2.2-2.6)

What treatment is indicated here for bone protection?

A. Alendronic acid
B. Calcichew
C. Colecalciferol
D. Denosumab
E. No treatment required

D. Denosumab

Bisphosphonates and denosumab can be used to prevent pathological fractures in bone metastases. If the eGFR < 30, denosumab is preferred

Bone protection is indicated here due to the presence of bony metastases. This woman has an estimated glomerular filtration rate (eGFR) of approximately 25. As the eGFR is < 30, **denosumab** is preferred for bone protection over bisphosphonates such as alendronic acid.

\ As mentioned, bone protection is indicated here due to the presence of bony metastases however as the eGFR is < 30, denosumab would be preferred to a bisphosphonate such as **alendronic acid**.

This woman is hypercalcaemic as a result of her bony metastases. Therefore, calcium supplements such as **calcichew** are not indicated and therapies that promote osteoblastic activity are more effective for bone protection.

This woman has normal vitamin D levels and hypercalcemia. Therefore, a vitamin D analogue such as **colecalciferol** would not be indicated and therapies that promote osteoblastic activity should be prescribed instead.

As there are bony metastases here, bone protection should be prescribed. Prescribing **no treatment** would therefore be inappropriate and is incorrect.

Question 36 of 178

A 21-year-old man presents to his general practitioner with several months of low back pain. He wakes up with stiffness in the back that persists for more than an hour. There has been no obvious injury. He is a keen sportsman and finds that symptoms improve with exercise. Sitting at his office desk for prolonged periods appears to exacerbate symptoms. There is no significant past medical history except for an episode of uveitis approximately one year ago.

What would be the most appropriate first-line investigation to support the diagnosis?

A. Cervical X-ray

B. HLA-B27

C. MRI spine

D. Pelvic X-ray

E. Rheumatoid factor

D. Pelvic X-ray

Diagnosis of ankylosing spondylitis can be best supported by sacro-ilitis on a pelvic X-ray

This patient's history is strongly suggestive of ankylosing spondylitis, a seronegative spondyloarthropathy. It typically presents in young men; inflammatory back pain is the main clinical feature. The most important diagnostic feature is **pelvic X-ray** - the presence of sacroiliitis is a requirement for fulfilling the New York classification criteria for ankylosing spondylitis.

Whilst a **cervical X-ray** is useful to look for syndesmophytes and the 'bamboo spine', these signs are more uncommon and appear much later in the disease progression. As such, pelvic X-ray remains more useful.

HLA-B27, although associated with the disease in textbooks, is not particularly useful in making the diagnosis. It is not particularly specific, nor sensitive. 10% of patients with ankylosing spondylitis are HLA-B27 negative, and 10% of the healthy population are HLA-B27 positive.

A **spinal MRI** may be useful to demonstrate abnormalities when X-rays have appeared normal. However, it would not be first-line - a pelvic X-ray should be done first.

Ankylosing spondylitis is a seronegative spondyloarthropathy. Thus, testing for **rheumatoid factor** will not diagnose the condition.

Question 37 of 178

A 54-year-old woman attends with worsening shortness of breath. She has also complained of general arthralgia and severe fatigue. Her left knee had previously been red and swollen but improved with an intra-articular steroid injection. She notices around 10 minutes of early morning stiffness and then has generalised joint pains throughout the day that are worsened by overexertion.

She has a past medical history of depression and Raynauds.

On examination, she has fine inspiratory crepitations in the lower zones. She has a reduction in her range of movement when abducting her shoulders with associated pain. The skin on her chest feels thickened. She has white nodules around her right wrist and distal interphalangeal joints.

Pulmonary function tests show:

FEV1 55% of predicted

FVC 65% of predicted FEV1/FVC 0.85

DLCO 60% of predicted

Given the likely diagnosis, what is most likely to be raised in this patient?

A. ANCA
B. Anti-Scl-70 antibodies
C. Anti-centromere antibodies
D. Rheumatoid factor
E. Urate

B. Anti-Scl-70 antibodies

Anti-scl-70 antibodies are associated with a higher risk of severe interstitial lung disease in systemic sclerosis

This patient has shortness of breath with fine crackles and a restrictive pattern on lung function testing. She has scleroderma over her chest and shoulders, as indicated by the thickened skin to palpate and reduction in shoulder abduction. The white nodules on her wrist and fingers are likely calcinosis. The most likely diagnosis here is diffuse cutaneous systemic sclerosis.

Anti-scl-70 antibodies is correct. Anti-scl-70 antibodies are associated with diffuse cutaneous systemic sclerosis. The pattern of disease here - truncal scleroderma, respiratory involvement - is the classical findings in diffuse cutaneous systemic sclerosis rather than the limited type. The antiscl-70 antibodies are associated with a higher risk of severe lung disease.

Anti-centromere antibodies is incorrect. Anti-centromere antibodies are associated with limited cutaneous systemic sclerosis. The pattern of disease here - truncal scleroderma, respiratory involvement - is the classical findings in diffuse cutaneous systemic sclerosis rather than the limited type.

Rheumatoid factor is incorrect. Although 30% of patients diagnosed with systemic sclerosis are rheumatoid factor positive, this patient likely has diffuse cutaneous sclerosis (given the truncal thickened skin and lung involvement). Studies have shown that up to 70% of patients with diffuse systemic sclerosis will have positive anti-scl-70 antibodies, this is, therefore, the better answer. Rheumatoid factor can also be associated with rheumatoid arthritis. Rheumatoid arthritis wouldn't explain the skin changes over her chest and shoulders, and it is less common for rheumatoid arthritis to present with monoarticular large joint effusion and exertional pain. You would expect to see an asymmetrical polyarthritis particularly in the small joints of the hands, with morning stiffness and improvement once 'getting going'.

ANCA is incorrect. ANCA positivity is seen in multiple autoimmune conditions, particularly vasculitis. Although this patient has some arthralgia and previous joint swelling, her other symptoms are not particularly suggestive of vasculitis. ANCA vasculitis can rarely lead to pulmonary vasculitis via progressive small-volume alveolar haemorrhage and scarring caused directly by anti-MPO antibodies, but this is unusual.

Urate is incorrect. Although they can appear similar, the white lesions described would be more in keeping with calcinosis than gouty tophi given the rest of the clinical picture. Gout would not explain the restrictive lung pictures or skin changes across her chest.

Question 38 of 178

A 91-year-old woman is admitted to the hospital with an exacerbation of her chronic lower back pain and general all over body aches. A lumbosacral x-ray showed new wedge compression fractures at L4 and L5.

Bilirubin	12 µmol/L	(3 - 17)
ALP	210 u/L	(30 - 100)
ALT	20 u/L	(3 - 40)
Î³GT	10 u/L	(8 - 60)
Albumin	25 g/L	(35 - 50)

Calcium	1.95 mmol/L	(2.1-2.6)
Phosphate	0.8 mmol/L	(0.8-1.4)
Magnesium	0.81 mmol/L	(0.7-1.0)
Vitamin D	6 nmol/L	(>50)

What is the most appropriate management option?

A. 4.0000 units colecalciferol once weekly for 7 weeks and calcium supplements
B. Give denosumab
C. Start bisphosphonate and calcium supplements
D. Start colecalciferol 400 units with calcium carbonate
E. Start colecalciferol 800 units once daily and calcium supplements

A. 4.0000 units colecalciferol once weekly for 7 weeks and calcium supplements

Osteomalacia is managed using vitamin D supplementation (often with an initial loading dose regime)

This woman has vitamin D deficiency with a level of just 6 nmol/L. Vitamin D levels are sufficient if >50 nmol/L, insufficient if 25-50 nmol/L, and deficient if <25 nmol/L.

40,000 units colecalciferol once weekly for 7 weeks and calcium supplements: NICE recommends rapid replacement for vitamin D deficient patients if they are symptomatic or going to be starting on an anti-reabsorptive agent (e.g. zoledronate, denosumab, or teriparatide). Bone and muscular pains, low mood, fatigue, and weakness are all potential symptoms of vitamin D deficiency. This patient has all over body aches and so warrants high dose loading.

Denosumab: denosumab is not recommended for patients with vitamin D deficiency due to the increased risk of hypocalcaemia.

Start colecalciferol 400 units with calcium carbonate: because this patient is symptomatic and has a vitamin D level <25 nmol/L high dose colecalciferol replacement is recommended in the first instance.

Bisphosphonates and calcium supplements: bisphosphonates should not be started in a vitamin D deficient patient due to the risk of precipitating hypocalcaemia. Vitamin D deficiency also decreases the effectiveness of bisphosphonates in improving bone mineral density.

Start colecalciferol 800 units once daily and calcium supplements: high dose colecalciferol replacement is recommended in the first instance as this patient is symptomatic of vitamin D deficiency.

Question 39 of 178

A 52-year-old lady was seen in the general medicine clinic with aches and pains. The pains were present in her arms and legs, and not associated with her joints. They have been present for several months, and she was unable to identify any precipitating factors. She also felt that on occasion she felt generally weak and tired, though denied the presence of any specific weakness. Her past medical history comprised of epilepsy which was well controlled with phenytoin 500mg BD for several years, as well as hypertension and asthma. In addition to phenytoin 500mg BD she was prescribed ramipril 5mg OD, Clenil modulite 200mcg BD, salmeterol 100mcg BD and Elleste duo for the last six months. Upon specific questioning, she stated that she ate a nutritionally balanced diet, and that she had not suffered a previous fracture. Her mother was diagnosed with osteoporosis when she was 64-years-old, and she did not smoke. She drank 10 units of alcohol per week.

On examination, she was systemically well, with a blood pressure of 132/68 mmHg, heart rate 84, respiratory rate 16/min and body mass index of 23. Examination of her cardiovascular system revealed the presence of normal heart sounds and was unremarkable. Examination of the respiratory and gastrointestinal systems was likewise unremarkable except for the presence of gingival hypertrophy. Examination of the musculoskeletal system revealed the presence of Heberden's nodes but was also otherwise unremarkable with a full range of movement in all joints. Examination of the neurological system was normal with a power of 5/5 in all muscle groups and normal sensation, tone and coordination. Cranial nerve and fundoscopy examinations were unremarkable. Examination of the thyroid gland was unremarkable.

Investigations revealed the following results:

Bilirubin	22 µmol/l
ALP	262 u/l
ALT	23 u/l
Albumin	42 g/l
Protein	76 g/l
Globulin	34 g/l
Adjusted calcium	2.06 mmol/l
Phosphate	0.78 mmol/l
Vitamin D level	pending result
Parathyroid hormone	88 (NR 11-54 pg/ml)
IgG	11.2 g/L (NR 7.0 18.0)
IgA	3.2 g/L(NR 0.8 4.0)
IgM	2.1 g/L (NR 0.4 2.5)

Urinary Bence Jones Protein: negative

What is the most likely underlying diagnosis?

A. Fibromyalgia
B. Paget's disease
C. Primary hyperparathyroidism
D. Osteoporosis
E. Osteomalacia

E. Osteomalacia

Bone pain, tenderness and proximal myopathy (→ waddling gait) → ?osteomalacia

Phenytoin is known to affect the metabolism of vitamin D, and over prolonged periods of time may result in osteomalacia. This patient is also likely suffering other sequelae of prolonged phenytoin use as manifested by the presence of gingival hypertrophy. She has a raised parathyroid hormone level secondary to the low vitamin D level, and there is no evidence of osteoporosis.

Question 40 of 178

A 34-year-old woman is reviewed in clinic. She has been suffering from increasing tiredness for the last three months. She has no other symptoms and specifically denies pain, bleeding, weight loss and mood problems. She has a past medical history of asthma but has not needed her blue inhaler in over a year. She has no allergies. Her only regular medication is the oral contraceptive pill. Blood tests show a normal FBC, U&Es, calcium, parathyroid hormone but low vitamin D (32 nmol/L).

What is the most appropriate treatment in regards to her low vitamin D?

A. Dietary advise only
B. Loading dose vitamin D
C. Maintenance dose vitamin D
D. Combined calcium and vitamin D
E. No treatment needed

C. Maintenance dose vitamin D

Osteomalacia is managed using vitamin D supplementation (often with an initial loading dose regime)

This patient has vitamin D insufficiency which may explain her tiredness. This should be managed with maintenance dose vitamin D. Loading dose would be appropriate if her serum level was less than 30 nmol/L. Dietary advice would be appropriate for patients with adequate levels, above 50 nmol/L. As calcium is normal and there is no evidence of low calcium diet there is no need for calcium supplementation.

Vitamin D deficiency versus insufficiency

Description	Serum levels	Treatment
Adequate vitamin D	>50 nmol/L	dietary recommendations
Insufficient vitamin D	30-50 nmol/L	maintenance dose vitamin D
Deficient vitamin D	<30 nmol/L	loading dose vitamin D

Question 41 of 178

A 75-year-old male presents to the emergency department with two episodes of loss of consciousness over the last 48 hours. Both episodes were witnessed by his wife, onset while sitting in his chair at home, without any witnessed limb jerking, urinary incontinence or tongue biting. He denies any chest pain or shortness of breath normally but reports gradually being able to walk increasingly shorter distances, which he attributed to old age. He has no other significant past medical history, lives with his wife and is a lifelong non-smoker. On examination, he has a significant thoracic kyphosis. On flexion of the lower back, the marked distance increased from 15 cm to 18 cm. He also has a poverty of spinal lateral flexion and bilateral spinal rotation. His cardiovascular examination reveals heart sounds I and II with an early diastolic murmur. Respiratory examination reveals fine inspiratory crackles at both apices. His lying and standing blood pressures are unremarkable. A CT head demonstrated only mild microangiopathic disease. The patient is currently comfortable and alert, requesting to go home. He is attached to cardiac telemetry.

What do you expect his ECG to show?

A. Sinus bradycardia
B. Trigeminy
C. Fast atrial fibrillation with ventricular response greater than 100
D. Atrial flutter
E. Bradycardia with 1st degree heart block

E. Bradycardia with 1st degree heart block

Ankylosing spondylitis features - the 'A's

- Apical fibrosis Anterior uveitis
- Aortic regurgitation
- Achilles tendonitis
- AV node block Amyloidosis

The clinical description is of a patient with ankylosing spondylitis, associated with multiple extraarticular features, commonly remembered by MRCP candidates as the As. They classically include atlantoaxial subluxation, arachnoiditis, apical fibrosis, anterior uveitis, aortitis, aortic regurgitation, AV block, amyloidosis, IgA nephropathy, Achilles tendonitis and associations with plantar fasciitis

and inflammatory bowel disease (the last two are a bit of a stretch!). In this case, the patient is Schober's positive with a murmur of aortic regurgitation from aortitis. The most likely cause of these episodes of syncope are cardiac in origin. AV node block results in first-degree heart block on ECG, resulting in symptoms if bradycardia leads to transient cerebral hypoperfusion.

Question 42 of 178

A 71-year-old woman is reviewed in the oncology clinic. She has a past medical history of metastatic breast cancer, hypertension and chronic kidney disease. She is treated with doxorubicin, cyclophosphamide, amlodipine and ramipril. She does not smoke or drink alcohol. She lives with her husband and is a retired nurse.

Examination reveals evidence of bilateral mastectomies but is otherwise unremarkable. Blood tests:

Hb	121 g/L	Male: (135-180)Female: (115 - 160)
Platelets	489 * 10 /L 9	(150 - 400)
WBC	8.2 * 10 /L 9	(4.0 - 11.0)
Na +	136 mmol/L	(135 - 145)
K +	4.2 mmol/L	(3.5 - 5.0)
Urea	21.4 mmol/L	(2.0 - 7.0)
Creatinine	234 µmol/L	(55 - 120)
eGFR	27 mL/min/1.73m2	(>90)
CRP	44 mg/L	(< 5)
Bilirubin	14 µmol/L	(3 - 17)
ALP	189 u/L	(30 - 100)
ALT	28 u/L	(3 - 40)
Î³GT	42 u/L	(8 - 60)
Albumin	31 g/L	(35 - 50)
Calcium	2.32 mmol/L	(2.20-2.6)

A recent staging CT scan demonstrates bony metastatic disease in the vertebral column.

Based on the clinical history, what is the most appropriate medication to prevent pathological fractures?

A. Alendronic acid

B. Denosumab

C. Hormone replacement therapy (HRT)\

D. Teriparatide

E. Zoledronic acid

b. Denosumab

Bisphosphonates and denosumab can be used to prevent pathological fractures in bone metastases. If the eGFR < 30, denosumab is preferred

Denosumab is the correct answer. This can be used to prevent pathological fractures in those patients with bone metastases. It is the preferred option when the estimated glomerular filtration rate is between 20-30, as in this case.

Alendronic acid is incorrect. This is an option for preventing pathological fractures in bone metastases but is contraindicated when the estimated glomerular filtration rate is < 30.

Zoledronic acid is incorrect. This is an option for preventing pathological fractures in bone metastases but is contraindicated when the estimated glomerular filtration rate is < 30.

Teriparatide is incorrect. This medication is contraindicated in the presence of bone metastases.

HRT is incorrect. This is not a medication used to prevent pathological fracture and is associated with increased breast cancer risk.

Question 43 of 178

A 72-year-old woman attends the clinic with widespread joint pain. On examination, she has prominent synovitis of her proximal interphalangeal (PIP) and metacarpophalangeal (MCP) joints of the hands, the wrists, and small joints of the feet including the metatarsophalangeal (MTP) joints.

Blood results are as follows:

Hb	110 g/L	Male: (135-180)Female: (115 - 160)
Platelets	524 * 10 /L 9	(150 - 400)
WBC	12.2 * 10 /L 9	(4.0 - 11.0)
Na +	135 mmol/L	(135 - 145)
K +	3.6 mmol/L	(3.5 - 5.0)
Urea	6.8 mmol/L	(2.0 - 7.0)
Creatinine	92 µmol/L	(55 - 120)
CRP	2 mg/L	(< 5)

Bilirubin	14 µmol/L	(3 - 17)
ALP	34 u/L	(30 - 100)
ALT	22 u/L	(3 - 40)
Î³GT	42 u/L	(8 - 60)
Albumin	34 g/L	(35 - 50)

A decision is made to start her on methotrexate. What monitoring blood tests are required?

A. Full blood count and renal and liver function in 4 weeks

B. Full blood count in 1 week

C. Full blood count, bone profile and renal and liver function in 1 week

D. Full blood count, bone profile and renal and liver function in 4 weeks

A. Full blood count and renal and liver function in 4 weeks

Methotrexate may cause hepatotoxicity - monitor LFTs

Full blood count and renal and liver function in 1 week is correct. Patients who have started methotrexate should have full blood count and renal and liver function tests repeated every 1-2 weeks until therapy stabilised. Thereafter patients should be monitored every 2-3 months.

Full blood count and renal and liver function in 4 weeks is incorrect. The BNF recommends that they are checked every 1-2 weeks until therapy is stabilised.

Full blood count in 1 week is incorrect. The BNF recommends that liver and renal function are also monitored.

Full blood count, bone profile and renal and liver function in 1 week is incorrect. There is no requirement to monitor bone profile.

Full blood count, bone profile and renal and liver function in 4 weeks is incorrect. There is no requirement to monitor bone profile. Furthermore, the BNF recommends that they are checked every 1-2 weeks until therapy is stabilised.

Question 44 of 178

A 72-year-old male from the Dominican Republic presents with a 4-month history of constant bilateral anterior thigh pain and lower limb weakness, starting first on the left before progressing to bilateral symptoms. He reports being an extremely active man prior to these symptoms, continually to work as a landscape gardener until four months ago, and now he is wheelchair and bed-bound. He denies any history of trauma to his hips, previous tuberculosis infection or contact with heavy metals. His thigh pain appears excruciating, described as burning in nature along the anterior aspects of both thighs to the origin of the patella tendon bilaterally. He reports no recent weight loss, pyrexia or skin changes. His past medical history includes hypertension, hypercholesterolaemia, hypothyroidism and type 2 diabetes mellitus (diagnosed 6 years ago).

On examination, the patient has mildly reduced muscle bulk in bilateral hip flexors and bilateral fasciculations in both thighs. Examination of power demonstrates 2+/5 bilaterally in hip flexion 4+/5 hip extension bilaterally and 5/5 all other lower limb movements. Sensory examination revealed reduced sensation to cotton wool in both thighs and distal feet, reduced proprioception in both toes. Reflexes were 2+ at both knee jerks, absent ankle jerks bilaterally and downgoing plantars bilaterally. His blood tests are as follows:

Hb	134 g/l
Platelets	383 * 10 /l
WBC	4.5 * 10 /l

Na	135 mmol/l
K	4.6 mmol/l
Urea	8.6 mmol/l
Creatinine	112 µmol/l
CRP	1 mg/l
Creatine kinase	116 IU/l (50-335)
HbA1c	68 mmol/mol
TSH	2.1 mu/l
Free T4	15.4 nmol/l

An MRI scan was performed of his lumbosacral plexus, demonstrating no appreciable structural lesion. Nerve conduction studies and EMG are awaited.

What is the most optimal treatment?

A. Optimise diabetic control
B. Riluzole
C. Intravenous immunoglobulin
D. Intravenous methylprednisolone
E. Lumbar puncture

A. Optimise diabetic control

The positive symptoms include bilateral proximal myopathy, severe neuropathic pain in thighs and absence of lumbosacral structural lesions, in a HIV negative patient, the diagnosis if most likely diabetic amyotrophy. The onset of proximal pain as a result of a microvascular lumbosacral plexopathy is usually asymmetric before becoming more symmetrical.

The lack of upper motor neurone signs makes this unlikely to be motor neuron disease (and hence requiring riluzole). The involvement of sensory components rules out multifocal motor neuropathy with conduction block, typically responsive to IVIg. The proximal location of the symptoms and prominent neuropathic pain is classical of amyotrophy instead of chronic inflammatory demyelinating polyneuropathy (CIDP), for which intravenous methylprednisolone can be therapeutic.

The best treatment for diabetic amyotrophy is to optimise blood sugar control and symptomatic treatment neuropathic agents. Most patients report at least partial, if not full motor recovery and much-improved pain symptoms.

Question 45 of 178

A 23-year-old comes in with a painful right knee. He denies any specific trauma. He has no past medical history. On examination, he has a red and warm knee with a moderate effusion. His observations show a respiratory rate of 24/min, blood pressure 120/72 mmHg, temperature 37.8ºC. His knee is aspirated which is cloudy in appearance. Laboratory testing shows calcium pyrophosphate crystals and the Gram stain is awaited.

What is the most appropriate treatment plan?

A. S Admit for intravenous antibiotics
B. Home with analgesia
C. Admit awaiting cultures
D. Check ferritin
E. Intra-articular depo-medrone ubmit answer

A. Admit for intravenous antibiotics

Septic arthritis has not been ruled out until there are no organisms seen. The patient should be treated with intravenous antibiotics as soon as the aspirate has been taken and only be discharged once it is confirmed that they do not have septic arthritis.

Although calcium pyrophosphate crystals are most associated with pseudo gout they do not rule out septic arthritis. The ferritin would be raised in septic arthritis so using it as a screen for haemochromatosis would not be advisable in this setting.

Question 46 of 178

A 55-year-old woman was called by an out of hours GP and told to attend the emergency department due to abnormalities found on her blood tests which were taken at her annual well woman's check-up.

Her past medical history includes systemic sclerosis. She uses topical emollients on her hands and has no allergies. She works as a receptionist, is an ex-smoker and denies alcohol consumption. Her blood results are shown below:

Hb	110 g/L	(115 - 160)
Platelets	151 * 10 /L 9	(150 - 400)
WBC	4.5 * 10 /L 9	(4.0 - 11.0)
Na +	137 mmol/L	(135 - 145)
K +	4.2 mmol/L	(3.5 - 5.0)
Urea	11.5 mmol/L	(2.0 - 7.0)
Creatinine	164 µmol/L	(55 - 120)
eGFR	30 ml/min	(>90)

The nurse does her observations and informs you her blood pressure is high. In the right arm, it is 187/95mmHg and in the left arm is 191/94mmHg.

What would be the most appropriate treatment to initiate in this patient?

A. Lisinopril
B. Amlodipine
C. Bendroflumethiazide
D. Doxazosin
E. Labetalol

A. Lisinopril

Renal complications of systemic sclerosis - ACE-inhibitors

Renal complications of system sclerosis often present with hypertension and AKI. ACE inhibitors are used 1st line in this instance, therefore lisinopril is the correct choice. A severe sclerodermic renal crisis may also present with microangiopathic haemolytic anaemia.

Amlodipine is a calcium channel blocker that is used in the management of hypertension. Bendroflumethiazide is a thiazide diuretic that can also be used to manage hypertension. Doxazosin is an α blocker used to treat hypertension.

Labetalol is a β blocker. It is commonly used to acutely lower blood pressure in haemorrhagic strokes.

Question 47 of 178

A 45-year-old female attends the acute medical unit with fever and rigors. Her past medical history includes rheumatoid arthritis. Her regular medicines include methotrexate and sulfasalazine. She was recently treated with trimethoprim for a urinary tract infection.

Blood results are as follows:

Hb	110 g/l	Na +	138 mmol/l
Platelets	94 * 10 /l 9	K +	3.8 mmol/l
WBC	1.2 * 10 /l 9	Urea	7.8 mmol/l
Neuts	0.6 * 10 /l 9	Creatinine	104 µmol/l
Lymphs	0.4 * 10 /l 9	CRP	212 mg/l

What treatment is indicated?

A. Platelet transfusion
B. Folinic acid
C. Folate
D. Sodium bicarbonate
E. Reassurance

B. Folinic acid

Folinic acid is the treatment of choice for methotrexate toxicity

The patient is on methotrexate which is a dihydrofolate reductase antagonist. Trimethoprim is a selective inhibitor of dihydrofolate reductase and is therefore absolutely contraindicated in patients who take methotrexate. The patient has developed bone marrow suppression secondary to methotrexate toxicity, and neutropenic sepsis.

Folinic acid can bypass the effect of methotrexate on dihydrofolate reductase and replete the intracellular supply of folate. The patient should, therefore, receive folinic acid urgently.

A platelet transfusion is generally reserved for cases when the platelet count falls below 50 * 10 /l.

In addition, the patient should be treated for neutropenic sepsis with broad-spectrum antibiotics.

Urinary alkalisation with sodium bicarbonate has been shown to enhance drug excretion, however the evidence is limited.

Question 48 of 178

A 36-year-old woman presents with recurrent oral ulcerations. She also has widespread joint pain and swelling. More recently she has been losing hair on her scalp.

Blood results are as follows:

Hb	110 g/L	Male: (135-180)Female: (115 - 160)
Platelets	92 * 10 /L 9	(150 - 400)
WBC	2.2 * 10 /L 9	(4.0 - 11.0)
Na +	135 mmol/L	(135 - 145)
K +	4.8 mmol/L	(3.5 - 5.0)
Urea	14.8 mmol/L	(2.0 - 7.0)
Creatinine	162 µmol/L	(55 - 120)
CRP	2 mg/L	(< 5)
C3	0.22 g/L	(0.75 - 1.96)
C4	0.04 g/L	(0.14 - 0.54)

What antibody is most likely to be detected?

A. ANA
B. Anti-Smith
C. Anti-U1 RNP
D. Anti-dsDNA
E. Rheumatoid factor

A. ANA

Over 99% of patients with SLE are ANA positive, therefore it is a useful rule out test

ANA is correct. The patient fulfils the diagnostic criteria for systemic lupus erythematosus (SLE). ANA is the most sensitive antibody being positive in >99% of cases.

Anti-Smith is incorrect. Anti-Smith is highly specific for SLE (>99%), but not very sensitive (30%),

Anti-U1 RNP is incorrect. This antibody is neither sensitive nor specific for SLE.

Anti-dsDNA is incorrect. Anti-dsDNA is highly specific to SLE (>99%), but less sensitive (70%).

Rheumatoid factor is incorrect. This antibody is neither sensitive nor specific for SLE.

Question 49 of 178

A 62-year-old man presents to ED with a six-week history of dry cough and worsening breathlessness. On examination his temperature was 38.4ºC and he had fine bibasal crackles on inspiration.

His past medical history was significant for hypertension and psoriatic arthritis for which he had been taking methotrexate for 2 years. Other medications included amlodipine and ramipril. He worked as a farmer and prior to this illness had been quite well.

A chest x-ray showed diffuse ground glass opacities and infiltrates, predominantly affecting the lower lobes.

Hb	142 g/L	Male: (135-180)Female: (115 - 160)
Platelets	161 * 10 /L 9	(150 - 400)
WBC	8.2 * 10 /L 9	(4.0 - 11.0)
Neuts	6.7 * 10 /L 9	(2.0 - 7.0)
Lymphs	1.4 * 10 /L 9	(1.0 - 3.5)
Mono	0.6 * 10 /L 9	(0.2 - 0.8)
Eosin	0.8 * 10 /L 9	(0.0 - 0.4)

What is the likely diagnosis?

A. Hypersensitivity pneumonitis (Farmer's lung)
B. Interstitial lung disease
C. Methotrexate induced pneumonitis
D. Pneumocystis jiroveci pneumonia
E. Viral pneumonitis

C. Methotrexate induced pneumonitis

Methotrexate may cause pneumonitis - typically presents with cough, dyspnoea and fever

This patient has presented with cough, dyspnoea and fever with bibasal fine crackles while on methotrexate. This, combined with the presence of infiltrates on chest x-ray, should raise the suspicion of **methotrexate** pneumonitis.

Hypersensitivity pneumonitis (Farmer's lung) would not be an unreasonable differential diagnosis. However, in acute farmer's lung patients would however present with a short history of fevers, chills, malaise, headache and myalgia alongside breathlessness and dry cough.

Interstitial lung disease would present with a longer history of breathlessness and without fever or infiltrates on chest x-ray. They may also have finger clubbing on examination.

Pneumocystis jiroveci pneumonia would be unusual without significant immunocompromise, such as infection with untreated HIV. It would however present with fever, exertional dyspnoea and dry cough and fine interstitial changes on chest x-ray. Given there is no history of significant immunocompromise, this is less likely than methotrexate-induced pneumonitis.

Viral pneumonitis such as COVID-19 infection is also not an unreasonable differential given the chest x-ray changes but it would be unusual to have a 6-week history and there is no significant upper respiratory tract symptoms or systemic features of rigors or myalgia which you might expect with a viral illness.

Question 50 of 178

A 54-year-old woman with a background of rheumatoid arthritis is being managed with a weekly dose of 15mg of methotrexate. She has presented for regular review and her blood tests indicate that she has had a significant fall in her cell counts. It is thought that she has methotrexate induced bone marrow failure.

Hb	110 g/l
Platelets	135* 10 /l9
WBC	3 * 10 /l9
Neutrophils	1.9*10 /l9

What is the best management option?

A. Folinic acid
B. Ferrous sulphate
C. Folic acid
D. Iron dextran
E. Palifermin

A. Folinic acid

Folinic acid is the treatment of choice for methotrexate toxicity

The recommended treatment for myelosuppression secondary to her methotrexate therapy is with folinic acid rescue therapy.

Palifermin is used for oral mucositis associated with methotrexate treatment for haematological malignancies. Folic acid is used to prevent methotrexate associated side effects. Ferrous sulphate and iron dextran are not indicated in this case as they are used for iron deficiency anaemia.

Question 51 of 178

A 67-year-old Caucasian man presents with progressive deafness and difficulty chewing. He also states that his father and paternal uncles suffered from similar symptoms that required medication. On examination, it is noted that there is frontal bossing. Further investigations find an elevated alkaline phosphatase and a serum calcium at the upper end of the normal range. His other investigations are normal.

What is the best first-line treatment for this man?

A. Calcium supplementation
B. Bisphosphonates
C. Calcium and Vitamin D supplementation
D. Calcitonin
E. Surgery

B. Bisphosphonates

Paget's disease of the bone is treated with bisphosphonates

This gentleman has findings consistent with Paget's disease of the bone given his clinical symptoms, ethnic background, family history and biochemical results. The NICE guidelines recommend bisphosphonates as first-line treatment in symptomatic patients. In asymptomatic patients, watchful waiting may be sufficient initially.

Supplementation is insufficient in this patient given that he is symptomatic.

Surgery may be required if there are complications such as fractures or severe osteoarthritis but not at this stage.

Question 52 of 178

A 28-year-old woman is referred to rheumatology by her general practitioner complaining of a one-year history of painful hands. She describes daily episodes of her hands changing colour and becoming painful, especially in cold weather. Firstly they go white, then blue and then red, which is followed by a resolution of the symptoms.

She denies any history of rash, chest pain, weakness, mouth ulcers, skin thickening, blood clots, fever or joint swelling. She has no significant past medical history and is on no regular medications. She does not smoke, drink alcohol or take any recreational drugs. There is no relevant family history. She works as a teacher. Despite wearing gloves regularly and trying to avoid the freezer aisle in the supermarket, her symptoms have persisted.

On clinical examination, her hands demonstrated a blue-white discolouration and were cool to touch. There was no evidence of digital ulceration or scarring. Her skin was normal and there was no synovitis. There was no clinical evidence of interstitial lung disease or pulmonary hypertension. Her pulses were present in her upper limbs. There was no proximal muscle weakness.

Blood test results:

Antinuclear antibody	negative
Rheumatoid factor	negative
ANCA	negative
Lupus anticoagulant	negative
Anticardiolipin antibody	negative
HIV	negative
Hepatitis B surface antigen	negative
Hepatitis C antibody	negative
Hepatitis B core antibody	negative

Given the likely diagnosis, what is the appropriate initial pharmacological management?

A. Aspirin
B. Intravenous iloprost
C. Nifedipine
D. Prednisolone
E. Sildenafil

C. Nifedipine

Nifedipine is a pharmacological option for Raynaud's phenomenon

Nifedipine is the correct answer. This is the first-line treatment in those with Raynaud's phenomenon where conservative measures have failed. This patient is likely to have primary Raynaud's phenomenon as there is no clinical or serological evidence of a predisposing underlying condition.

Aspirin is not the correct answer as it is not typically indicated in the symptomatic management of Raynaud's phenomenon. If the patient had a condition like anti-phospholipid syndrome, resulting in secondary Raynaud's phenomenon, then there might be a place for aspirin in management of the underlying condition.

Intravenous iloprost is incorrect. This is reserved for severe cases of Raynaud's phenomenon that have not responded to first-line treatment or those cases characterized by digital ischaemia.

Prednisolone is incorrect. This is used to induce remission in many autoimmune and connective tissue diseases. However, there is no suggestion from the clinical vignette that this patient has a serious underlying condition.

Sildenafil is incorrect. This can be used to treat Raynaud's phenomenon but would not be the firstline choice in primary Raynaud's

Question 53 of 178

A 65-year-old male presents to the emergency department with 5 days of haemoptysis, fever and joint pains. He has a past medical history of hypertension and chronic sinusitis. He takes amlodipine. He smokes 10 cigarettes daily and drinks wine 1-2x/week. He has recently returned from a holiday in Goa.

His observations are heart rate 101 beats per minute, blood pressure 167/94 mmHg, oxygen saturations 94%, respiratory rate 21/minute and temperature 37.9ºC.

Chest auscultation reveals scattered crackles and decreased air entry at the right base. There is mild tenderness and swelling at the wrists bilaterally. Abdominal examination is normal. There is no peripheral oedema.

Urinalysis:

Leucocytes	-ve
Nitrites	-ve

Protein	++
Blood	+++
Glucose	-ve

Blood tests:

Hb	124 g/L	Male: (135-180)Female: (115 - 160)
Platelets	189 * 10 /L	(150 - 400)
WBC	8.7 * 10 /L	(4.0 - 11.0)
Neuts	5.2 * 10 /L	(2.0 - 7.0)
Lymphs	3.2 * 10 /L	(1.0 - 3.5)
Mono	0.2 * 10 /L	(0.2 - 0.8)
Eosin	0.1 * 10 /L	(0.0 - 0.4)
Na	129 mmol/L	(135 - 145)
K	4.8 mmol/L	(3.5 - 5.0)
Bicarbonate	21 mmol/L	(22 - 29)
Urea	9.2 mmol/L	(2.0 - 7.0)
Creatinine	241 µmol/L	(55 - 120)

CRP	180 mg/dL	(<5)
Creatinine kinase	100 U/L	(38-174)

A chest x-ray reveals patchy airspace opacification in the lung fields bilaterally.

What is the most likely diagnosis?

A. Churg-Strauss syndrome
B. Dermatomyositis
C. Goodpasture's syndrome
D. Granulomatosis with polyangiitis
E. Systemic lupus erythematosusSubmit answer

D. Granulomatosis with polyangiitis

Renal impairment, respiratory symptoms, joint pain, systemic features → consider ANCA associated vasculitis

Granulomatosis with polyangiitis is the correct answer. This man presents with haemoptysis, fever and polyarthralgia and has potential evidence of glomerulonephritis (haematuria, proteinuria) with an acute kidney injury and likely pulmonary haemorrhage. The background of sinusitis makes this the most likely diagnosis from the options listed.

Goodpasture's syndrome is incorrect. This can cause a pulmonary-renal syndrome but joint and sinus involvement are less likely.

Churg-Strauss syndrome is incorrect. This can cause a pulmonary-renal syndrome but the eosinophil count is normal, essentially excluding this diagnosis.

Systemic lupus erythematosus is incorrect. This is a possible cause of pulmonary-renal syndrome, however, CRP tends to decrease in active SLE (it is a marker of disease activity), pulmonary haemorrhage is relatively rare in this condition and it is unlikely to present with lupus at age 65 in a male patient.

Dermatomyositis is incorrect. There is no rash or muscle weakness, making this an unlikely cause of pulmonary haemorrhage in this case.

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A 77-year-old woman presents to the emergency department with a fall. She tripped on a wet surface in her kitchen while cooking lunch and sustained an injury to her wrist. She has a past medical history of hypertension and takes ramipril. She does not smoke or drink alcohol. She is independent and lives with her husband. She paints in her spare time.

Examination reveals tenderness and swelling at the right wrist and is otherwise unremarkable. Plain radiography of the right wrist demonstrates a Colles fracture.

Blood tests:

Hb	120 g/L	Male: (135-180)Female: (115 - 160)
Platelets	189 * 10 /L 9	(150 - 400)
WBC	4.2 * 10 /L 9	(4.0 - 11.0)
Na +	136 mmol/L	(135 - 145)
K +	4.2 mmol/L	(3.5 - 5.0)
Urea	4.4 mmol/L	(2.0 - 7.0)
Creatinine	89 µmol/L	(55 - 120)
CRP	4 mg/L	(< 5)
Calcium	2.24 mmol/L	(2.20-2.6)
Vitamin D	75 nmol/L	(>50)
ALP	180 IU/L	(44-147)

She is managed conservatively without the need for an operation.

From the list below, what is the most appropriate next step in management?

A. Alendronic acid
B. Bone density (DEXA) scan
C. Denosumab
D. Teriparatide
E. IV Zoledronic acid

A. Alendronic acid

Start alendronate in patients >= 75 years following a fragility fracture, without waiting for a DEXA scan

Alendronic acid is the correct answer. In patients >= 75 years of age who have sustained a fragility fracture, bone protection can be started without the need for a DEXA scan. In the absence of contraindications, first-line management is typically an oral bisphosphonate such as alendronate.

Bone density (DEXA) scan is incorrect. Treatment can be commenced without this investigation given the clinical context.

IV Zoledronic acid is incorrect. IV bisphosphonates are a second line treatment for osteoporosis, typically used if there is an intolerance or contraindication to oral bisphosphonates.

Denosumab is incorrect. This is a second line treatment for osteoporosis, typically used if oral bisphosphonates are not tolerated or contraindicated. It is also a more appropriate choice if the creatinine clearance is < 30ml/minute.

Teriparatide is incorrect. This has strict criteria for usage and is typically only trialled after first-line and second-line treatments for osteoporosis have been used.

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A 45-year-old woman is referred to rheumatology with a new-onset Raynaud's phenomenon. She has no past medical history.

On examination, her fingers are cool and pale. The skin is hard and thick from the hands to the mid-forearms. There are small, dilated blood vessels on her hands and on her face.

Blood test:

anti Scl-70	positive

What is the most common cause of death in this condition?

A. Cardiac arrhythmia
B. Cirrhosis
C. Infection
D. Interstitial lung disease
E. Ischaemic heart disease

D. Interstitial lung disease

The most common cause of death in systemic sclerosis is respiratory involvement: **interstitial lung disease** and **pulmonary arterial hypertension**

Interstitial lung disease is correct. The patient has systemic sclerosis as evidenced by Raynaud's phenomenon, skin hardening, telangiectasias (small, dilated blood vessels near the surface of the skin) and positivity for Scl-70 antibody. The most common causes of mortality in systemic sclerosis generally are interstitial lung disease and pulmonary hypertension. Scl-70 antibody is associated with the development of interstitial lung disease.

Cardiac arrhythmia is incorrect. Arrhythmias may result from conduction system fibrosis in this condition. However, conduction disease is felt to be uncommon overall in this condition and is often asymptomatic and therefore not a significant cause of mortality.

Cirrhosis is incorrect. Primary biliary cirrhosis and autoimmune hepatitis can occur in systemic sclerosis. However, they are relatively rare complications and not associated with the same mortality as ILD in this condition.

Infection is not the right answer. Patients with systemic sclerosis are at higher risk of infectious complications due to structural damage from the disease and the use of immunosuppressive treatment. However, interstitial lung disease still represents a more common cause of mortality in this condition.

Ischaemic heart disease is incorrect. There is an increased rate of ischaemic heart disease in patients with systemic sclerosis compared to the general population. However, there is a less dramatic increase when compared with other autoimmune diseases like systemic lupus erythematosus and rheumatoid arthritis. It is a less common cause of death than ILD.

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A 31-year-old woman with presents with three days of right sided loin pain with two episodes of blood in her urine. She feels unwell and lethargic but denies any fevers or urinary dysuria.

She is usually well and has had two urinary tract infections previously. She is being seen by the anticoagulants clinic due to several miscarriages and is due for review next week. Her maternal aunt has previous renal calculi. She takes no regular medication at present.

On examination, she is tender in the right loin only. Her blood pressure is 180/105 mmHg, heart rate 85/min, respiratory rate 22/min and temperature 37.0ºC.

Na +	138 mmol/l
K +	4.2 mmol/l
Urea	5.6 mmol/l
Creatinine	87 µmol/l
Anti-cardiolipin antibodies	positive

Urine dip	blood +++, Leu -
Ultrasound KUB	no hydronephrosis or renal lesion seen

What is the likely diagnosis?

A. Renal calculus
B. Urinary tract infection
C. Renal cell carcinoma
D. Renal vein thrombosis
E. Tubulointerstitial nephritis

D. Renal vein thrombosis

Loin pain + haematuria in antiphospholipid syndrome -> renal vein thrombus

Patients with antiphospholipid syndrome (APS) are at risk of thromboses and loin pain with haematuria in hypercoagulable states is most likely a renal thrombus. Although not formally diagnosed, she has positive antibodies and multiple miscarriages diagnostic of APS. She could have a renal calculus though this less often has frank haematuria and there are no risk factors for it. A UTI is unlikely given the lack of lower urinary tract symptoms. Tubulointerstitial nephritis often has a rash and the urine dip has leucocytes and eosinophilic casts on microscopy.

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A 67-year-old gentleman suffering from Rheumatoid Arthritis is admitted to hospital with short history of weakness and numbness in the left side of his face. The doctor that assesses him in the emergency department refers him urgently to the stroke unit where a CT head took place that is unremarkable.

The symptoms did not resolve, and as such he was loaded and treated for potential cerebrovascular accident. An MRI was booked alongside a carotid Doppler both which were unremarkable.

You are asked to see him on-call by the nurse informing you that he has been pyrexial for a few days now despite analgesia with normal blood cultures. His urine dip however has consistently had proteinuria. He tells you that before his admission he was troubled by foot drop of the right foot, which has now resolved.

You examine this patient and beside the weakness and loss of sensation associated with the facial and trigeminal nerves, it is otherwise unremarkable. There are no added heart sound on chest auscultation, and no splinter haemorrhages or clubbing. Breathing sounds are vesicular. He is otherwise neurologically intact. No rashes are noted.

You review his bloods

Hb	105 g/l	Na^+	137 mmol/l
Platelets	$468 * 10^9/l$	K^+	4.5 mmol/l
WBC	$10.2 * 10^9/l$	Urea	13.4 mmol/l
Neuts	$7.2 * 10^9/l$	Creatinine	245 µmol/l
Lymphs	$2.1 * 10^9/l$	CRP	265 mg/l
Eosin	$0.6 * 10^9/l$	ESR	88 mm/h
ANA	1:640	ANCA	positive cANCA

What is the most appropriate investigation to verify this patient's cause of symptoms?

A. Renal biopsy
B. CT thorax/abdomen/pelvis
C. Virology screen
D. Transthoracic Echocardiography
E. Lumbar Puncture

A. Renal biopsy

Biopsy (renal, nasal, skin) can be useful in the diagnosis of ANCA associated vasculitis (e.g. glomerulonephritis with polyangiitis)

This patient presents with a history of potential mononeuritis multiplex. The causes of mononeuritis multiplex are numerous; however this history and the blood investigation findings (including raised ESR, CRP, raised cANCA, reduced eGFR and proteinuria) point to the potential of an ANCA associated vasculitis; in this case Granulomatosis with polyangiitis The gold standard of investigation in such cases is biopsy, with renal biopsy being the most appropriate option given the involvement of kidneys in this patient. It is understandable that a doctor could be tricked in identifying an infectious cause / malignant cause (paraneoplastic syndrome) however given the negative blood cultures, lack of examination findings associated with infection and strong indication of a systemic inflammatory response, a renal biopsy could be appropriate.

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You see a 29-year-old patient with a swollen left knee. It has been getting worse for a number of months and is now affecting his ability to work as a postman. The pain and stiffness are worse overnight and in the morning and gets better throughout the day. He denies any trauma or fever. He also has some pain and stiffness in his fingers which is also worse in the morning. His joints are worse after rest.

He is normally fit and well and denies any previous joint problems or psoriasis. However, his father has a history of psoriasis. He says there is no family history of joint problems to his knowledge.

On examination, his knee is red, warm and tender to palpate. There is a moderate knee effusion and flexion is limited due to pain. His distal interphalangeal joints (DIPs) are tender and slightly swollen.

There are no skin rashes. Although his knee is painful the patient looks well and has a heart rate of 80 beats per minute and his temperature is 36.8ºC.

Blood tests reveal a raised CRP at 85 but a negative rheumatoid factor.

What is the most likely diagnosis?

A. Juvenile idiopathic arthritis
B. Seronegative rheumatoid arthritis
C. Psoriatic arthritis
D. Septic arthritis
E. Rheumatoid arthritis

C. Psoriatic arthritis

Psoriatic arthropathy can present before psoriatic skin lesions - a positive family history of psoriasis may point towards this diagnosis

Given the inflammatory pattern of pain and swelling (worse in the morning and after rest), the joints involved (DIPs and knee) and family history of psoriasis, the most likely diagnosis is psoriatic arthritis. Psoriatic arthropathy can present before psoriatic skin changes. Therefore, option 3 is the correct answer.

Rheumatoid arthritis and seronegative rheumatoid arthritis classically affect the metacarpal phalangeal joints (MCPs) of the hands, not the DIPs. You would also expect the rheumatoid factor to be raised in rheumatoid arthritis (but not seronegative rheumatoid arthritis).

Idiopathic juvenile arthritis presents like rheumatoid arthritis in patients under 16 years old. Given the age of this patient, this is not a likely diagnosis.

Septic arthritis presents with a swollen, red and very painful joint. The patient would classically be pyrexial and unwell. Therefore, this is not the most likely diagnosis.

Therefore, option 3 is the most likely diagnosis.

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A 65-year-old man presents with lower back and buttock pain which has slowly worsened over the past 6 months He reports a burning pain which is not relieved by paracetamol. He reports the pain has become so severe he often has to sit down when walking downhill to the bus stop. He has no problems walking home from the bus stop and no bladder or bowel dysfunction.

What is the most likely diagnosis?

A. Prolapsed lumbar disc

B. Osteoarthritis

C. Sciatica

D. Spinal stenosis

E. Spinal cord compression

D. Spinal stenosis

Spinal stenosis is often relieved by sitting down or leaning forward

Spinal stenosis is the correct answer here as his symptoms are relieved by sitting down and are worse on walking downhill. Spinal stenosis is most common between in the 6th and 7th decades and can present on a background of lower back pain. Leaning forward opens up the spinal canal and can relieve symptoms. Patients are often said to have a characteristic flexed or stooped position when walking. As the condition progresses patients may also report numbness or tingling in the back, buttocks and leg as well as pain.

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A 52-year-old woman attends the emergency department following a fall down the bottom 2 steps of her staircase and sustaining an injury to her left arm. She denies any pre-syncopal symptoms but describes feeling generally more lethargic over the last few weeks with generalised body aches and cramps. Her past medical history includes chronic kidney disease stage 1 and she takes no regular medications.

An X-ray of her left forearm confirms a fracture of the distal radius. Laboratory tests:

Hb	124 g/L	(115 - 160)
Platelets	233 * 10 /L 9	(150 - 400)
WBC	9.6 * 10 /L 9	(4.0 - 11.0)
Na +	132 mmol/L	(135 - 145)
K +	4.1 mmol/L	(3.5 - 5.0)
Urea	7.8 mmol/L	(2.0 - 7.0)
Creatinine	144 µmol/L	(55 - 120)
Calcium	1.8 mmol/L	(2.1-2.6)
Phosphate	0.6 mmol/L	(0.8-1.4)
ALP	126 u/L	(30 - 100)
Parathyroid hormone	14 ng/L	(12-72)
ESR	22 mm/hr	< ((age + 10) / 2)

What is the most likely underlying diagnosis?

A. Osteomalacia

B. Osteopetrosis

C. Osteoporosis

D. Paget's disease

E. Tertiary hyperparathyroidismbmit answer

A. Osteomalacia

Osteomalacia

- low: calcium, phosphate
- raised: alkaline phosphatase

This patient has a diagnosis of **osteomalacia**. Osteomalacia is the demineralisation of bone, most commonly secondary to vitamin D deficiency. Patients with osteomalacia typically present with bony pain, muscle tenderness, proximal myopathy, and an increased tendency for fractures. Blood tests reveal low calcium and phosphate levels and raised alkaline phosphatase levels (ALP), as seen here. This patient describes a history of lethargy and body aches and cramps that is likely a result of her hypocalcaemia.

Osteopetrosis, also known as marble bone disease, is a disorder of osteoclast function that impairs bone resorption. This results in dense bones that are likely to fracture. Bone pains are common, like those described by this patient. However, on laboratory testing, calcium, phosphate and ALP levels are normal.

Osteoporosis is a reduced bone mineral density associated with multiple risk factors including menopause, drugs (e.g. glucocorticoids), chronic kidney disease and endocrine disorders. Similar to this patient's presentation, osteoporosis also increases the likelihood of fractures. However, unlike this patient, patients with osteoporosis have normal calcium, phosphate and ALP levels.

Paget's disease is most commonly seen in men of increasing age. It is usually asymptomatic although can present with bone pain, particularly in the pelvis and lumbar spine. It is uncommon in a young woman such as this patient. Furthermore, laboratory tests typically reveal an isolated raised ALP but with normal calcium and phosphate levels.

Tertiary hyperparathyroidism is commonly seen in patients with chronic kidney disease. Initially, there is compensatory hypertrophy of the parathyroid glands secondary to hypocalcaemia. As the disease progresses, persistent secondary hyperparathyroidism causes parathyroid hyperplasia (tertiary hyperparathyroidism). In tertiary hyperparathyroidism, calcium and phosphate are both raised with a raised parathyroid hormone.

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A 48-year-old woman attends with progressive shortness of breath on exertion. On examination, you note coarse bibasal inspiratory crepitations and thickening of the skin of her proximal limbs. Observations are as follows: heart rate 85 beats per minute, respiratory rate 22 breaths per minute, blood pressure 210/95 mmHg, temperature 37.5ºC, and oxygen saturation 95% on air. Her urinary output is 0.4 ml/kg/hour.

Blood results are as follows:

Hb	105 g/L	Male: (135-180)Female: (115 - 160)
Platelets	154 * 10 /L ^{9}	(150 - 400)
WBC	15.8 * 10 /L ^{9}	(4.0 - 11.0)
Na $^+$	132 mmol/L	(135 - 145)
K $^+$	4.9 mmol/L	(3.5 - 5.0)
Urea	18.2 mmol/L	(2.0 - 7.0)
Creatinine	426 µmol/L	(55 - 120)
CRP	3 mg/L	(< 5)
ANA	1:128	(<1:40)
Anti dsDNA	68 IU/mL	(<10)

A chest x-ray is performed:

Chest x-ray	Bilateral air space opacification with diversion of blood to the upper lobes

What treatment is indicated?

A. Dialysis
B. Losartan
C. Prostacyclin
D. Ramipril
E. Steroids and mycophenolate

D. Ramipril

Renal complications of systemic sclerosis - ACE-inhibitors

The clinical presentation is of diffuse cutaneous systemic sclerosis with a scleroderma renal crisis (SRC). SRC is characterised by malignant hypertension and oligo-anuric acute renal failure. A renal biopsy is not necessary to confirm the diagnosis of classical SRC.

Ramipril is correct. ACE inhibitors (ACEi) are the mainstay of therapy in SRC. Initiation of their use allowed for major increased survival rates of SRC over the last decades. The treatment goal is to achieve blood pressure control as soon as possible. Importantly, ACE inhibitors must be continued even if renal function is deteriorating.

Dialysis is incorrect. Indications for dialysis include uraemic encephalopathy, uraemic pericarditis, uraemic haemorrhage, oliguria resulting in clinically significant volume overload and respiratory distress (that is refractory to medical management), metabolic acidosis due to renal failure, and hyperkalaemia (>6.0) refractory to medical management. The patient does not fulfil any of these criteria at the current time.

Losartan is incorrect. Angiotensin II receptor blockade (ATII) may be useful but is anecdotally less effective than ACE inhibitors, although clinical experience with these agents has been limited.

Prostacyclin is incorrect. Continuous low doses of prostacyclin may be added to standard therapy although there is no evidence that it improves short-term and long-term prognosis.

Steroids and mycophenolate is incorrect. Steroids have been shown to precipitate and worsen SRC. Clinical trials have yet to demonstrate the efficacy of immunosuppressive agents for the treatment of SRC.

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A 74-year-old woman attends rheumatology clinic for a review of her osteoporosis treatment. The patient had been diagnosed with osteoporosis on a DEXA scan five years previously after she fell and sustained a Colles fracture on the left side. Following this diagnosis, the patient had been initiated on treatment with alendronic acid.

In clinic, the patient reported that she had recently been suffering from nagging back pain over the past few weeks. She denied any history of recent falls or other trauma.

Past medical history was significant for rheumatoid arthritis, diagnosed when the patient was 28 years old. Following this diagnosis, she had received prolonged treatment with corticosteroids in association with a variety of disease modifying drugs. Ultimately, good control of her arthritis had been achieved using methotrexate (10 mg weekly) and the patient had not required corticosteroid treatment for many years. The patient reported no family history of osteoporosis or fragility fractures. She did not smoke or drink any alcohol.

The patient reported no concerns or side effects associated with taking her weekly alendronic acid (70 mg weekly).

Examination of the patients spine demonstrated mid-line point tenderness around the T12 - L1 level. Neurological examination of the lower limbs was unremarkable.

Thoracolumbar spine x-ray: anterior height loss of T12 vertebrae, otherwise unremarkable

Height	150 cm
Weight	55 kg
Femoral neck BMD (5 years previously)	T - 3.2
Femoral neck BMD (present day)	T - 2.4
FRAX 10-year probability of major osteoporotic fracture	27 %
FRAX 10-year probability of hip fracture	8.7 %

What is the most appropriate management of the patient's osteoporosis?

A. Hold further osteoporosis treatment with repeat DEXA scan in two years
B. Hold further osteoporosis treatment with repeat DEXA scan in five years
C. Discontinue alendronic acid and initiate treatment with denosumab
D. Discontinue alendronic acid and initiate treatment with zoledronic acid
E. Continue treatment with alendronic acid with repeat DEXA scan in five years

E. Continue treatment with alendronic acid with repeat DEXA scan in five years

The patient's bone mineral density has improved secondary to her treatment with alendronic acid. However, her recent back pain and abnormal thoracolumbar x-ray suggest she has suffered from a osteoporotic vertebral fracture. In one study, continuing alendronic acid from five to ten years treatment reduced the incidence of clinical vertebral fractures in all patients regardless of T score. Therefore, continuing alendronic acid treatment would be recommended for this patient.

Due to increased awareness of the potential complications of long-term bisphosphonate treatment, treatment breaks (or 'drug holidays') are now employed for some patients. Specifically, a treatment break should be considered for patients less than 75 years old, with femoral neck T score > - 2.5, no history of osteoporotic vertebral fracture and deemed low risk following assessment by WHO Fracture Risk Assessment Tool (FRAX) and National Osteoporosis Guideline Group (NOGG) guidance. For such a patient, treatment would typically be suspended for two years with repeat DEXA scan at the end of that period, or sooner in the event of fragility fracture.

Parenteral osteroporosis treatments such as zoledronic acid or denosumab should be considered if patient is intolerant of oral bisphosphonates or in the event of treatment failure (defined as two or more fractures on treatment, or one fracture and a fall in bone mineral density).

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A 73-year-old woman attends rheumatology clinic for review of her osteoporosis treatment. The patient had been diagnosed with osteoporosis five years previously on a DEXA scan performed after she had sustained a fractured right neck of femur. Since the time of diagnosis, the patient had been receiving treatment with alendronic acid (70 mg weekly).

During clinic review, the patient reported taking her alendronic acid as prescribed without any associated side effects. A review of her past medical history revealed that the patient had sustained a left distal radius fracture the previous year following a trip at home. In addition, the patient had suffered from a deep vein thrombosis in her right leg three years before precipitated by a trans-continental flight and had been anti-coagulated with warfarin for six months.

The patient was a smoker (10 cigarettes per day) and also consumed around 20 units of alcohol per week. Her mother had suffered a fractured neck of femur at the age of 80. The patient had never been diagnosed with rheumatoid arthritis and had no significant exposure to corticosteroid treatment.

Clinical examination of the patient demonstrated no loss of height since previous measurement five years previously. There was no tenderness on palpation of the thoracic or lumbar spine.

Height	175 cm
Weight	95 kg
Femoral neck BMD (5 years previously)	T - 2.7
Femoral neck BMD (present day)	T - 2.9
FRAX 10-year probability of major osteoporotic fracture	60 %
FRAX 10-year probability of hip fracture	50 %

What is the appropriate management of the patient's osteoporosis?

A. Stop treatment with alendronic acid and start treatment with denosumab

B. Stop treatment with alendronic acid and start treatment with strontium ranelate

C. Continue treatment with alendronic acid with repeat DEXA scan in five years

D. Continue treatment with alendronic acid with repeat DEXA scan in two years

E. Stop treatment with alendronic acid with repeat DEXA scan in two years

A. Stop treatment with alendronic acid and start treatment with denosumab

Due to her extensive risk factors, the patient is at an extremely high risk of suffering from further fragility fractures and it is essential that she continues with osteoporosis treatment. Bisphosphonate treatment failure is defined as two or more fractures on treatment, or one fracture with a reduction in bone density (as in this patient). Therefore, continuing treatment with alendronic acid is not advisable. Of the alternative treatments listed as possible answers, strontium ranelate is contra-indicated in this patient due to her history of deep vein thrombosis, leading to treatment with denosumab as the most appropriate treatment option.

Due to increasing awareness of complications associated with long-term bisphosphonate treatment, treatment breaks (or 'drug holidays') can be considered for some patient's after five years treatment. Guidelines recommend consideration of treatment breaks for patients less than 75 years old with a femoral neck T-score greater than -2.5 and who are defined as low risk by WHO Fracture Risk Assessment Tool (FRAX) and National Osteoporosis Guideline Group (NOGG) guidelines. When a drug holiday is agreed, advice is to repeat DEXA scan after two years, or sooner in the event of a fracture. Treatment breaks are never recommended if a patient has ever suffered from a vertebral insufficiency fracture.

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A 52-year-old man presents with lethargy and reduced sensation in both feet. He reports a 2 month history of fevers and 4kg weight loss. He also reports intermittent testicular pain.

On examination there is livedo reticularis on both legs and reduced light touch and pain sensation on both feet.

Blood tests reveal:

Hb	116 g/l	Na 137	# mmol/l	Bilirubin	18 µmol/l
Platelets	487 * 10 /l 9	K +	4.8 mmol/l	ALP	92 u/l
WBC	8.3 * 10 /l 9	Urea	12.8 mmol/l	ALT	102 u/l
Neuts	6.3 *	Creatinine	182	γGT	16 u/l

	10 /l 9		µmol/l		
MCV	89 fL	ESR	78mm/hr	Albumin	34 g/l

Which investigation is most likely to reveal the diagnosis?

A. Hepatitis C serology

B. Renal angiogram

C. cANCA

D. pANCA

E. Hepatitis B serology

B. Renal angiogram

Fever, lethargy, neuropathy, testicular pain and renal dysfunction are consistent with polyarteritis nodosa (PAN).

Whilst the other investigations listed would be appropriate, angiography would be most likely to confirm the diagnosis (sensitivity 89%, specificity 90%).

ANCA is classically negative in PAN. Hepatitis serology would be appropriate as this can be associated with vasculitis however will not reveal the diagnosis in this case.

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A 40-year-old woman is seen in an outpatient rheumatology clinic. She was diagnosed with Systemic lupus erythematosus 5 years previously when she has presented with fatigue, anaemia and a rash. She also has a past medical history of hypertension, gout and psoriasis.

She had noticed that the joints in her hands were becoming deformed, however, she was able to complete day to day task without any functional impairment and denied pain in the affected joints.

On examination, she had symmetrical marked reducible ulnar subluxation and deviation at the MCP joints. X-rays of her hands showed no erosions.

What is the most likely diagnosis?

A. Jaccoud's arthropathy

B. Rheumatoid arthritis

C. Gout

D. Psoriatic arthritis

E. Sarcoid arthropathy

A. Jaccoud's arthropathy

The key here is the absence of pain which is more than likely to be present in options B to E hence the presence of psoriasis is a distracting factor. Additionally, gout usually affects the first metatarsal head and sarcoid arthropathy is very rare hence Jaccoud's arthropathy which is none erosive and associated with systemic lupus erythematosus is the correct answer.

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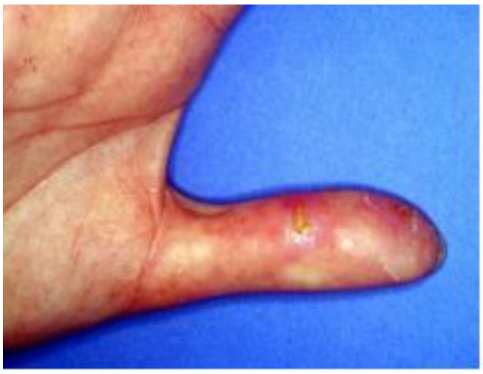
This patient is known to suffer from Raynaud's phenomenon:



What does the lesion on her thumb most likely represent?

A. Arterial ulcer

B. Gouty tophus

C. Calcium deposit

D. Orf

E. Xanthomata

C. Calcium deposit

This lesion represents calcinosis.

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A 75-year-old female was recently started on alendronate for treatment of osteoporosis following a fragility fracture. She returns to your clinic as she has suffered troubling upper gastrointestinal side effects.

What is the most appropriate next step in her management?

A. Continue alendronate

B. Change alendronate to strontium ranelate

C. Change alendronate to risedronate

D. Change alendronate to raloxifene

E. Change alendronate to denosumab

C. Change alendronate to risedronate

NICE guidance recommends that if patients suffer significant upper gastrointestinal side effects from the use of alendronate, then this should first be changed to risedronate or etidronate

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A 77-year-old lady is reviewed in the Rheumatology Clinic with a 4-week history of malaise and bilateral hip pain.

The pain is poorly localised and affects the anterior and posterior aspects of the pelvis as well as the upper thighs. It is typically worse in the mornings and associated with feelings of stiffness that take several hours to improve.

She also reports the recent onset of a right-sided headache, which is constant and has been present for the past 2 weeks. 24 hours ago, she developed an episode of transient visual darkening although she is unable to recall which eye was affected.

Her past medical history is remarkable for hypertension and hypothyroidism. Her regular medications include amlodipine 5mg once daily and levothyroxine 75 micrograms once daily.

On examination, her visual acuity is 6/9 in both eyes. Her temperature is 37.3ºC, her pulse is 73bpm and her blood pressure is 143/81mmHg. Neurological examination reveals no focal abnormality although the pulsation of her right temporal artery is difficult to feel.

Her blood results are as follows:

Hb	124 g/l	Na +	141 mmol/l
Platelets	444 * 10 /l 9	K +	3.9 mmol/l
WBC	11.2 * 10 /l 9	Urea	4.3 mmol/l
Neuts	8.1 * 10 /l 9	Creatinine	78 µmol/l
Lymphs	2.3 * 10 /l 9	CRP	102 mg/l
Eosin	0.02 * 10 /l9		

A temporal artery biopsy is performed and a 0.8cm sample is obtained. It is reported as being 'negative for giant cell arteritis (GCA)'.

What is the most appropriate treatment strategy?

A. IV methylprednisolone 1 gram once daily
B. Prednisolone 15mg once daily
C. Amitriptyline 10mg once daily
D. Explanation, reassurance and referral to physiotherapy for graded aerobic exercise
E. Prednisolone 60mg once daily

A. IV methylprednisolone 1 gram once daily

This lady's presentation is consistent with GCA. The picture is complicated by additional features of polymyalgia rhuematica (PMR) although it is important to bear in mind that the two often occur together in clinical practice.

A normal temporal artery biopsy does not rule out GCA due to the possibility of skip lesions giving rise to a false negative result. 7-44% of patients with GCA will have a negative temporal artery biopsy and such falsely reassuring results are more likely to occur when shorter arterial specimens are obtained. For this reason, the British Society for Rheumatology (BSR) recommends that biopsy specimens should no less than 1cm in length.

Transient visual loss can herald the onset of permanent blindness and the BSR recommend that these patients receive IV methylprednisolone 500-1000mg daily for 3 days.

Those with established visual loss or uncomplicated GCA should receive 60mg prednisolone daily.

Prednisolone 15mg daily is the treatment for isolated PMR and would not be appropriate in this case. The remaining options are treatments for fibromyalgia and are therefore incorrect.

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A 28-year-old woman presents to the gastroenterology clinic for review. She has been diagnosed with coeliac disease some 2 years earlier, and has been suffering from severe tiredness, muscle aches and proximal weakness for the past few months. On examination her blood pressure is 112/70 mmHg, pulse is 75 beats per minute and regular. You confirm proximal muscle weakness.

Investigations

Ca ++	2.0 mmol/l
Alkaline phosphatase	275 IU/l

Which of the following is the most useful next investigation?

A. CK
B. Parathyroid hormone
C. Vitamin D
D. Muscle biopsy
E. Electromyography

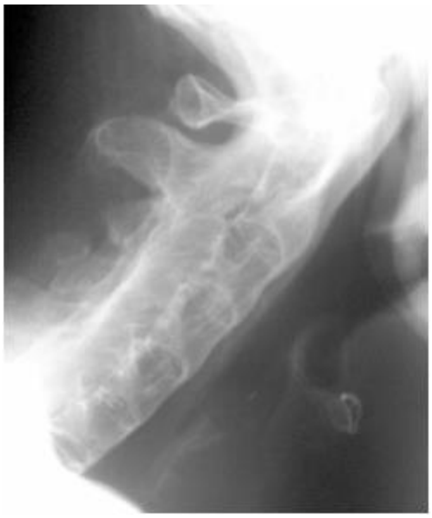
C. Vitamin D

Coeliac disease is known to interfere with absorption of fat soluble vitamins, including vitamin D, and the low calcium and elevated alkaline phosphatase, coupled with symptoms of proximal myopathy fits with a diagnosis of osteomalacia. Vitamin D levels are therefore the next investigation of choice.

CK and muscle biopsy are indicated for possible inflammatory myositis, and the limited proximal weakness seen here, coupled with low calcium is much more consistent with osteomalacia. Parathyroid hormone may be elevated, but this is secondary to low vitamin D and calcium. Electromyography is most useful for assessment of motor neuropathy, which doesn't fit with the painful proximal muscle weakness seen here.

Question 70 of 178

A 30-year-old man presents to the emergency department with worsening neck pain, which limits his participation in contact sports. He is generally well otherwise and does not take any regular medications. He smokes ten cigarettes daily and drinks alcohol occasionally. The plain film radiograph of his C-spine showed:



What is the next best step in his management?

A. Commence etanercept

B. Commence regular naproxen

C. Commence sulfasalazine

D. Limit physical exercise

E. Urgent referral to spine surgery

B. Commence regular naproxen

The neck radiograph shows syndesmophytes, squaring of the vertebrae and bamboo spine, characteristic of ankylosing spondylitis.

Commence regular naproxen is the correct answer. NSAIDs are the first-line treatment for ankylosing spondylitis. Therefore the patient should be started on oral NSAIDs first.

Commence etanercept is incorrect. The 2010 European Alliance of Associations for Rheumatology (EULAR) guidelines suggest: 'anti-TNF therapy should be given to patients with persistently high disease activity despite conventional treatments'.

Commence sulfasalazine is incorrect. Disease-modifying drugs used to treat rheumatoid arthritis (such as sulphasalazine) are only useful in ankylosing spondylitis if peripheral joint involvement exists.

Limit physical exercise is incorrect. Physiotherapy and regular exercise are recommended treatments for ankylosing spondylitis as per the EULAR guidelines.

Urgent referral to spine surgery is incorrect. The most common surgical procedure for ankylosing spondylitis is a laminectomy to relieve pressure on the nerve roots. However, as this patient has no features of nerve root compression, medical therapy is the next best step in his management.

Question 71 of 178

A 53-year-old has just been diagnosed with rheumatoid arthritis whilst having a severe flare. She is started on methotrexate 15mg once weekly, folic acid 5mg once weekly, hydroxychloroquine 200mg BD, naproxen 250mg TDS and prednisolone 15mg OD. She returns one month later complaining of mouth ulcers. Bloods show the following:

Hb	142 g/l
Platelets	225 * 10 /l9
WBC	6 * 10 /l9
Na +	136 mmol/l
K +	4.2 mmol/l
Urea	4 mmol/l
Creatinine	95 µmol/l
Bilirubin	6 µmol/l
ALP	105 u/l
ALT	92 u/l

What is the most appropriate course of action?

A. Admit for IV methylprednisolone

B. Stop methotrexate, hydroxychloroquine and naproxen

C. Increase folic acid to two days a week

D. Stop hydroxychloroquine and discuss with rheumatology

E. Stop methotrexate and discuss with rheumatology

E.Stop methotrexate and discuss with rheumatology

According to BSR guidelines, if new oral ulceration starts whilst a patient is on methotrexate then it should be withheld initially and discussed with the specialist team.

In this patient, the alanine transaminase (ALT) is not two times the upper range of normal so that does not affect your decision. Often the folic acid is increased to six days a week (apart from the day of methotrexate) to mitigate side effects.

Hydroxychloroquine and naproxen are not associated with oral ulceration.

Question 72 of 178

A 60 year old woman attended her General Practitioner and reported a three month history of bilateral shoulder muscle and bilateral hip girdle aches and pain. She also experienced stiffness affecting these areas that lasted for up to two hours each morning. These symptoms were limiting her day to day activities and were unresponsive to simple analgesics.

The patient denied symptoms of headache, visual disturbance or jaw claudication. Intermittent episodes of dry mouth and dry eyes had been present for several years. There was no history of unexplained skin rashes. Past medical history included coeliac disease diagnosed twenty years previously that was well controlled on a gluten-free diet. The patient was a non-smoker and drank alcohol only occasionally.

Examination revealed mild muscular tenderness across the shoulder and hip girdles although with no other inflamed or tender joints. Cardiovascular and respiratory examination was unremarkable.

Blood tests requested by her GP demonstrated an elevated ESR of 65. A diagnosis of PMR was made and a course of 20 mg prednisolone daily prescribed. However 6 weeks later the patients symptoms had not significantly improved and she was referred to rheumatology clinic. Repeat blood tests and other investigations are listed below.

Haemoglobin	110 g / dL
White cell count	8.9 * 10 /l
Neutrophils	7.8 * 10 /l
Platelets	456 * 10 /l
Urea	6.2 mmol / L
Creatinine	87 micromol / L
Sodium	138 mmol / L
Potassium	4.1 mmol / L
Ferritin	180 ng / mL
Erythrocyte sedimentation rate	75 mm / h
Rheumatoid factor	Negative
Connective tissue ANA	Negative
Anti-CCP antibodies	58 EU (reference < 20)
Creatinine kinase	89 U / L (reference 5-130)

X-ray hands: minor degenerative change in multiple inter-phalangeal joints of both hands; no evidence of erosive arthropathy

What is correct diagnosis?

A. Polymyalgia rheumatica

B. Rheumatoid arthritis

C. Polymyositis

D. Sjorgren's syndrome

E. Systemic lupus erythematous

B. Rheumatoid arthritis

Rheumatoid arthritis can present with a polymyalgic syndrome prior to clinically detectable sinovitis. In this case this is suggested by the lack of response to trial of prednisolone and the positive anti-CCP antibody. Observational studies have shown a greater clinical and laboratory response to steroids in polymyalgia rheumatica than polymyalgic onset rheumatoid arthritis. AntiCCP antibodies are rarely present in polymyalgia rheumatica but are strongly associated with rheumatoid arthritis.

Sjorgren's syndrome and SLE are unlikely given the lack of anti-nuclear antibodies. Polymyositis is excluded by the normal CK.

Question 73 of 178

A 65-year-old man presented to his General Practitioner with a 3-month history of bilateral shoulder aches and pains. The symptoms were associated with stiffness in the mornings taking up to two hours to resolve after waking. The patient denied any symptoms of headache, jaw claudication or visual disturbance. The patient had no symptoms of dry eyes or mouth, no skin or hair changes, no weight loss and no fevers.

Past medical history included hypertension and chronic obstructive pulmonary disease. Regular medications included ramipril, simvastatin and inhaled salbutamol as required. The patient was an ex-smoker who drank 25 units of alcohol per week. The patient had recently retired having spent his working life as a train driver.

The examination did not reveal any inflamed joints excepting slight tenderness across the shoulder girdle. There was no evidence of scalp tenderness. The cardiovascular and respiratory examination was unremarkable.

Investigations requested by the General Practitioner are listed below.

Haemoglobin	134 g / L
White cell count	7.5* 10 /l
Neutrophils	6.0 * 10 /l
Platelets	356 * 10 /l
Urea	8.9 mmol / L
Creatinine	110 micromol / L
Sodium	132 mmol / L
Potassium	4.9 mmol / L
Erythrocyte sedimentation rate	85 mm / h
Rheumatoid factor	Negative
Creatinine kinase	121 U / L (reference 5-130)
Calcium (adjusted)	2.25 mmol / L (reference 2.18-2.58)
Alkaline phosphatase	67 U / L (reference 35-100)
Thyroid stimulating hormone	2.5 microU / L
Protein electrophoresis	Normal

What is the appropriate next management step for this patient?

A. Stop statin therapy and review in 6 weeks

B. Ultrasound study of shoulders and hips

C. Referral for specialist rheumatology opinion

D. Prednisolone 15 mg daily with dose tapering over 2 years

E. Prednisolone 40 mg daily with dose tapering over 1 year

D. Prednisolone 15 mg daily with dose tapering over 2 years

This patient presents with a classical history for polymyalgia rheumatica and a raised ESR. There are no factors in the history or investigations that suggest an alternative diagnosis (for example, giant cell arteritis, other connective tissue disease, myeloma, malignancy or occult infection). The normal CK would make statin-induced myopathy unlikely.

In such cases, a trial of steroid therapy for likely polymyalgia rheumatica is appropriate. The diagnosis will be confirmed by rapid resolution of symptoms following initiation of treatment. Observational studies suggest a typical starting dose of prednisolone around 15 mg with a median time to stopping therapy of two years.

Musculoskeletal ultrasound often identifies inflammation around the shoulders and hips although these findings are not unique to polymyalgia rheumatica. The usefulness of this technique is yet to be determined outside of specialist settings.

Question 74 of 178

A 40-year-old woman was diagnosed with fibromyalgia 6 months previously following review in the Rheumatology outpatient clinic. She was subsequently discharged from the clinic with a recommendation for a trial of pregabalin therapy. The patient has now returned to her General Practitioner to report on-going symptoms of severe widespread body pain, severe fatigue and difficulty in concentrating on daily activities. The patient did not feel that starting pregabalin 6 months previously had offered any improvement in her symptoms. In fact, her symptoms had been causing more problems and she had recently been unable to attend her work as a teaching assistant.

The patients past medical history included a duodenal ulcer five years previously, induced by the combination of non-steroidal anti-inflammatory drug use and alcohol consumption. She also had a previous diagnosis of irritable bowel disease and had a tendency to become severely constipated. There were no known allergies to medications. The patient lived alone and in addition to her teaching assistant job was her elderly mother's primary carer.

On assessment by her General Practitioner, there was no evidence of inflammatory arthritis but multiple tender spots were demonstrated across the patient's body. The patient was clearly distressed and frustrated with her on-going symptoms and had concerns about her ability to continue in paid employment. The patients affect was otherwise unremarkable with a good rapport maintained throughout. She denied any symptoms of low mood or thoughts of self-harm.

Following further discussion, the patient was keen to try a further pharmacological therapy as treatment for her fibromyalgia symptoms. She was reluctant to engage with suggested psychological therapies.

What is appropriate next line pharmacological treatment for the patient's fibromyalgia?

A. Continue pregabalin, start duloxetine
B. Continue pregabalin, start ibuprofen as required
C. Stop pregabalin, start duloxetine
D. Stop pregabalin, start amitriptyline
E. Continue pregabalin, start fluoxetine

C. Stop pregabalin, start duloxetine

The evidence base for pharmacological treatment of fibromyalgia includes many trials with small participant numbers and short follow-up periods. In addition, many trials have exclusion criteria (such as co-morbid psychiatric illness or chronic physical illness) that make generalisation to patients seen in clinical practice difficult.

Recent network meta-analysis has demonstrated that once trials with fewer than 50 participants are excluded then there is only evidence of effectiveness of duloxetine and pregabalin to improve pain and quality of life. Current recommendation is to use an agent at an effective dose for at least four weeks then assess response. If no response obtained then initial treatment should be held before a new agent is started, for example stopping pregabalin before a trial of duloxetine as in this case.

Using ibuprofen or amitriptyline would likely be inappropriate for this patient given the previous history of peptic ulceration and constipation.

Question 75 of 178

A 62-year-old man presents with a week of right-sided shoulder pain. He denies any trauma and denies radiation of the pain. There is no associated weakness or numbness. The pain is more noticeable at the extremities of movement and is affecting activities of daily living. His past medical history includes type 2 diabetes mellitus and asthma.

On examination, the affected shoulder is painful and restricted, with both active and passive movement. External rotation is most severely limited. Pain is elicited when applying direct pressure to the coracoid process.

Given the most likely diagnosis, what is the most appropriate next step?

A. Sub Arthroscopy
B. MRI scan
C. Physiotherapy
D. Ultrasound
E. X-ray

C. Physiotherapy

Adhesive capsulitis is a clinical diagnosis and does not need imaging or arthroscopy to confirm the diagnosis

Important for me Less im

The diagnosis here is most likely to be adhesive capsulitis (frozen shoulder). This is evidenced by the reduction in movement, associated pain, and external rotation being more affected. The background of diabetes mellitus is also a risk factor. Adhesive capsulitis is generally a clinical diagnosis and so imaging/investigation is not required. **Physiotherapy** is therefore the correct answer and is the mainstay of treatment. It should be initiated as early on as possible.

Arthroscopy is inappropriate. This may be considered much further down the line as a therapeutic option if previous measures have failed to control the symptoms.

MRI scanning is unwarranted. This may be useful if the diagnosis were not as clear-cut and other pathologies needed to be ruled out.

Ultrasound may be considered before MRI if the diagnosis is uncertain and differentials need to be ruled out. However, in a case such as this, the clinical picture is sufficient to make the diagnosis.

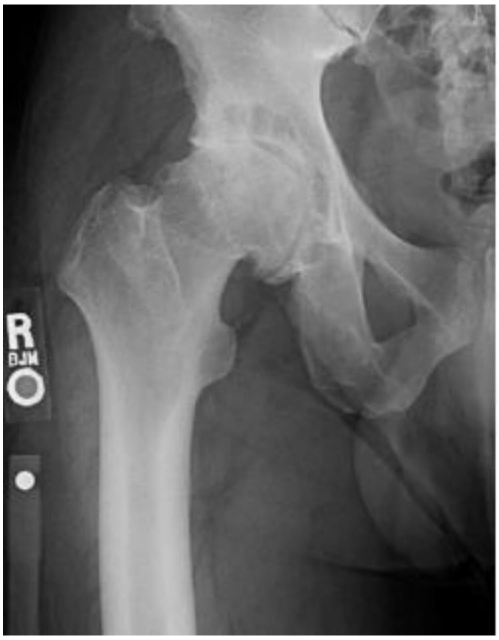
An **X-ray** may be considered to exclude dislocations/fractures. However, in the history above, the picture is very strongly suggestive of adhesive capsulitis and so imaging is not needed. Physiotherapy should be initiated.

Question 76 of 178

A 55-year-old female presents with increasing right-sided groin pain on activity. She recently joined a gym in an attempt to lose weight after being diagnosed with obesity. She has been started a number of activities including cycling, running and aerobic classes. The patient first noted the pain 3 months ago following a running session. She was able to continue with her exercise but the pain has become progressively worse and she now finds her gym sessions very difficult to complete.

Other than being overweight the patient has known hypothyroidism and asthma which both are well controlled on treatment.

An X-ray of the patient's hip is performed as below.



What is the patient's most likely diagnosis?

A. Avascular necrosis

B. Greater trochanteric pain syndrome (GTPS)

C. Inflammatory arthritis

D. Osteoarthritis

E. Hip dysplasia

D. Osteoarthritis

This patient has presented with the typical history and x-ray findings of osteoarthritis (OA). Risk factors for OA include obesity, increasing age and female gender. OA normally affects the knees and hip although most joints can be involved. When presenting with hip OA patients commonly report chronic, progressive groin pain on or post-exercise. Diagnosis is normal clinical and based on x-ray findings, as seen on this patient's imaging, which includes joint space loss, osteophyte formation, cyst formation and subchondral sclerosis.

Avascular necrosis (AVN) is bone breakdown due to a disruption of the blood supply. It is very rare in the absence of risk factors such as fracture, joint dislocations, alcoholism or high-dose steroids use. This patient does not have any clear risk factors of AVN, nor does she have any of the typical x-ray findings, which are normally only apparent in advanced disease, including bone flatting, subchondral radiolucent lines and collapsed femoral head.

Greater trochanteric pain syndrome (GTPS) is inflammation of the trochanteric bursa and is often a manifestation of an acute hip injury. Although this patient has the typical signs of pain on movement and activity, x-ray changes are very rare in GTPS.

Inflammatory arthritis includes conditions such as rheumatoid arthritis which are due to systemic disease. As such isolated joint involvement is rare especially of the hip with the condition commonly affecting the hands, feet and knees. X-ray findings include subchondral cysts and evidence of soft tissue swelling.

Hip dysplasia is an abnormality of the hip joint that normally develops at birth or in early life. As such hip dysplasia is screened for, via examination, in all newborns. In hip dysplasia, the acetabulum does not fully cover the femoral head resulting in an increased risk of joint dislocation. If unrecognised it can result in arthritis and movement issues, but again these normal present soon after a child starts walking

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A 64-year-old man was admitted to the hospital after falling and developing acute pain in his left hip, a fracture was confirmed on CT scan.

His past medical history includes Paget's disease and depression for which he takes venlafaxine. He was treated for acute lymphocytic leukaemia as a child.

The orthopaedic team have asked for some advice as the CT scan has been reported as showing some demineralisation in the femur and pelvis. You recommend a DEXA scan which confirms osteoporosis.

You also request some blood tests which are reported as follows:

Calcium	2.0 mmol/L	(2.1-2.6)
Phosphate	0.9 mmol/L	(0.8-1.4)
Magnesium	0.9 mmol/L	(0.7-1.0)
Thyroid stimulating hormone (TSH)	8.9 mU/L	(0.5-5.5)
Free thyroxine (T4)	3 pmol/L	(9.0 - 18)
Free testosterone	82 ng/dL	(in ages >50 years: 193 - 740)
ALP	202 U/L	(60-306)

What aspect of this man's medical history and blood tests would predispose him to developing osteoporosis?

A. Acute lymphocytic leukaemia

B. Hypothyroidism

C. Low testosterone levels

D. Paget's disease

E. Venlafaxine use

C. Low testosterone levels

Osteoporosis in a man - check testosterone

Low testosterone levels: correct, low testosterone levels are associated with reduced bone mineral density and increased risk of osteoporosis and fractures.

Acute lymphocytic leukaemia: multiple myeloma and lymphomas are two haematological malignancies associated with osteoporosis, not acute lymphocytic leukaemia.

Hypothyroidism: hyper rather than hypothyroidism is associated with osteoporosis. **Paget's disease**: this is incorrect, Paget's is not associated with osteoporosis they are entirely separate conditions. Paget's is associated with increased osteoclast activity which leads to increased bone resorption. Osteoblasts respond to this by rapidly increased but abnormal new bone formation.

Venlafaxine: SSRIs and anti-epileptics are associated with osteoporosis, but there is not a link with venlafaxine.

Question 78 of 178

A 78-year-old woman is admitted following a fall. An x-ray of her right wrist showed distal ulnar and radial fractures.

Her past medical history includes diet-controlled diabetes and hypertension. She wants to know what can be done to prevent further fractures in the future.

What would be the most appropriate investigation and treatment option for her?

A. Arrange a DEXA scan and commence alendronate depending on the results
B. Commence alendronate
C. Commence denosumab
D. Commence zolendronate
E. Start calcium and vitamin D supplementationanswer

B. Commence alendronate

Start alendronate in patients >= 75 years following a fragility fracture, without waiting for a DEXA scan

Commence alendronate is the correct answer. The patient, in this case, is over 75 years of age and has had a fragility fracture, NICE guidance advises commencing bisphosphonates in this group of patients without waiting for a DEXA scan.

Commence denosumab is incorrect. Although this may be an appropriate treatment at some point, it is not yet a first-line treatment for osteoporosis due to cost.

Arrange a DEXA scan and commence alendronate depending on the results is incorrect. NICE guidance advises starting bisphosphonates in this group of patients without waiting for a DEXA scan.

Commence zolendronate is incorrect. Intravenous bisphosphonates are not generally a first-line option for the treatment of fragility fractures. In some cases, such as following hip fractures, IV zolendronate can be considered a first-line option.

Start calcium and vitamin D supplementation is incorrect. Although this would be recommended to assist in improving bone health, it is not the most appropriate treatment option for this patient given she has had a fragility fracture already.

Question 79 of 178

An 82-year-old woman presents to the GP with a 2-week history of worsening lower back pain. A lumbar x-ray is organised which reveals a wedge fracture of the L3 vertebral body.

Her past medical history includes hypertension, for which she takes amlodipine, and dietcontrolled type 2 diabetes mellitus.

What is the correct statement regarding this case?

A. A DEXA scan should be arranged if her QFracture score confirms high risk of fragility fracture
B. A DEXA scan should be arranged if she takes regular high-dose corticosteroids
C. A DEXA scan should be arranged regardless of risk factors
D. She should be offered alendronic acid
E. She should be offered referral to a specialist in osteoporosis

E. She should be offered alendronic acid

Start alendronate in patients >= 75 years following a fragility fracture, without waiting for a DEXA scan

The correct answer is **she should be offered alendronic acid**. This patient suffered a fragility fracture over the age of 75. Such patients are generally assumed to have osteoporosis and should be offered bone-sparing treatment. Bisphosphonates such as alendronic acid are recommended first line.

A DEXA scan should be arranged if her QFracture score confirms the high risk of fragility fracture is incorrect. Risk scores such as QFracture and FRAX can be used to determine risk in younger patients before organising a DEXA scan. However, over 75, a patient with a fragility fracture should be offered treatment without needing a DEXA.

A DEXA scan should be arranged if she takes regular high-dose corticosteroids is incorrect. Corticosteroids are a risk factor for osteoporosis and this is factored into the risk assessment. However, this patient's risk is sufficiently high by the nature of her age and the presence of a fragility fracture that she should be offered treatment regardless of other risk factors.

A DEXA scan should be arranged regardless of risk factors is incorrect. As the patient is over 75 with a fragility fracture, the risk is sufficiently high to commence treatment here. Delaying treatment for a DEXA scan would not be appropriate.

She should be offered referral to a specialist in osteoporosis is incorrect. This option can be considered in more complex cases, such as young patients at high risk. However, a patient over 75 with a fragility fracture should be offered treatment immediately in primary care - a referral is not necessary.

Question 80 of 178

A 73-year-old woman attends your clinic with the results of her DEXA scan:

Left neck of femur	-2.8
Right neck of femur	-3.0

Her medical history is notable for previous pulmonary embolism and hyperparathyroidism.

You discuss bone protection, but she developed a severe rash to her bisphosphonates and is not willing to restart any drug of that class.

Which of these describes the mechanism of action of the next most appropriate therapy?

A. Selective oestrogen receptor modulator (SERM)
B. Recombinant parathyroid hormone
C. Incorporation into bone in place of calcium
D. RANKL inhibitor
E. Farnesyl diphosphate synthase (FPPS) inhibitor

D. RANKL inhibitor

Denosumab - RANKL inhibitor

This woman has critical osteoporosis and is susceptible to a fragility fracture. Many patients have reactions to bisphosphonates and being aware of the contraindications for the numerous other medications is important.

Raloxifene is a SERM and is contraindicated in previous thromboembolic disease. Strontium is also contraindicated in previous venous thromboembolism and works by being incorporated into bone in place of calcium (due to it's atomic similarities).

Teriparatide is a recombinant form of parathyroid hormone and is contraindicated in previous hyperparathyroidism.

FPPS inhibition is the mechanism by which bisphosphonates have their effect.

As such, denosumab is the most appropriate next step. It is a RANKL inhibitor and requires 6 monthly subcutaneous injections. It is recommended by NICE for patients who are unable to tolerate or are precluded from the other options.

Question 81 of 178

A 72-year-old woman presents to the emergency department because she is struggling to cope at home. She has noticed that she has been struggling to get out of bed over the last month and not been feeling herself. She denies any fevers or specific systemic symptoms other than feeling weak and tired and her arms have been feeling heavy in the morning. Both her shoulders have been aching, and her appetite has been reduced. She has a past medical history of stroke, from which she made a complete recovery, atrial fibrillations, heart attack with coronary stenting and hypertension. Her observations are normal apart from a temperature of 37.7 degrees. Her joints are not tender and not swollen. She has normal power in her shoulders. Her visual acuity is normal.

Blood tests:

Hb	122 g/l
Platelets	366 * 10 /l9
WBC	8.9 * 10 /l9
Na +	137 mmol/l
K +	4.1 mmol/l
Urea	6.2 mmol/l
Creatinine	122 µmol/l

What is the most likely diagnosis?

A. Polymyalgia rheumatica
B. Rheumatoid arthritis
C. Dermatomyositis
D. Osteoarthritis
E. Adhesive capsulitis

Polymyalgia rheumatic is characterised by abrupt onset of bilateral early morning stiffness in the over 60s

The most likely diagnosis is polymyalgia rheumatica due to the sudden onset of bilateral proximal difficulty in moving arms in a patient over 50 years old without weakness. This is commonly, but not always, described as a stiffness. These are some of the key features of polymyalgia rheumatic which help diagnosis, and the presence of loss of appetite, low-grade fever and morning symptoms are supportive. Dermatomyositis is a differential diagnosis that can be hard to exclude but the absence of rash, detectable weakness and muscle tenderness makes this unlikely. Rheumatoid arthritis would be unusual at this age without symmetrical polyarthritis but the acute onset makes it even less likely. Osteoarthritis normally affects hips and knees to a greater extent and would have a gradual onset without fever. Adhesive capsulitis is possible as a bilateral diagnosis but would need a reduced range of motion in both passive and active ranges and would not be as systemic as in this patient.

Question 82 of 178

A 68-year-old female diagnosed with rheumatoid arthritis four years ago presents gradually increasing tenderness in the small joints of both hands over the past 5 months. She continues to work as a legal secretary, involving significant amounts of time at a computer. She is currently on maximum doses of methotrexate and sulphasalazine on diagnosis and maintained on the same doses since. Her DAS score today is 5.8, it was 4.7 when you saw her in clinic last 1 month ago.

What is the next management step?

A. Continue methotrexate and sulphasalazine. Short-course oral prednisolone

B. Stop current DMARDs. Start etanercept
C. Stop current DMARDs. Start infliximab
D. Admit for pulsed intravenous methylprednisolone
E. Prescribe regular long-term celecoxib in addition to methotrexate and sulphasalazine

A. Continue methotrexate and sulphasalazine. Short-course oral prednisolone

Current NICE guidelines recommend the starting of biologic therapy when the patient has been on at least two DMARDs, including methotrexate, reporting two DAS 28 scores of greater than 5.1 at least one month apart . A short course of oral prednisolone may be appropriate for flares for symptomatic control. However, this is not an option if the patient does not wish to take any steroids. Intravenous pulsed steroids or long-term treatments are not appropriate. Regular COX-2 inhibitors are not recommended by NICE guidelines. NSAIDs are appropriate for short-term symptomatic control but only at lowest doses for as short a period as possible.

Question 83 of 178

A 54-year-old woman presents to her general practitioner with three days of right shoulder pain and stiffness. She has a past medical history of type 2 diabetes and takes metformin and gliclazide. She does not smoke or drink alcohol. She works as a screenwriter.

On examination, she has globally restricted active and passive movements of the right shoulder. External rotation is most affected. There is no crepitus.

Given the likely diagnosis, what is the best method of confirming the diagnosis?

A. Arthroscopy
B. Clinical examination
C. MRI
D. Ultrasound
E. X-ray

A. Clinical examination

Adhesive capsulitis is a clinical diagnosis and does not need imaging or arthroscopy to confirm the diagnosis

Clinical examination is the correct answer. The patient presents with three days of shoulder stiffness and pain. The clue to the diagnosis is that shoulder movement is limited both on active and passive movement, suggesting adhesive capsulitis. The typical differential diagnosis for shoulder pain and stiffness includes rotator cuff disorders, osteoarthritis and adhesive capsulitis. Rotator cuff disorders principally limit active movement. While osteoarthritis may cause pain on active and passive movement, typically the joint can still be moved through its range of motion on passive movement, unlike in adhesive capsulitis where the stiffness cannot be overcome. Adhesive capsulitis is more common in females and those with type 2 diabetes. It is a clinical diagnosis and does not require imaging or arthroscopy.

Arthroscopy is the gold standard investigation to diagnose many shoulder disorders, allowing the surgeon direct visualization of anatomic structures. However, it is an invasive procedure and therefore is only indicated if conventional imaging is insufficient to make a diagnosis or where intervention is expected.

MRI can be used to diagnose a wide variety of shoulder disorders, including adhesive capsulitis.

However, its use is limited by cost. Given that adhesive capsulitis can be diagnosed clinically, it is superfluous in this case.

Ultrasound is typically used to diagnose pathology associated with rotator cuff disorders e.g. supraspinatus tendinopathy or tear. Examination findings in rotator cuff disorders typically affect active movements rather than passive movements and so a cuff disorder is unlikely in this case.

X-ray can be used to diagnose osteoarthritis and fractures. The examination findings, in this case, suggest adhesive capsulitis rather than osteoarthritis. Osteoarthritis would be a more indolent history than a three-day onset of pain and stiffness. Additionally, the degree of restriction, in this case, favours adhesive capsulitis.

Question 84 of 178

A 56-year-old man presents with a scaly rash affecting the back of his hands. The erythematous rash is present on the extensor aspects of his fingers particularly located over the MCP and PIP joints. In addition, he has noticed a violaceous swelling of his left upper eyelid. These have troubled him for the last two weeks and he has trialled an emollient cream without any benefit. His two brothers both have psoriasis and apart from childhood eczema he has been fit and well since.

In addition to these rashes, he also has a palpable mass in the left iliac fossa that is non tender and a nodular liver edge can be felt in the right upper quadrant. His conjunctiva are pale. He reports that his bowels can vary between diarrhoea and occasional constipation. He had started taking mebeverine but did not find any benefit. There are no other rashes present.

What is the likely nature of this rash?

Hb	94 g/l	Na 138 mmol/l+
Platelets	400 * 10 /l 9	K 5.0 mmol/l+
WBC	3.9 * 10 /l 9	Urea 6.0 mmol/l
Neuts	3.1* 10 /l 9	Creatinine 80 µmol/l
MCV	69 fL (range 80-100)	
Ferritin	2 ng/ml (range 20-230)	
Creatine kinase	340 U/L (range 40-320)	

A. Paraneoplastic dermatomyositis
B. Atopic eczema
C. Chronic plaque psoriasis

D. Erythema gyratum repens
E. Idiopathic dermatomyositis

A. Paraneoplastic dermatomyositis

Dermatomyositis is commonly a paraneoplastic phenomenon

The description gives Gottron's papules on the hand and a heliotrope rash on the face. This is pathognomonic for dermatomyositis which can be idiopathic but is commonly secondary to malignancy. The abdominal mass could be a rectal tumour and the nodules in the liver may be metastases. Erythema gyratum repens is another paraneoplastic rash that can occur but is very distinctive and covers large areas.

Question 85 of 178

A 24-year-old lady attends her GP with a cough for the past 4 days. She is normally well but has been suffering knee pains and has started ibuprofen and physio for her knee. During her recovery, she started exercising at the gym and noted that she felt breathless and her chest was tight. This has been followed by a cough which is worse at night and is non productive. She sleeps with two pillows and has no ankle swelling. She is normally fit and well and only has been seen as a child for breathing problems in which she took inhalers but these have long since resolved. She admits to smoking 20 cigarettes a day for the past ten years. She takes no regular medication at present and denies any foreign travel or ill contacts.

On examination, she has a dry cough and is able to speak in full sentences. She has a clear chest with mild wheeze heard in the left lower base. Her peak flow is 220 and she saturates at 98% in air with a respiratory rate of 21 breaths per minute.

Na +	139 mmol/l
K +	4.2 mmol/l
Urea	5.1 mmol/l
Creatinine	68 µmol/l

Hb	110 g/l
Platelets	390 * 10 /l9
WBC	10.0 * 10 /l9

Chest x-ray	clear lung fields with good chest expansion, no active lung lesion

What is the likely diagnosis?

A. Bronchiectasis
B. Asthma
C. COPD
D. Anxiety
E. Occult pneumothorax

A. Asthma

NSAIDs -> trigger asthma exacerbation

This lady has developed SOB and a cough which are new for her. Although she smokes, it is unlikely for her to have COPD unless she had alpha-1-antitrypsin deficiency which is rare. She could have bronchiectasis given that it is caused by viral and other respiratory illnesses in children but this presents with a productive cough and infection. An occult pneumothorax is unlikely and does not explain tightness or the nocturnal cough. Her peak flow is clearly reduced and this suggests it is not just anxiety. Given that she has previously taken inhalers as a child and now has developed a tight chest and reduced peak flow after starting and NSAID, it is likely this is asthma. Note that asthma does not always have audible wheeze and most investigations are normal.

Question 86 of 178

A 75-year-old gentleman presents to the emergency department complaining about left eye visual disturbance with peripheral vision loss noted. He tells you that over the last 2 weeks he has had constant headache, worse on the left side of his skull. It is worse when he applies pressure to it and has noted that he cannot lie on that side.

Examination is consistent with peripheral loss of vision (temporal lower quadrant) of his left eye. In addition he is quite tender over the left temporal region, with pain on his shoulders as well. He is otherwise neurologically intact but it appears that he has mild difficulty in rising from sitting.

His bloods from today include:

Hb	131 g/l	Na +	133 mmol/l
Platelets	378 * 10 /l 9	K +	4.4 mmol/l
WBC	9.2 * 10 /l 9	Urea	8.9 mmol/l
Neuts	5.6 * 10 /l 9	Creatinine	98 µmol/l
Lymphs	3.1 * 10 /l 9	CRP	133 mg/l
Eosin	0.4 * 10 /l 9	ESR	79 mm/h

He is obviously concerned regarding his symptoms and is wondering what needs to be done next:

A. Discharge home with oral steroids (prednisolone 60 mg OD) and arrange outpatient rheumatology review
B. Arrange for review by ophthalmology on next opportunity that arises
C. Organise urgent CT head
D. Admit to hospital urgently for IV methylprednisolone infusion
E. Prescribe aspirin 300 mg OD STAT

D. Admit to hospital urgently for IV methylprednisolone infusion

Urgent admission and administration of corticosteroids is important in giant cell arteritis associated with visual loss

Given the history this patient is suffering from temporal arteritis (giant cell arteritis) with visual loss. Such cases represent major emergencies that require urgent admission to hospital for IV administration of steroids. Any delay to its management can lead to permanent visual loss. It is understandable that one that one may be concerned regarding a transient ischaemic attack or indeed a cerebrovascular accident and be tempted to organise an urgent CT of the head, however the history, examination findings and blood investigations point more towards the direction of giant cell arteritis.

Question 87 of 178

A 59-year-old lady comes to the blood pressure clinic with accelerated hypertension which isn't responding to lifestyle modifications.

The patient denies any headaches or blurred vision. However, she admits to a chronic cough and frequently passes pale, loose stools. She frequently gets cold hands and tells you they go red, white and then blue in the winter.

On examination, she is a thin lady with a blood pressure of 190/100 mmHg and a heart rate of 68 beats per minute. Although her nails are a normal colour she has tight, shiny skin over her hands. An ECG shows sinus rhythm.

Hb	106 g/l
Platelets	451 * 10 /l9
WBC	8.9 * 10 /l9
Na +	136 mmol/l
K +	4.9 mmol/l
Urea	7.1 mmol/l
Creatinine	174 µmol/l

Which of the following is the most appropriate initial therapy?

A. Bisoprolol

B. Candesartan

C. Captopril

D. Indapamide

E. Amlodipine

C. Captopril

The patient describes Raynaud's phenomenon and mentions bowel and possibly lung involvement along with tight skin. This could point towards scleroderma. In this case, then, the raised creatinine and hypertension are suggestive of a scleroderma renal crisis. The first line treatment, in this case, is an ACE inhibitor.

Amlodipine would be the antihypertensive of choice if this were essential hypertension.

Question 88 of 178

A 41-year-old woman presents with tightening of fingers, mild difficulty swallowing, and mild shortness of breath on exertion. She takes pantoprazole for reflux. On examination there is tightening of skin in her fingers, however the rest of the skin is normal. Her joints are not inflamed. The rest of her examination was normal (including chest examination). Her chest X-ray is also normal. There is mild decrease in DLCO on lung function tests.

Which of the following antibodies are indicative of the underlying diagnosis?

A. Anti-Scl-70 antibody

B. Anti-dsDNA antibody

C. Rh factor antibody

D. Anti-centromere antibody

E. Anti-Jo-1 antibody

D. Anti-centromere antibody

The underlying diagnosis is limited scleroderma. Limited scleroderma has tightened hard skin below the elbows. Dysphagia and some lung fibrosis occurs in both limited and diffuse scleroderma. Limited scleroderma is associated with anti-centromere antibody with Anti-scl-70 antibody associated with diffuse scleroderma. Diffuse scleroderma is characterised by lesions proximal to the elbow, trunk and face (limited can have some facial involvement). Limited scleroderma has a 90% 5-year survival, diffuse scleroderma 70% 5-year survival. In the past, renal crises were the most common cause of death in patients with scleroderma. Aggressive treatment with blood pressure lowering drugs, particularly those known as ACE inhibitors, is proving to be successful in reducing this risk.

Anti-dsDNA and Rh factor are indicative of SLE an RA/Sjogrens/others, but there is no evidence of arthritis or cutaneous rashes. Anti-Jo-1 antibody is a marker of polymyositis.

Question 89 of 178

A 21-year-old male presents to his GP complaining of muscle cramps that prevent him from competing in his local park 5 km race. He has always had muscle pains when warming up with exercise but these gradually diminish after 20 minutes. There was no weakness and no abnormalities on neurological exam.

Creatinine kinase was elevated at 1215 IU/L and myoglobinuria was noted on urinalysis. The electromyography (EMG) demonstrated myotonic discharges and fibrillations.

What is the likely diagnosis?

A. Sub Hypokalaemic periodic paralysis

B. Von Gierke disease

C. McArdle disease

D. Pompe disease

E. Gaucher diseasemit answer

C. McArdle disease

McArdle disease (also known as myophosphorylase deficiency or Glycogen storage disease V) often presents in adolescence with exercise **intolerance**, cra` mps and weakness. Unfortunately it is often misdiagnosed as chronic fatigue syndrome. Inheritance is autosomal recessive. The diagnostic clues are:

No rise in venous blood lactate on exercise

Muscle biopsy demonstrates elevated glycose concentration and muscle phosphorylase deficiency

Investigations usually reveal an elevated CK and myoglobinuria. Diagnosis is confirmed with forearm muscle exercise testing or genetic testing. Management involves avoidance of low carbohydrate diets and low intensity aerobic exercise.

Question 90 of 178

A 26-year-old female presented with lethargy, intermittent low grade fever, bilateral wrist pain, pleuritic type chest pain and an erythematous scaly rash perinasally. She had no significant medical history and her family history was significant for an aunt with autoimmune hepatitis and a brother with type 1 diabetes and pernicious anaemia.

Examination reveals a well young lady. Blood pressure 110/78mmHg and pulse 76/min. Abdominal and cardiovascular examination were normal. There was dullness to percussion over both lung bases. She had swelling and tenderness of her wrists. A malar rash on a background of moderate facial acne with some scaring were noted. She gives a history of acne which has improved ever since she started taking minocycline prescribed by her GP 3 months ago. She is also on oral contraceptives for irregular periods.

Investigations:

Hb	10.3 g/dl
MCV	79 fl
Platelets	256 * 10 /l9
WBC	7 * 10 /l9
Creatinine	88 umol/L
Na+	140 mmol/L
K+	3.6 mmol/L
ANA	Positive
Anti dsDNA	Negative
Chest X-ray	Blunting of costophrenic angles bilaterally
Complements C3 & C4	normal

What investigation is most likely to confirm the suspected diagnosis?

A. Serum rheumatoid factor, anti Ro, anti La
B. Renal biopsy
C. Wrist X-ray
D. Anti histone antibody
E. Repeat serum anti dsDNA to rule out lab error

D. Anti histone antibody

Answer: Serum anti histone antibodies.

This is a typical case of drug induced lupus (DIL). Patients give symptoms similar to systemic lupus erythematosus (SLE) but they are unlikely to have renal and CNS involvement.

Auto antibody screening would reveal positive anti histone antibodies and negative anti-dsDNA antibodies. ANA positivity is common.

Some common causes of drug induced lupus include minocycline, isoniazid, hydralazine, procainamide. Minocycline in particular uniquely causes a repeat episode of drug induced lupus when patient is re-challenged.

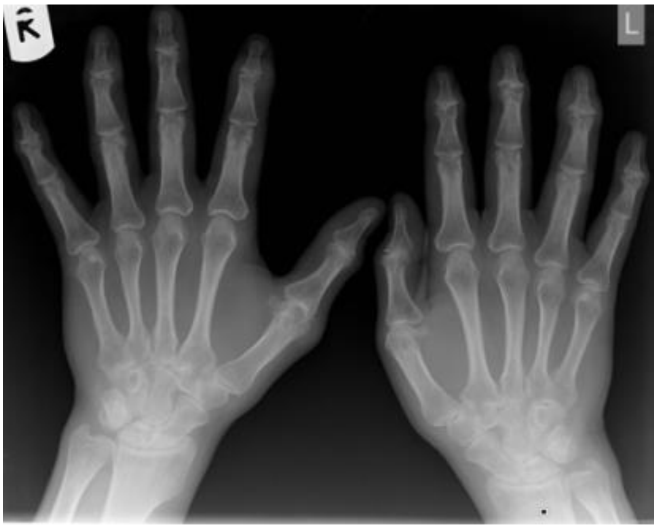
The main treatment is stopping the offending medication.

Features differentiating drug induced lupus from SLE:

- SLE is more common in patients of African descent.
- Drug induced lupus is more common in patients of European descent.
- Renal and CNS involvements are rare in drug induced lupus whereas they are features of systemic lupus erythematosus.
- Though anti histone antibodies can be found in up to 50% of patients with SLE, they are much more likely to be associated with DIL, whilst anti ds-DNA is more specific to systemic lupus erythematosus.
- It is also important to note that complement levels are normal in DIL but low in SLE.

Question 91 of 178

A 70-year-old man presents with a two-year history of stiffness and pain in his hands. The stiffness is worse in the mornings and tends to gradually ease after around an hour. On examination you note synovitis and swelling affecting the distal interphalangeal joints in both hands. An x-ray is requested and shown below:



What is the most likely diagnosis?

A. Osteoarthritis
B. Myeloma
C. Gout
D. Rheumatoid arthritis
E. Psoriatic arthritis

E. Psoriatic arthritis

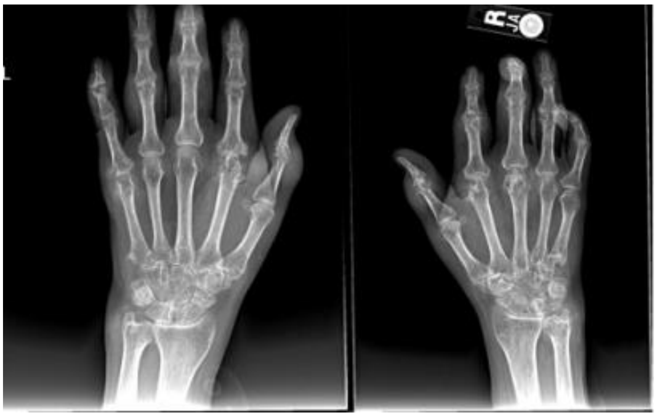
Note that the DIPs are predominately affected, rather than the MCPs and PIPs as would be seen with rheumatoid. Extensive juxta-articular periostitis is seen in the DIPs but the changes have not yet progressed to the classic 'pencil-in-cup' changes that are often seen with psoriatic arthritis.

Question 92 of 178

A 60-year-old man is referred to the rheumatology clinic with progressive pains in his hands. His past medical history includes hypertension for which he takes ramipril and indapamide. The referral letter includes the following bloods taken during a recent flare:

CRP	54 mg/l
Rheumatoid factor	negative
Adj calcium	2.51 mmol/l

An x-ray is taken:



What is the most likely diagnosis?

- A. Metastatic prostate cancer
- B. Rheumatoid arthritis
- C. Osteoarthritis
- D. Gout
- E. Primary hyperparathryoidism

D. Gout

The x-ray shows multiple periarticular erosions bilaterally with adjacent large soft tissue masses and relatively preserved joint spaces. In the right hand, these findings are most prominent at the 1st interphalangeal, 2nd-4th proximal interphalangeal, 1st-3rd metacarpohalangeal and carpometacarpal joints. In the left hand, the findings are most prominent at the ulnar styloid, scapholunate joint, first and fifth carpometacarpal joints, second and fifth metacarpophalangeal joints and 1st interphalangeal joint. These findings are consistent with gout.

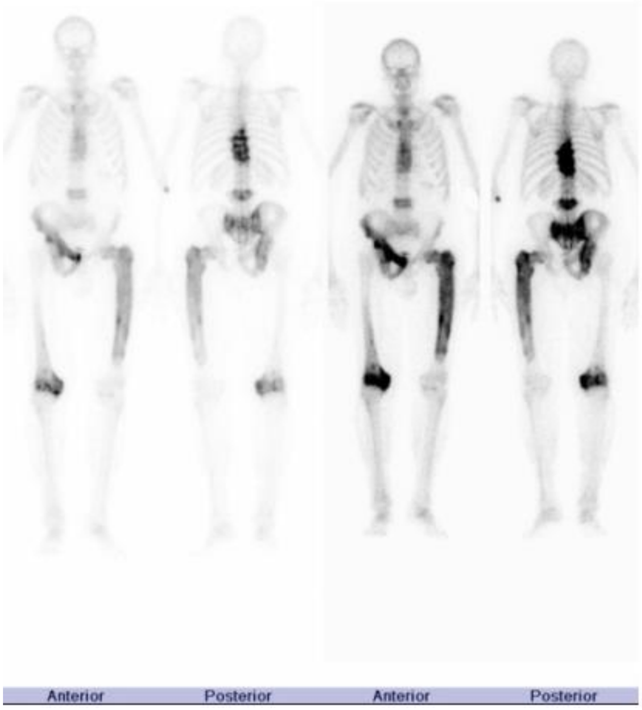
Indapamide is a risk factor for gout.

A normal calcium goes against primary hyperparathyroidism.

Question 93 of 178

A 76-year-old is investigated for persistent and progressive pain in his back and left hip. This is no longer responding to standard analgesia and has resulted in him taking regular modified-release morphine sulphate. Standard plain films of his left hip did not show changes consistent with osteoarthritis.

An isotope bone scan is therefore ordered to investigate his pain further:



What is the most likely cause of his pain?

- A. Multiple myeloma
- B. Metastatic prostate cancer
- C. Paget's disease of the bone
- D. Osteoporosis
- E. Ankylosing spondylitis

C. Paget's disease of the bone

The bone scan demonstrates intense uptake involving several lower thoracic vertebrae, L3, right hemipelvis, sacrum, left proximal femur and right knee. There is expansion and bowing of the involved femur. These changes are typical of Paget's disease.

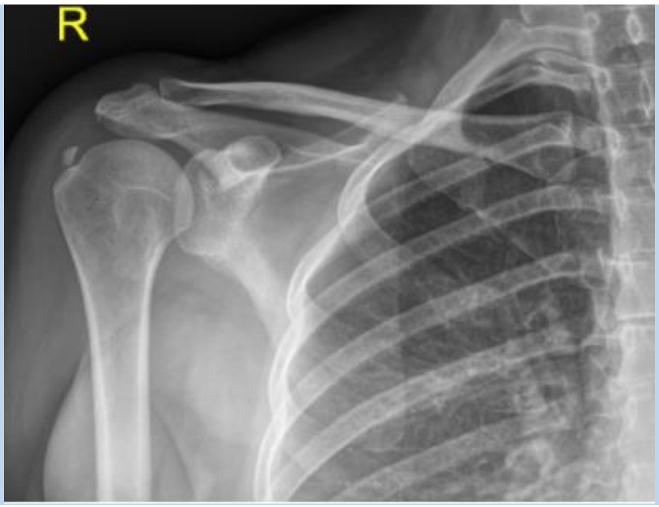
Isotope bone scans are not useful for asssessing multiple myeloma or osteoporosis

Question 94 of 178

A 48-year-old woman presents with progressively worsening pain in the right shoulder over the past few weeks. She is generally fit and well but smokes 20 cigarettes/day.

On examination there is diffuse mild tenderness over the lateral aspect of the right shoulder. The pain is recreated when abducting the should to around 70-80 degrees.

A shoulder x-ray is requested:



What is the most likely diagnosis?

A. Pancoast tumour
B. Supraspinatus tendonitis
C. Adhesive capsulitis
D. Humeral head fracture
E. Avascular necrosis

B. Supraspinatus tendonitis

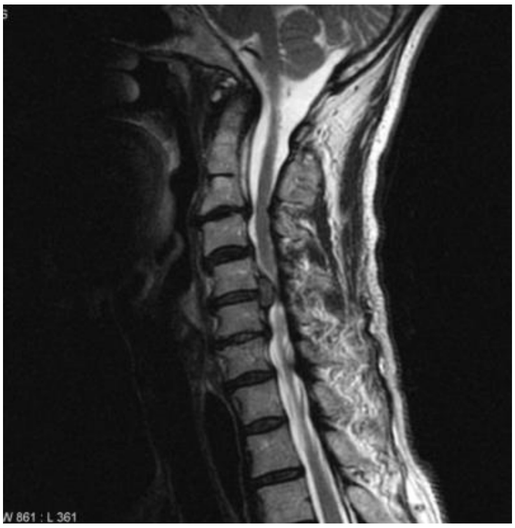
The x-ray shows calcification of the supraspinatus tendon consistent with prolonged inflammation. On examination the patient exhibits the classical 'painful arc' associated with this condition.

Question 95 of 178

A 37-year-old woman presents with left-sided pain shoulder pain. This has been present for around 6 months and is described variably as a 'toothache' or an 'electric shock' sensation which extends from her neck to the elbow.

On examination there is reduced power when abducting the left shoulder and reduced sensation to light touch just inferior to the deltoid muscle. The biceps reflex on the left side is absent. Pain and temperature sensation are normal in the left arm. Examination of the right arm is normal.

An MRI neck is requested:



What is the most likely diagnosis?

A. Bony metastases
B. Brown-Sequard syndrome
C. Arnold-Chiari malformation
D. Syringomyelia
E. Cervical disc prolapse

E. Cervical disc prolapse

This patient has signs consistent with a C5/6 myelopathy. Note the very large extruded disc at C5/6. There is also fusion of C2 and C3 with adjacent segment degeneration and cord volume loss suggesting myelomalacia.

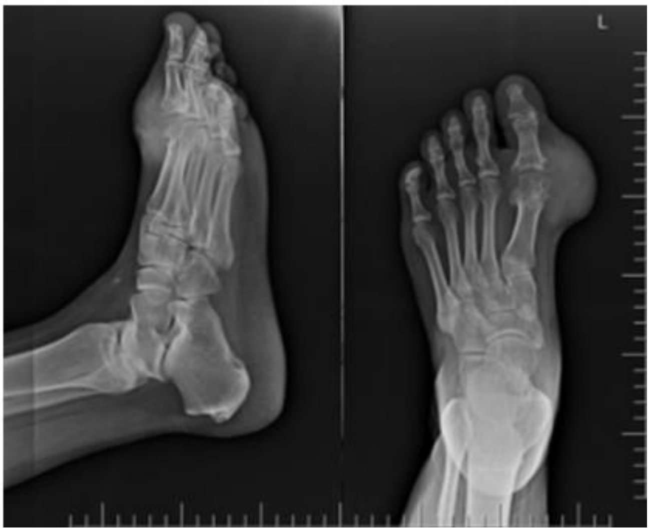
Question 96 of 178

A 40-year-old man presents to the Emergency Department with pain in his left foot. He thinks this may have been triggered by dropping a heavy box on it at work a few days ago. He is known to have type 2 diabetes mellitus which is managed with metformin. On examination there is erythema, tenderness and swelling in the distal, medial aspect of the left foot.

Bloods show the following:

Hb	145 g/l
Platelets	311 * 10 /l
WBC	6.3 * 10 /l
CRP	56 mg/l

An x-ray is requested:



What is the most appropriate management?

A. Naproxen
B. Allopurinol
C. Below-the-knee plaster cast
D. Intravenous antibiotics
E. Supportive stocking e.g. TubiGrip

A. Naproxen

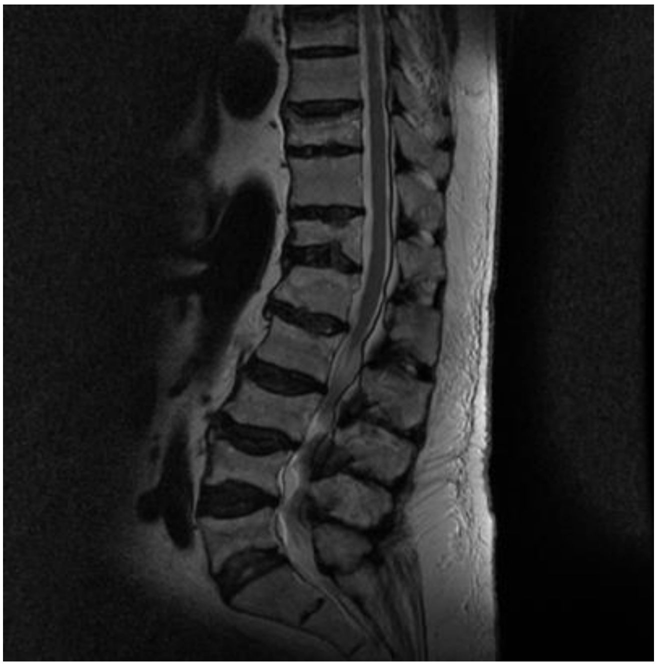
The x-ray demonstrates juxta-articular erosive changes around the 1st MTP joint with overhanging edges and associated with a moderate soft tissue swelling. The joint space is maintained. These findings are consistent with gout.

Allopurinol should not be used in the acute phase of gout. NSAIDs such as naproxen are generally used first-line, if there are no contraindications.

Question 97 of 178

A 71-year-old man with a history of chronic obstructive pulmonary disease (COPD) is investigated for back pain. Over the past 10 years he has had numerous admissions for infective exacerbations of COPD and currently uses long-term oxygen therapy. The pain came on suddenly whilst he was at his local supermarket.

A MRI scan is requested:



What is the most likely underlying cause of the back pain?

A. Osteomyelitis
B. Multiple myeloma
C. Pott's disease
D. Metastatic lung cancer
E. Osteoporosis

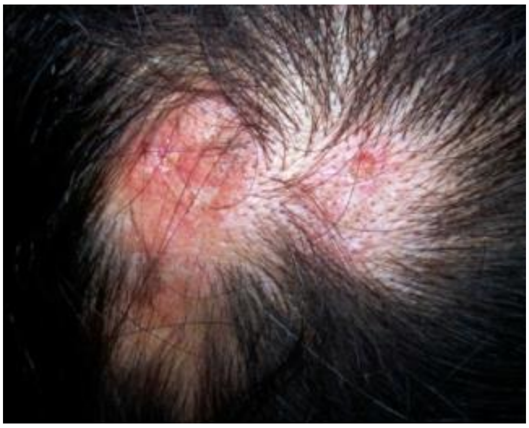
E. Osteoporosis

The MRI shows osteoporotic fractures of the 10th and 12th thoracic vertebrae. This is likely to have

been caused by repeated courses of steroids to treat exacerbations of COPD.

Question 98 of 178

A 35-year-old woman presents with alopecia. For the past few months she has noticed some 'scaly' patches on her scalp. Once healed they normally leave a scar and no hair seems to grow back. Her scalp has the following appearance:



What is the most likely diagnosis?

A. Discoid lupus erythematosus
B. Alopecia areata
C. Psoriasis

D. Trichotillomania
E. Squamous cell carcinoma

A. Discoid lupus erythematosus

Remember that alopecia may be divided into scarring (destruction of hair follicle) and non-scarring (preservation of hair follicle):

- Scarring alopecia
- trauma, burns
- radiotherapy
- lichen planus
- discoid lupus
- tinea capitis (if untreated)

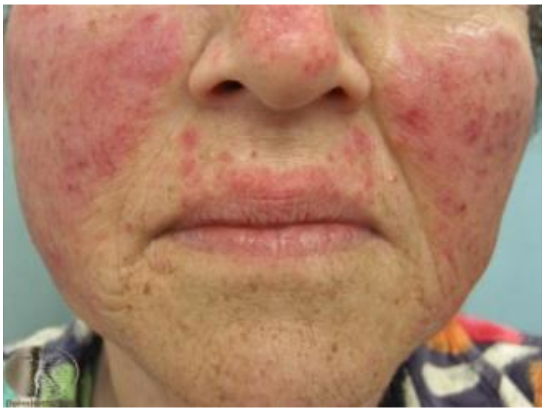
Non-scarring alopecia

- male-pattern baldness
- drugs: cytotoxic drugs, carbimazole, heparin, oral contraceptive pill, colchicine
- nutritional: iron and zinc deficiency
- autoimmune: alopecia areata
- telogen effluvium (hair loss following stressful period e.g. surgery)
- trichotillomania

Even if you are not familiar with the appearance of discoid lupus erythematosus (along with most non-dermatologists) this leaves it as the only possible answer.

Question 99 of 178

A 67-year-old woman presents with a rash. For the past two weeks she has felt tired and 'achey'. She also has a dry cough and some pleuritic chest pain. She is most concerned however with a new rash on her face:



Which drug is most likely to cause this presentation?

A. Procainamide
B. Digoxin
C. Sodium valproate
D. Methyldopa
E. Allopurinol

A. Procainamide

The correct answer is **Procainamide**. This patient's presentation of fatigue, dry cough, pleuritic chest pain, and a facial rash is suggestive of drug-induced lupus erythematosus (DILE). Procainamide is a well-known cause of DILE. It is an antiarrhythmic medication that can induce an autoimmune response with symptoms resembling systemic lupus erythematosus (SLE), including the characteristic malar rash seen in this patient.

Digoxin is incorrect because it is a cardiac glycoside used to treat heart failure and atrial fibrillation. Its side effects include gastrointestinal disturbances, visual disturbances, and arrhythmias; however, it does not typically cause a rash or symptoms resembling lupus erythematosus.

Sodium valproate is also incorrect. Sodium valproate is an anticonvulsant and mood stabilizer used in the treatment of epilepsy and bipolar disorder. Its side effects are mainly gastrointestinal, such as nausea and vomiting, as well as tremors, hair loss, and weight gain. It does not usually cause rashes or symptoms similar to lupus erythematosus.

Methyldopa is an alpha-2 adrenergic agonist used for the management of hypertension. Its side effects can include drowsiness, dry mouth, and headache but it does not typically cause rashes or symptoms resembling lupus erythematosus.

Finally, Allopurinol is incorrect. Allopurinol is a xanthine oxidase inhibitor prescribed for the treatment of gout by reducing uric acid production. While allopurinol can cause skin reactions such as a rash or Stevens-Johnson syndrome in rare cases, it does not generally lead to symptoms that resemble lupus erythematosus.

Question 100 of 178

A 54-year- old lady with a back pain is being managed with 1g paracetamol four times a day and ibuprofen which has been titrated up to the maximum dose of 2.4g/daily. Although she is tolerating this well, she is still complaining of ongoing pain.

What is the best option to improve her pain control?

A. Stop paracetamol, continue ibuprofen and commence naproxen
B. Continue paracetamol, continue ibuprofen and commence naproxen
C. Stop paracetamol, stop ibuprofen and commence morphine
D. Continue paracetamol, stop ibuprofen and commence naproxen
E. Continue paracetamol, continue ibuprofen and commence morphine

D. Continue paracetamol, stop ibuprofen and commence naproxen

Mild to moderate pain should be managed in a stepwise fashion:

Step 1 - paracetamol. Increase to the maximum dose of 1 gram four times a day, before switching to (or combining with) another analgesic.

Step 2 - substitute the paracetamol with ibuprofen and increase the dose of ibuprofen to a maximum of 2.4 grams daily

Step 3 - add paracetamol to ibuprofen

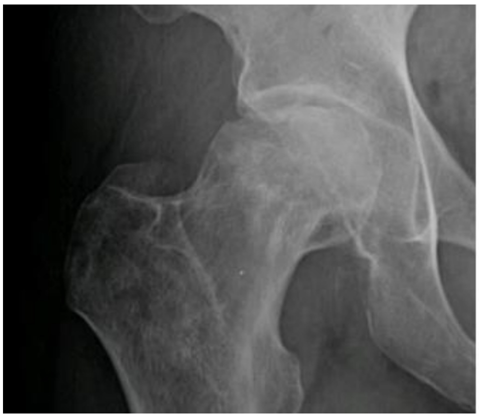
Step 4 - continue with paracetamol 1 gram four times a day. Replace the ibuprofen with an alternative NSAID (such as naproxen)

Step 5 - start a full therapeutic dose of a weak opioid in addition to full-dose paracetamol (1 gram four times a day) and/or an NSAID.

Two NSAIDs should not be used concomitantly as this increases the risk of adverse effects and it is too early to start using a strong opioid such as morphine for this patient. Therefore the best answer is to continue paracetamol and switch the ibuprofen to naproxen.

Question 1 of 79

A 60-year-old man presented with worsening right hip pain and decreased mobility. He has a history of colitis and is on long-term steroids. No other medical issues were reported. He lives alone and does not smoke. He drinks two bottles of wine daily and works as a truck driver. His hip x-ray shows:



What is the most likely diagnosis?

A. Avascular necrosis of hip
B. Occult fracture neck of femur
C. Osteoarthritis
D. Osteoporosis
E. Rheumatoid arthritis

A. Avascular necrosis of hip

The collapse of the articular surface resulting in the crescent sign (curvilinear lucent subchondral line) is characteristic of avascular necrosis of the hip. The reduced bone density in the proximal femur is suggestive of osteopaenia.

Avascular necrosis of hip is the correct answer. Osteonecrosis is also called avascular necrosis (AVN) or aseptic necrosis. Although it can occur in any bone, osteonecrosis most commonly affects the hip. It is more common in males. Risk factors include long-term steroid use, alcohol excess, and trauma.

Occult fracture neck of femur is incorrect. There is no evidence of disruption in the continuity of the femoral neck. The white line between the greater and lesser trochanter is the opacification from the intertrochanteric crest.

Osteoarthritis is incorrect. Osteoarthritis results in characteristic X-ray appearances, including joint space narrowing, osteophytes (bone spurs), articular surface cortical irregularity and sclerosis, and sub-cortical cysts (geodes) formation, which are not seen in this patient.

Osteoporosis is incorrect. While the proximal femur is suggestive of osteopaenia, the collapse of the articular surface resulting in the crescent sign is characteristic of avascular necrosis of the hip. In the context of the worsening hip pain, risk factors and x-ray findings, the most likely diagnosis is AVN.

Rheumatoid arthritis is incorrect. The radiographic hallmarks of rheumatoid arthritis are soft tissue swelling, osteoporosis, narrowing of the joint spaces and marginal erosions. Moreover, rheumatoid arthritis often affects the smaller joints first.

Question 2 of 79

A 54-year-old woman presents to the medical clinic with an itchy rash. She says that she has noticed a bluish-purple patchy rash mostly on sun-exposed areas. On examination, she has purple eyelids and rough raised purple areas on her knuckles Her nails show ragged cuticles, and blood vessels are seen on the nail fold. A purple, poorly defined rash is present on both her arms going up to her shoulders.

What is the likely diagnosis?

A. Dermatomyositis
B. Discoid lupus
C. SLE
D. Lichen planus
E. Lupus pernio

A. Dermatomyositis

The correct answer is dermatomyositis. The patient has a photosensitive rash, as well as a heliotrope rash around the eyelids, and a description of Gottron's papules. The distribution is also in keeping with a dermatomyositis rash. Lupus would normally have a macular erythematous and photosensitive butterfly rash over the face, and there can be a history of joint and neurological involvement. Lichen planus is a violaceous and itchy rash in patches, with a distribution similar to psoriasis.

Question 3 of 79

An 82-year-old woman with type 2 diabetes mellitus (T2DM), hypertension and chronic kidney disease (CKD) stage 4 is admitted under the geriatrics team with falls. Throughout her admission, her rehabilitation is significantly limited by bony pains. She is otherwise well with no systemic symptoms.

Calcium	1.82 mmol/L	(2.1-2.6)
Phosphate	2.12 mmol/L	(0.8-1.4)
ALP	# u/L	(30 - 100)
PTH	103 ng/L	(10-60)

What is the most likely explanation for her bony pains?

A. Bony metastases
B. Primary hyperparathyroidism
C. Sarcoidosis
D. Secondary hyperparathyroidism
E. Tertiary hyperparathyroidism

D. Secondary hyperparathyroidism

Low serum calcium, raised serum phosphate, raised ALP and raised PTH - chronic kidney disease leading to secondary hyperparathyroidism

In this scenario, the patient is experiencing bony pain, and her blood tests reveal hypocalcaemia, hyperphosphataemia, elevated ALP, and raised PTH. These biochemical findings are consistent with **secondary hyperparathyroidism**, likely resulting from her CKD stage 4. In CKD, reduced phosphate excretion leads to hyperphosphataemia, and diminished vitamin D activation causes hypocalcaemia. This, in turn, triggers a physiological increase in PTH levels in an attempt to raise serum calcium. Renal mineral bone disease often presents relatively early in CKD, with bone pain being a prominent feature. Treatment focuses on normalising phosphate levels through dietary measures or phosphate binders and correcting calcium levels with calcium supplementation and activated vitamin D.

Bony metastases are an unlikely cause of this biochemical picture. While bony metastases can In this scenario, the patient is experiencing bony pain, and her blood tests reveal hypocalcaemia, hyperphosphataemia, elevated ALP, and raised PTH. These biochemical findings are consistent with **secondary hyperparathyroidism**, likely resulting from her CKD stage 4. In CKD, reduced phosphate excretion leads to hyperphosphataemia, and diminished vitamin D activation causes hypocalcaemia. This, in turn, triggers a physiological increase in PTH levels in an attempt to raise serum calcium. Renal mineral bone disease often presents relatively early in CKD, with bone pain being a prominent feature. Treatment focuses on normalising phosphate levels through dietary measures or phosphate binders and correcting calcium levels with calcium supplementation and activated vitamin D.

Bony metastases are an unlikely cause of this biochemical picture. While bony metastases can increase ALP, they typically cause hypercalcaemia and a correspondingly suppressed PTH level.

In **primary hyperparathyroidism**, hypercalcaemia and an inappropriately elevated PTH are observed. Normally this is caused by a solitary parathyroid adenoma, but can also be caused by multiglandular parathyroid hypertrophy, ectopic parathyroid tissue or parathyroid cancer. Treatment options include medical management with cinacalcet, which mimics calcium to reduce PTH levels or surgical intervention with parathyroidectomy.

Sarcoidosis is a multisystem disease characterised by macrophage activation, with hypercalcaemia as a potential manifestation. Classic features include erythema nodosum, bilateral hilar lymphadenopathy, and hypercalcaemia. In sarcoidosis, hypercalcaemia is associated with a correspondingly suppressed PTH level, making the current biochemical picture more consistent with secondary hyperparathyroidism.

If left untreated, secondary hyperparathyroidism can progress to **tertiary hyperparathyroidism**. In this advanced state, longstanding secondary hyperparathyroidism leads to hypertrophy of the parathyroid glands, resulting in autonomous PTH production regardless of serum calcium levels. Tertiary hyperparathyroidism typically causes hypercalcaemia alongside elevated or normal PTH levels.

Question 4 of 79

A 6 year-old boy from Sierra Leone presents with a 1 week history of painful left arm. He is homozygous for sickle cell disease. On examination the child is pyrexial at 40.2ºC and there is bony tenderness over the left humeral shaft. Investigations are:

Hb	7.1 g/dL
Blood culture	Gram negative rods

X-ray left humerus: Osteomyelitis - destruction of bony cortex with periosteal reaction.

What is the most likely responsible pathogen?

A. Escherichia coli
B. Non-typhi Salmonella
C. Pseudomonas aeruginosa
D. Staphylococcus Aureus
E. Parvovirus B19

B. Non-typhi Salmonella

Blood and bone infections caused by non-typhi salmonella (NTS) are typically associated with malaria and homozygous sickle cell disease, especially in children. The reason for this perceived susceptibility is not fully understood - but it may be in part due to the haemolysis and subsequent iron availability to the bacteria, which is 'siderophilic' in nature.

E.coli and *P. aeruginosa* are not typically linked to sickle cell disease and *Staphylococcus aureus* is a gram positive coccus.

The haemoglobin level is normal for a child homozygous for sickle cell disease. Therefore 'aplastic anaemia' should not be considered and parvovirus can be ruled out. Parvovirus does not cause osteomyeltitis.

Question 5 of 79

A 77-year-old male presents to the Emergency Department with a two-day history of right temporal, throbbing headache, constant in nature and 8/10 severity. He reports this being the first ever episode of this headache and is different to his previous migraines, which have been typically in the left occipital region, lasting minutes, and fairly stereotyped over the past 60 years. Apart from migraines, he has no other medical history. On examination, his right scalp is tender and a prominent right temporal artery is noted. He is apyrexic with no skin rashes. His blood tests are as follows:

Hb	138 g/l
Platelets	552 * 10 /l9
WBC	11.5 * 10 /l9
ESR	85 mm/hr

Na +	146 mmol/l
K +	4.4 mmol/l
Urea	9.6 mmol/l
Creatinine	115 µmol/l
CRP	23 mg/l

You empirically start him on 60mg prednisolone. He undergoes temporal artery biopsy within 24 hours of his admission demonstrating no signs of temporal arteritis.

What is the most appropriate next step?

A. Repeat temporal artery biopsy
B. Continue prednisolone but at reduced dose 10mg OD
C. Discharge
D. Continue prednisolone at 60mg
E. Start anti-migraine medication

D. Continue prednisolone at 60mg

All the clinical features point to right temporal arteritis. Remember that a negative temporal artery biopsy does not rule out temporal arteritis! Shorter lengths of biopsy or removal of a 'skip lesion' result in a 9% false negative rate. There is no indication for a repeat temporal artery biopsy on the same or contralateral side, an ultrasound temporal artery may be helpful instead. Someone who has clinical temporal arteritis should thus maintain on high dose prednisolone to protect their vision. This headache is clearly different from the patient's normal migraines; anti-migraine medication is thus inappropriate

Question 6 of 79

A 32-year-old female with known rheumatoid arthritis presents to clinic and would like some advice. She would like to start a family with her partner. Her rheumatoid arthritis is current wellcontrolled on methotrexate and sulphasalazine, she has not required changing of doses for 2 years. She is reluctant to stop medications unless she has to, she had a number of flares when doses were reduced 3 years ago.

What would you advise regarding her plans for pregnancy?

A. She should reconsider her plans for pregnancy. Stopping medications would make her disease uncontrollable and continuing medications will affect her child
B. Continue sulphasalazine and methotrexate
C. Stop sulphasalazine, continue methotrexate
D. Continue sulphasalazine and stop methotrexate
E. Stop both sulphasalazine and methotrexate

D. Continue sulphasalazine and stop methotrexate

This is a relatively common scenario in rheumatology clinics: rheumatoid arthritis has a preponderance for females and a large number of RA patients are of child-bearing age . The first consideration is the need for treatment, then balancing the risks of disease flares and medical foetal toxicity. The majority of RA patients, as in most autoimmune disorders, experience improvements in their condition during pregnancy. Methotrexate is highly teratogenic and should be stopped between one and three months before pregnancy. Hydroxychloroquine and sulphasalazine are generally accepted to be DMARDs that can be continued during pregnancy. Sulphasalazine should, however, be avoided in male patients attempting conception due to the risks of oligospermia. Glucocorticoids cross the placenta in low doses and can be used after 14 weeks of pregnancy. Before this cut-off, there is an increased risk of cleft palate and gestational hypertension. Low doses of prednisolone is an option should flares occur when off methotrexate.

Question 7 of 79

A 56-year-old male presents with progressive bilateral tinnitus of gradual onset over the past 3 months. He also reports worsening hearing during the same time period, intermittent headache and increasing lower limb oedema. He denies diplopia, vertiginous symptoms, dysphagia or dysarthria. He denies any urinary symptoms or weight loss. There is a past medical history of hypertension and insulin-dependent diabetes. The patient also reports a family history of prostate carcinoma, with both his father and uncle previously undergoing resections. On examination, cranial nerves are unremarkable except bilateral hearing loss. You note no limb weakness but significant spinal kyphosis. Heart sounds I and II are heard and no added sounds. Bibasal crackles are auscultated. His abdomen is soft and non-tender. His initial serum markers are as follow:

Platelets	264 * 10 /l9
WBC	9 * 10 /l9
Neuts	5.4 * 10 /l9

Na +	142 mmol/l
K +	4.3 mmol/l
Urea	7.8 mmol/l
Creatinine	90 µmol/l

Bilirubin	6 µmol/l
ALP	902 u/l
ALT	28 u/l
CRP	16 mg/l

Parathyroid hormone and vitamin D normal range.

Which other biochemical marker will be abnormal?

A. Corrected calcium
B. Gamma glutamyltransferase (GGT)
C. Phosphate
D. C-telopeptide (CTx)
E. Prostate specific antigen (PSA)

D. C-telopeptide (CTx)

The patient describes a combination of hearing loss with tinnitus, signs of heart failure and associated skeletal deformities, associated with elevated alkaline phosphatase in the absence of other liver function test abnormalities. This is consistent with a diagnosis of Paget's disease, with skull complications (possible cochlear involvement or skull enlargement) resulting in hearing loss; high output heart failure and long bone deformities. Serum non-isomerised C-telopeptide (CTx) is an extremely sensitive marker of increased bone turnover observed in Paget's disease and is useful in monitoring disease progression or treatment efficacy . Diagnosis is often made on clinical features, radiological imaging and raised bone-specific ALP, with exclusion of other causes of bone pathology. CTx has largely superseded old tests of urinary hydroxyproline classically used.

Question 8 of 79

A 20-year-old male presents with a 4-day history of joint pain in both his wrists, left 2nd metacarpal-phalangeal (MCP) joint and right knee; blood in his urine and a new rash on his cheeks, which particularly bothers him. He also complains of chest pain of non-specific nature, onset about one week ago. On examination, you note bilateral swollen MCP joints, a hyperpigmented, raised erythematous rash on both cheeks. Neurological examination reveals a mild distal tremor at rest and activity, with bilateral KayserFleischer rings. He was diagnosed with Wilsons disease aged 18 years old and has no other past medical history. He is currently a research assistant and lives alone. His medications include ibuprofen as required, penicillamine started on diagnosis 2 years ago and he states he has been buying zinc supplements over the counter after reading in a journal that it may be helpful for his condition. Urine dip demonstrates 3+ blood, 1+ protein, no leucocytes or nitrites.

Which blood test is most likely to be diagnostic of his most recent admission?

A. Serum zinc
B. Urinary zinc
C. Anti-histone antibody
D. Anti-C1q antibody
E. Anti-double-stranded DNA antibody (dsDNA)

C. Anti-histone antibody

The patient describes haematuria, a new erythematous rash on sun-exposed regions and arthritis, on a background of previous penicillamine use: this is consistent with drug-induced lupus erythematosus (DLE). A number of medications are known to induce DLE, including penicillamine, procainamide, minocycline, hydralazine and a number of anti-epileptics. There is no specific diagnostic test for DLE: a combination of a drug known to induce DLE, the presence of ANA and resolution of symptoms on offending drug withdrawal. However, it is known that anti-histone antibodies are particularly sensitive to DLE, positive in up to 95% of DLE patients. Anti-C1q antibody can be present but is particularly predictive of lupus nephritis later in the disease. Unlike systemic lupus erythematosus (SLE), anti-dsDNA is often not present in DLE.

The patient's symptoms are not consistent with that of zinc poisoning, classically presenting with abdominal pain, vomiting and diarrhoea. The treatment of Wilsons disease is initially involves a copper-chelating agent, either penicillamine or trientene. Zinc is generally not used unless the patient is intolerant of either. Liver transplantation is also considered for those presenting in acute liver failure only.

Question 9 of 79

A 66-year-old Caucasian female presents with 3 week history of worsening headache and 2 day history of shortness of breath. She reports disturbed sleeping at night due to an inability to lie down, due to her shortness of breath. She has no known past medical history and drug history. On examination, you note bilateral splinter haemorrhages, 4 on the right and 2 on the left, with calcium deposits distally and black spots in the pulp of the fingers. Perioral skin puckering is also noted. Cardiovascular examination is unremarkable, chest examination reveals bilateral coarse inspiratory crackles. Neurological examination is unremarkable except fundoscopy revealing papilloedema, cotton wool spots and flame haemorrhages. The patient is apyrexic, Sats 95% on 2 litres, respiratory rate 24/min, blood pressure 195/115 mmHg, HR 90/min and regular. Chest x-ray demonstrates bilateral pleural effusion with bilateral alveolar shadowing.

What is the most important immediate management?

A. Oral amlodipine
B. Oral captopril
C. Intravenous labetalol
D. Oral high-dose prednisolone
E. Renal dialysis

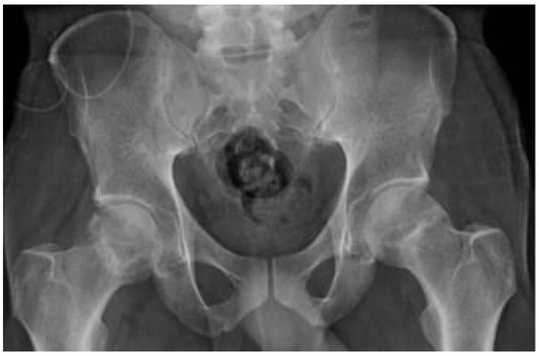
B. Oral captopril

This patient presents with signs of cutaenous manifestations of systemic sclerosis, grade 4 hypertensive retinopathy and heart failure. She is is scleroderma renal crisis (SRC), an emergency that if left untreated, is fatal. The optimal drug of choice is an ACE inhibitor, preferably captopril, which has been trialed with the greatest experience, but other ACEi are also likely to be beneficial. In a 15 year prospective cohort, one year survival increased from 15% to 76% with the use of ACEi against other anti-hypertensives . Steroids should be strictly avoided in SRC, they increase the risk of SRC prior to the event and may exacerbate SRC in the acute setting. Renal dialysis may be required in patients who progress to end-stage renal failure despite ACEi treatments.

Question 10 of 79

A 70-year-old woman presents with a 2-year history of worsening hip pain on the right side. This is worse on movement but also occurs at rest, including at night. She does not have any other joints affected. Her body mass index is 32 kg/m².

Her past medical history includes hypertension, mild asthma, and temporal arteritis. Her pelvic x-ray is shown below.



What is the most common risk factor associated with this condition?

A. Age
B. Bisphosphonate use
C. Inflammatory arthritis
D. Raised body mass index
E. Steroid use

E. Steroid use

This x-ray shows avascular necrosis of the femoral head on the right side - this is shown by the increased bone density, deformity, and flattening of the femoral head. The joint space is relatively preserved, differentiating this from osteoarthritis of the hip joint. Avascular necrosis is a type of osteonecrosis, which is due to damage to the arterial blood supply to the head of the femur. The most common cause is **steroid use**, particularly long-term steroid use. The patient in this scenario has temporal arteritis, which is often managed with prolonged high-dose steroids.

Age is incorrect. Increasing age is not a risk factor for AVN of the hip, outside of the presence of other risk factors. Around half of cases are present in patients less than 50 years old.

Bisphosphonate use is incorrect. Bisphosphonate use is associated with osteonecrosis of the jaw, but not the femoral head.

Inflammatory arthritis is incorrect. These conditions are not generally associated with AVN unless there is concurrent steroid use. There may be some association between systemic lupus erythematosus and AVN independent of steroid use, but this is much less common as a risk factor than steroid use in general.

Raised body mass index is incorrect. This is the most significant risk factor for osteoarthritis of the hip but is not a risk factor for AVN

Question 11 of 79

A 50-year-old patient comes in with a six-month history of polyarthralgia in her hands. Blood tests show she is rheumatoid factor positive, anti-CCP antibody positive and anti nuclear antibody positive with a high titre. An ultrasound scan confirms active synovitis in the metacarpophalangeal joints of her hands bilaterally.

What drug regime would you start this lady on?

A. Methotrexate and prednisolone
B. Methotrexate and hydroxychloroquine
C. Sulfasalazine and hydroxychloroquine
D. Sulfasalazine, hydroxychloroquine and prednisolone
E. Methotrexate, hydroxychloroquine and prednisolone

A. Methotrexate and prednisolone

This patient has seropositive rheumatoid arthritis. In 2018 NICE updated their guidelines. They now recommend

disease-modifying antirheumatic drug (DMARD) monotherapy with a short-course of bridging prednisolone. In the past dual DMARD therapy was advocated as the initial step

Question 12 of 79

A 24-year-old who is known to have psoriasis presents with arthralgia. She has noticed that her knuckles have become swollen and her psoriasis has got much worse over the last four months. On examination, she has severe plaque psoriasis on her extensors and scalp leading to alopecia. Her metacarpophalangeal joints are clearly swollen and tender. She is currently on naproxen 500mg BD, paracetamol 1g TDS, topical steroids and calcipotriol.

What medication would you add?

A. Leflunomide
B. Sulfasalazine
C. Hydroxychloroquine
D. Methotrexate
E. Infliximab

D. Methotrexate

Methotrexate is very effective at improving psoriatic arthritis but in addition to this it also has a dramatic effect on skin disease to a much greater extent than the other disease modifying antirheumatic drugs (DMARDs) listed. Therefore it is the DMARD of choice in psoriatic arthritis. If there was a contraindication to methotrexate leflunomide would be used second line for peripheral psoriatic arthritis. You would only use infliximab or another anti-TNF drugs first line if the patient had a predominantly axial spondyloarthropathy. This is according to the European League Against Rheumatism (EULAR) guidelines, please see the link: http://ard.bmj.com/content/71/1/4.full

Hydroxychloroquine can worsen skin disease and has little efficacy on psoriatic arthritis and is therefore not used routinely in the condition.

Question 13 of 79

A 30-year-old lady with rheumatoid arthritis, presents feeling unwell. She has felt lethargic for the last two days and over the last 24 hours has developed a severe sore throat. She has been on the following prescription for the last six months; paracetamol 1g QDS, naproxen 500mg PRN, methotrexate 15mg once weekly, folic acid 5mg once weekly and prednisolone 5mg OD. She is septic on examination with observations as follows: respiratory rate 26/min, heart rate 120/min, blood pressure 100/67mmHg, temperature 37.9ºC. She is tolerating oral fluids and small amounts of food. Adequate fluid resuscitation and antibiotics are started.

With regards to her regular medications what should be done?

A. Hold methotrexate and half dose of prednisolone
B. Hold methotrexate and increase prednisolone to 10mg once daily
C. Half dose of methotrexate and increase prednisolone to 10mg once daily
D. Refer to rheumatology for urgent review
E. Hold methotrexate and start IV methylprednisolone

B. Hold methotrexate and increase prednisolone to 10mg once daily

This lady should be presumed to be septic, she has been started on the relevant antibiotics and fluids as it states in the question. She needs her immunosuppression held and therefore no methotrexate should be given. Given that she is on long term steroids these need to be doubled to make sure she does not become Addisonian.

A referral to rheumatology would be appropriate following adequate resuscitation and treatment of the patient. Currently, her rheumatoid arthritis is not the priority.

Question 14 of 79

A 50-year-old lady presented with a 2 month history of breathlessness on exertion, increasing leg swelling and more recently a rash across her face. She was found to be in congestive heart failure, acute kidney injury and she was being treated with diuretics. Her echocardiogram showed severe left ventricular dysfunction. A few days into her admission she sustained a ventricular fibrillation cardiac arrest, which was successfully treated and the patient was started on amiodarone and betablocker. Her blood tests following the cardiac arrest did not show any electrolyte abnormalities. She also had a coronary angiogram which revealed normal coronary arteries. Subsequently, she was diagnosed with systemic lupus erythematosous and associated lupus myocarditis and she started appropriate immunosuppressive treatment for lupus.

What would be the best management for her ventricular fibrillation cardiac arrest?

A. Continue amiodarone indefinitely

B. Implantable cardioverter defibrillator immediately and stop amiodarone

C. Continue amiodarone and medical treatment of lupus and review progress in clinic todecide about implantable cardioverter defibrillator

D. Implantable cardioverter defibrillator immediately and amiodarone indefinitely

E. None of the above

C. Continue amiodarone and medical treatment of lupus and review progress in clinic todecide about implantable cardioverter defibrillator

This question aims to raise awareness about some of the aspects of the management of cardiac arrest survivors.

According to ACC/AHA/HRS 2008 guidelines, implantable cardioverter defibrillator (ICD) is indicated in survivors of ventricular fibrillation cardiac arrest after investigations to identify the cause of the arrest and exclude any reversible causes. Electrolyte abnormalities are among the usual reversible causes, but there is a variety of other reversible causes as well.

This patient sustained a cardiac arrest and had evidence of severe left ventricular systolic dysfunction. However, lupus myocarditis is a potentially reversible condition and so medical management is warranted before insertion of ICD. An ICD might be indicated in the near future if the patients left ventricular function does not improve following treatment of her underlying condition. This is something that can be reviewed in clinic with a repeat echocardiogram.

Amiodarone has a multitude of side effects and it should be continued indefinitely only if there is a justifiable reason to do so.

Question 15 of 79

A 30-year-old women with systemic lupus erythematosus is seen in the rheumatology clinic for annual follow-up. Recently she has felt well in herself and continues on hydroxychloroquine. She has not required additional steroid or analgesia for the last three years.

Her routine blood tests are as follows:

Hb	112 g/l	Na +	136 mmol/l
Platelets	252 * 10 /l 9	K +	4.9 mmol/l
WBC	6 * 10 /l 9	Urea	15 mmol/l
Neuts	4.5 * 10 /l 9	Creatinine	180 µmol/l
Lymphs	1 * 10 /l 9	CRP	23 mg/l

Her urine dipstick shows 3+ blood and 2+ protein.

Given that her renal function was previously normal, her rheumatologist refers her for ultrasound kidneys which shows normal sized kidneys with no hydronephrosis and normal renal artery dopplers.

Following discussion at MDT, it is recommended she undergo kidney biopsy for suspected lupus nephritis.

Which class of lupus nephritis would carry the worst prognosis?

A. Focal
B. Diffuse
C. Minimal mesangial
D. Mesangial proliferative
E. Membranous

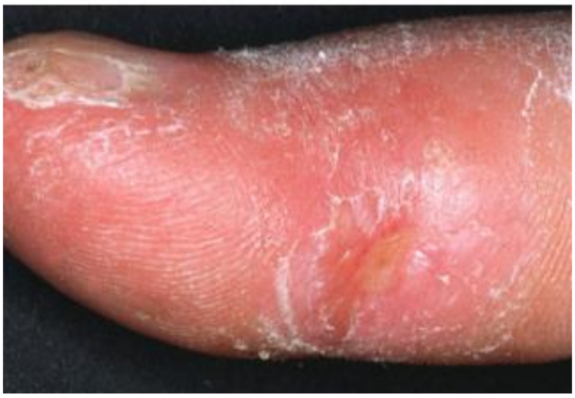
B. Diffuse

Lupus nephritis is split into 5 classes, of which class IV, diffuse proliferative, carries the worst mortality and renal outcome.

Question 16 of 79

A 45-year-old woman presents to the rheumatology clinic with a 6-week history of painful hands and fingers. Her past medical history includes hypothyroidism for which she takes levothyroxine 125 micrograms/day and a recent diagnosis of gastro-oesophageal reflux.

On examination, her observations are within normal limits. Her hands are examined, as shown below:



What investigation will most likely confirm the probable diagnosis?

A. Anti-CCP antibody
B. Anti-Jo antibody
C. Anti-centromere antibody
D. Anti-nuclear antibody
E. HLA-B27 testing

C. Anti-centromere antibody

Anti-centromere antibody is correct. This patient's finger is erythematous with dry and tightened skin with evidence of digital ulceration. The fingernails also appear fragile and brittle with ragged cuticles. The examination findings combined with the history of painful hands, and a recent diagnosis of gastro-oesophageal reflux disease consistent with systemic sclerosis. Systemic sclerosis (or scleroderma) is an autoimmune connective tissue disease that is characterised by the fibrosis of the skin and organs secondary to the overproduction of collagen. Other symptoms include vasculopathy with Raynaud's phenomenon. Multiple antibodies can be present in systemic sclerosis. However, one of the more specific antibodies for systemic sclerosis is the anti centromere antibody that is most commonly associated with limited cutaneous systemic sclerosis.

Anti-CCP antibody is incorrect. Anti-CCP is associated with rheumatoid arthritis. Rheumatoid arthritis can cause painful joints, particularly smaller joints in a symmetrical distribution. Dermatological features are less commonly seen and include rheumatoid nodules and erythema nodosum. Rheumatoid arthritis does not explain this patient's presentation.

Anti-Jo antibody is incorrect. This is positive in dermatomyositis which is an autoimmune inflammatory disorder characterised by myopathy and dermatological manifestations including skin lesions (e.g. heliotrope rash, Gottron's papules and nail fold capillary dilatation). This is not what is seen in this patient.

Anti-nuclear antibody is incorrect. Although ANA antibodies are present in up to 90% of patients with systemic sclerosis, it is not a specific finding as ANA antibodies are present in numerous inflammatory conditions.

HLA-B27 testing is incorrect. This is present in numerous seronegative spondyloarthropathies including ankylosing spondylitis and reactive arthritis. However, neither condition gives rise to the skin changes seen in this patient.

Question 17 of 79

A 37-year-old woman with known rheumatoid arthritis was reviewed at her annual follow-up at rheumatology clinic. The diagnosis of rheumatoid arthritis had been made ten years previously after the patient experienced severe inflammation of her meta-carpal phalangeal joints of both hands. Symptoms had been controlled with an initial reducing course of oral steroids and had been subsequently maintained on 15 mg of subcutaneous methotrexate weekly. She had experience one significant flare of her symptoms 18 months previously that had necessitated a single intra-muscular dose of corticosteroids.

On this occasion, the patient reported no further swelling, pain or redness of the joints of her hands or other joints. She did report however that over the past 6 months she had experienced on-going severe pains throughout her body. In addition, she had been feeling tired and lethargic and had been finding it hard to concentrate on her work at as a computer programmer. She denied any history of skin rashes, photosensitivity, hair loss, swallowing difficulties or dry eyes and she had not lost any weight.

The examination did not demonstrate any evidence of active synovitis. A minor ulnar deviation of the digits of both hands was noted which the patient denied caused her any functional impairment. The patient was noted to be tender on palpation of the muscles of her arms, legs and paraspinal muscles. However, there was no associated muscle weakness with patient able to rise unaided from a chair without using the assistance of her arms. There was no thickening of the skin of the hands or face. The cardiovascular, respiratory and abdominal examination was unremarkable and there were no skin rashes.

Investigations requested following clinic review are listed below.

Haemoglobin	134 g / L
White cell count	6.6 * 10 /l
Platelets	198 * 10 /l
Sodium	139 mmol / L
Potassium	4.3 mmol / L
Urea	4.8 mmol / L
Creatinine	75 micromol / L
Erythrocyte sedimentation rate	15 mm / h
Rheumatoid factor	Positive
Anti-nuclear antigen	Negative
Anti-citrullinated protein antibodies	37 units (reference < 20)

What is the likely cause of the patient's new symptoms?

A. Flare of rheumatoid arthritis
B. Mixed connective tissue disease
C. Chronic regional pain syndrome
D. Inclusion body myositis
E. Fibromyalgia

E. Fibromyalgia

The patient has chronic widespread pain associated with lethargy and difficulty concentrating and multiple tender points on palpation. The patient has immunological results consistent with her previous diagnosis of rheumatoid arthritis but no clinical or biochemical evidence of a flare of this disease or the development of a new connective tissue disease or myositis. Chronic regional pain syndrome is associated with persistent burning pain in one limb, usually after a minor injury.

The patient's symptoms are consistent with the diagnostic entity known as fibromyalgia. It is important to be aware that fibromyalgia is not a diagnosis of exclusion and can co-exist with other diseases as in this case.

Question 18 of 79

A 29-year-old woman is referred to Rheumatology clinic after experiencing all over pain throughout her body over the past two years. This has been associated with not feeling refreshed in the morning after a nights sleep and the patient finding difficulty in concentrating on her work in a call centre. She denied any history of skin rashes, photosensitivity, hair loss, swallowing difficulties or dry eyes and she had not lost any weight. Past medical history was significant for a previous diagnosis of mild depression treated with a course of cognitive behavioural therapy. There was no family history of connective tissue disease. The patient lived with her husband and two young children and reported some on-going concerns over the family finances. She smoked 10 cigarettes per day but rarely drank alcohol.

Initial review of the patient had demonstrated no evidence of inflammatory arthritis but showed the patient had significant muscular tenderness at multiple sites throughout the body.

Initial blood tests requested after clinic review had been unremarkable and included negative rheumatoid factor and negative anti-nuclear antibody. X-rays of the patient's hands and feet did not demonstrate any evidence of erosive arthropathy.

At a follow-up review of the patient with the above results, it was discussed that no evidence of inflammatory arthritis had been uncovered and that the patient's symptoms were most likely consistent with fibromyalgia. Given her previous experience with cognitive behavioural therapy, the patient was keen to adopt positive lifestyle strategies to reduce her symptoms rather than pharmacological treatment.

What treatment is most likely to be effective for this patient?

A. Strength training
B. Balneotherapy
C. Aerobic exercise
D. Electrotherapy
E. Acupuncture

C. Aerobic exercise

Many non-pharmacological therapies for fibromyalgia have been tried although evidence for their effectiveness is limited. Aerobic exercise has the strongest evidence of benefit with a Cochrane review showing that regular aerobic exercise improved wellbeing, aerobic capacity, tenderness and pain compared with no exercise. Strength training has also been shown to have some benefit but with a lower quality of evidence than for aerobic exercise.

There is only weak evidence to support passive physical therapies such as electrotherapy or balneotherapy (hot spa treatments). Acupuncture is often used in fibromyalgia although evidence of long-term benefit is available.

Question 19 of 79

A 55-year-old man is referred to rheumatology for management of severe tophaceous gout. The patient had been experiencing intermittent gout attacks over the previous few years, typically affecting the first metatarsophalangeal joints of both feet. However, during the last two months the patient had developed inflammation of multiple small joints of his hand preventing the patient from continuing his work as a train driver. A trial of Colchicine prescribed by the patient's General Practitioner had been discontinued after the patient experienced severe diarrhoea. Past medical history included an upper GI bleed secondary to a duodenal ulcer six months previously.

Examination demonstrated severe asymmetrical inflammation of multiple metacarpalphalangeal, distal interphalangeal and proximal interphalangeal joints across both hands. Yellow-white tophi were present across the inflamed joints. Blood tests taken prior to clinic attendance are listed below.

Hb	15.2 g/dl
Platelets	265 * 10 /l9
WBC	6.5 * 10 /l9

Na +	134 mmol/l
K +	4.2 mmol/l
Urea	9.5 mmol/l
Creatinine	175 µmol/l
eGFR	62 ml/min
Urate	370 µmol/l

What is the best treatment for this patient's acute gout?

A. Intra-articular steroid injection
B. Naproxen
C. Allopurinol
D. Febuxostat
E. Short course prednisolone

E. Short course prednisolone

The best option in this case is a short course of oral prednisolone (30 mg daily for five days). Two randomised controlled trials have shown this treatment to have a similar efficacy compared to NSAIDs. Intra-articular steroid injection is felt to be an effective treatment for acute gout affecting large joints, but is less appropriate for treatment of multiple small joints of the hands. Naproxen would be contra-indicated in this case due to the history of peptic ulceration and renal impairment.

Allopurinol and Febuxostat are both used to lower serum urate levels as part of gout prophylaxis and have no role in the treatment of an acute attack.

Question 20 of 79

A 78-year-old female falls whilst taking out the bins. She immediately has severe right hip pain and her right leg is shortened and externally rotated. She is taken to the Emergency Department where an X-ray is performed, which confirms a right-sided intracapsular neck of femur fracture. She has definite operative management the following day.

Following surgery, how should this patient's bone health be managed to reduce the risk of further fragility fractures?

A. DEXA scan before prescribing
B. MRI scan of the spine and pelvis before prescribing
C. Start alendronate, no imaging required
D. Start calcium and vitamin D, no imaging required
E. Start calcium and vitamin D whilst awaiting a DEXA scan

C. Start alendronate, no imaging required

Start alendronate in patients >= 75 years following a fragility fracture, without waiting for a DEXA scan

In patients over the age of 75 who present with a fragility fracture a diagnosis of osteoporosis is presumed and does not need to be confirmed with imaging. Starting first-line therapy without a DEXA scan is recommended in this age group. First-line therapy is currently alendronate 70 mg once per week.

In patients under 75 years who present with a fragility fracture, a DEXA scan is recommended prior to prescribing alendronate. With the results of the DEXA scan, a FRAX assessment is completed to assess the patient's future fracture risk and provides guidance on prescribing on this basis. The patient in this question is over 75-years-old.

The preferred method of imaging to assess the severity of osteoporosis is a DEXA scan rather than an MRI scan. An MRI scan may be useful in the context of a pathological malignancy when underlying malignancy is suspected.

Vitamin D and calcium replacement is not an adequate treatment for osteoporosis to prevent further fragility fractures.

As above, vitamin D and calcium replacement is not an adequate treatment for osteoporosis to prevent further fragility fractures. Furthermore, this patient is over 75-years-old and therefore does not require a DEXA scan to make the diagnosis of osteoporosis.

Question 21 of 79

A 64-year-old male presents with known lumbosacral poly-radiculopathy caused by neurosarcoidosis is on immunosuppression with high dose steroids for several months and methotrexate. He is admitted with breathlessness and a dry cough. He is found to have a type 1 respiratory failure. A CT chest shows ground glass shadowing suggesting *Pneumocystis jirovecii* pneumonia. You discuss with the microbiology team who suggest also covering the patient for a bacterial pneumonia and viral pneumonitis.

Which of the following medications could have a potentially life threatening interaction with his current medications?

A. Tazocin
B. Clarithromycin
C. Co-trimoxazole
D. Ciprofloxacin
E. Aciclovir

C. Co-trimoxazole

Co-trimoxazole contains trimethoprim and therefore should never be prescribed with methotrexate

Co-trimoxazole is a combination of sulfamethoxazole and trimethoprim. These are bacteriostatic antibiotics that work by interfering with bacterial folate metabolism. However, they can also affect the hosts folate metabolism. Methotrexate is a dihydrofolate reductase inhibitor. Therefore, together these medications can cause potentially life-threatening bone marrow suppression secondary to folate deficiency.

The BNF does not list interactions between corticosteroids and the remaining medication listed. Ciprofloxacin may possibly reduce methotrexate excretion, so monitoring for signs of potential toxicity needs to be undertaken. No interactions are listed in the BNF between methotrexate, and tazocin, aciclovir and clarithromycin.

Question 22 of 79

A 38-year-old Armenian visitor presents with 3 day history of pyrexia, shortness of breath, chest pain and abdominal pain, associated with temperature of 38.5 degrees. She has no other known past medical history and reports at least 2 other episodes of similar pain, both times spontaneously resolving without treatment or diagnosis. On examination, she has a pleural rub and a swollen, tender left 3rd metcarpal-phalangeal joint. Her mother has recently been admitted for similar symptoms last month. Her blood tests are as follow:

Hb	14.5 g/dl
Platelets	560 * 10 /l9
WBC	17.8 * 10 /l9

Na +	143 mmol/l
K +	4.6 mmol/l
Urea	5.2 mmol/l
Creatinine	78 µmol/l
CRP	78 mg/l

A chest radiograph demonstrates mild bilateral pleural effusions with no significant focus of consolidation, her Mantoux test is negative. Urine dip is negative, urine MC+S grows no organisms, urinary porphobilinogen is negative. A rheumatology review was requested regarding the synovitis and colchicine prescribed. She responds well with resolution of all symptoms within 24 hours. An infectious diseases opinion and induced sputum is awaited.

What is the most likely diagnosis?

A. Tuberculosis
B. Acute intermittent porphyria (AIP)
C. Coxsackie B virus infection
D. Familial mediterranean fever
E. Systemic lupus erythematous (SLE)

D. Familial mediterranean fever

Familial Mediterranean fever (FMF) is an autosomal recessive, hereditary inflammatory disorder characterised by reoccurring episodes of abdominal pain, fever, arthralgia, and chest pain

The patient is of Mediterranean descent is experiencing an acute attack of abdominal pain, chest pain, synovitis and pyrexia with an acute phase response. There appears to be a previous history of similar symptoms and a possible genetic element. Familial Mediterranean fever would fit with all these symptoms, with almost all patients presenting with abdominal pain, pleuritis and synovitis, associated with fever greater than 38 degrees. The main differentials in this case are with SLE and AIP: during an acute event, urinary porphobilinogen is likely positive in AIP. The distinguishing feature against SLE is the resolution of symptoms with colchicine, which is a key diagnostic feature

Question 23 of 79

A 65-year-old gentleman is reviewed in rheumatology clinic, as he has been referred by his general practitioner regarding his recurrent gout. The patient had already been started on allopurinol due to recurrent attacks of gouts. Unfortunately he was unable to tolerate it, as he developed a rash. In addition, he was not happy with the gastrointestinal upset that it caused him. Furthermore, his eGFR (see bloods) does not permit an increase of allopurinol above 100 mg OD.

On examination today you note an obese gentleman who is otherwise asymptomatic. He informs you that in the past the gout attacks affected his 1st metatarsophalangeal joint in the left foot and his right knee.

He is known to suffer from hypertension, type II diabetes mellitus and has had previous myocardial infarction. He is on aspirin 75 mg OD, ramipril 3.25 mg OD, amlodipine 5 mg OD, atorvastatin 80 mg OD, bisoprolol 1.25 mg OD, metformin MR 1 g OD.

You review his bloods and his previous aspiration results.

3 months prior

Na^+	137 mmol/l
K^+	4.3 mmol/l
Urea	6.8 mmol/l
Creatinine	123 µmol/l

Now

Na^+	138 mmol/l
K^+	5.2 mmol/l
Urea	10.3 mmol/l
Creatinine	235 µmol/l

What options exist with regards to his long-term management of gout?

A. Continue with allopurinol 100 mg OD
B. Initiate febuxostat 80 mg OD
C. Initiate indomethacin MR 75 mg OD
D. Initiate prednisolone 30 mg OD
E. Initiate colchicine 500 micrograms BD

B. Initiate febuxostat 80 mg OD

Febuxostat can be used in the prevention of gout in patients where the allopurinol side effects have been deemed intolerable or the renal function does not allow sufficient dose escalation

NICE guidelines alongside the British Society of Rheumatology guidelines on the management of gout suggest that febuxostat is an alternative to allopurinol in cases where the side-effects are deemed bad by the patient and no effective dose adjustment to 300 mg OD has been made due to declining renal function. Febuxostat is a non-purine selective xanthine oxidase inhibitor, inhibiting oxidised and reduced versions of the enzyme. It does not inhibit purine or pyrimidine metabolism as allopurinol does. Recent studies do mention that there may be an elevated risk of cardiovascular events with the use of febuxostat, however no change in guidelines has been made. Indomethacin (an NSAID), prednisolone and colchicine are all useful in the acute treatment of gout, however their preventative use is limited. Colchicine can be used as cross cover while a xanthine oxidase inhibitor is first prescribed to avoid acute gout.

Question 24 of 79

A 67-year-old female presented to the accident and emergency department with severe headache and shortness of breath for the last six hours followed by seizures which occurred twice during the last hour.

The patient is a known case of diffuse cutaneous systemic sclerosis diagnosed two years ago and she is on steroids and cyclophosphamide.

On examination, she looks ill, agitated and dyspnoeic. Her pulse rate is 100 beats per minute, regular and her blood pressure is 220/110 mmHg.

Her JVP is raised, there is a gallop rhythm and bilateral basal crackles. There is lower limb oedema and brisk reflexes.

Fundoscopy showed grade 3 hypertensive retinopathy.

Investigations done two weeks previously showed:

Serum sodium	140 mmol/L
Serum potassium	5.7 mmol/L
Serum urea	17 mmol/L
Serum creatinine	250 mol/L
Urinalysis	protein ++, blood ++

What is the most appropriate immediate treatment to lower her blood pressure?

A. IV sodium nitroprusside
B. IV labetalol
C. Oral ACE inhibitor
D. IV hydralazine
E. Nitrate infusion

C. Oral ACE inhibitor

This patient has developed scleroderma renal crisis which presents as malignant hypertension, heart failure and rapid deterioration of renal function progressing to acute renal failure.

This hypertensive emergency should be managed with gradual reduction of blood pressure at a rate of 10-15 mmHg per day with an oral ACE inhibitor as the pathology of scleroderma renal crisis is vasospasm.

IV sodium nitroprusside and IV labetalol should be avoided as they lead to sudden reduction of blood pressure and renal hypoperfusion that leads to acute tubular necrosis, making the already deranged renal function even worse.

Indeed, this patient requires ICU admission for management of her heart failure and acute renal failure.

Question 25 of 79

A 35-year-old woman presents to rheumatology clinic with a 2-month history of symmetrical swelling of the ankles and fingers. She also complains of joint pain and stiffness. The stiffness is primarily worse in the early morning and eases with use. Apart from a recent sore throat, she is otherwise well. She has a family history of type 1 diabetes mellitus. She does not take any prescribed medication but has found herself relying on over-the-counter analgesics to get through the day.

On examination, she has bilateral swelling of the index, ring and middle fingers and bilateral ankle swelling. She has a full range of movement in the fingers, wrists and ankles. There is marked swelling and tenderness to palpation at the distal interphalangeal joints in the index, middle and ring fingers on both sides. There are no skin changes, but yellowing and pitting of the nails are noted.

Blood tests show:

Hb	11.1 g/dl
Platelets	305 * 10 /l
WBC	7.8 * 10 /l

Na	141 mmol/l
K	4.2 mmol/l
Urea	5.8 mmol/l
Creatinine	64 µmol/l

Bilirubin	13 µmol/l
ALP	83 u/l
ALT	15 u/l
ESR	50mm/hr
CRP	39 mg/L
Rheumatoid factor	negative

Hand X-ray shows mild erosion at the distal interphalangeal joints of the index, middle and ring fingers on both hands.

What is the diagnosis?

A. Rheumatoid arthritis
B. Reiters syndrome
C. Ankylosing spondylitis
D. Yellow nail syndrome
E. Psoriatic arthritis

E. Psoriatic arthritis

Although the patient does not have a psoriatic rash, she has classic symptoms of psoriatic arthritis. She has dactylitis and distal interphalangeal swelling, as well as ankle involvement. Nail signs are well-documented in psoriasis. The diagnosis is clinched by the negative rheumatoid factor and raised inflammatory markers. A slightly low haemoglobin is also a common feature of the disease. The pattern of joint involvement points more towards psoriatic arthritis than rheumatoid arthritis, in which the metacarpophalangeal joints and wrists are more commonly affected. Reiter's syndrome is a reactive arthritis that typically follows a gastrointestinal or venereal infection. Conjunctivitis and urethritis are seen alongside arthritis. Back pain would be expected to accompany ankylosing spondylitis. Yellow nail syndrome is a rare disorder of uncertain pathogenesis. It presents with nail discolouration, lymphoedema and pleural effusions.

Psoriatic arthritis can manifest in the absence of skin signs, particularly if the patient has a family history of psoriasis. The patient may develop a rash later or have signs limited to the nails.

Question 26 of 79

A 28-year-old woman with systemic lupus erythematosus attends the pre-conception clinic. She would like some advice regarding her medications prior to getting pregnant. She has never been pregnant before and her lupus has been stable on her current medications: mycophenolate and hydroxychloroquine for over 12 months. She also has asthma, which is well controlled with beclomethasone and salbutamol inhalers, and she takes regular omeprazole for gastrooesophageal reflux.

What is the most appropriate medication amendment?

A. Half omeprazole dose
B. Stop beclomethasone inhaler
C. Stop hydroxycholorquine
D. Add ramipril 1.25mg
E. Change mycophenolate to azathioprine

E. Change mycophenolate to azathioprine

Unlike the majority of autoimmune conditions, pregnancy increases the likelihood of a lupus flare. It is essential that a patients lupus is well controlled and quiescent for at least six months prior to pregnancy. Given that mycophenolate is teratogenic in pregnancy, it must be stopped. It is common practice to change to azathioprine, which has been shown to be safe in pregnancy - but still used with caution! However knowledge of common medications used/not used in pregnancy should also lead the candidate to this option.

There is no need to stop the beclomethasone or hydroxycholoquine, as they are both safe in pregnancy. Omeprazole is also safe in pregnancy and the dose does not need to be changed. ACE inhibitors are contraindicated in pregnancy and must be stopped/changed to another agent.

Question 27 of 79

A 58-year-old gentleman of Afro-Caribbean origin presents to clinic. He complains of lethargy, fever and joint pain, particularly in both knees. He has also noticed an intermittent rash on his arms and his neck. These symptoms have progressed over the last six months. He has a past medical history of congestive cardiac failure and chronic renal failure secondary to hypertension. His medication includes bisoprolol, aspirin and hydralazine/isosorbide dinitrate.

On examination the patient has no evidence of joint swelling. He has discrete patches of scaling on his neck and forearms. He has normal power in his arms and legs.

The following blood tests were obtained;

Rheumatoid Factor	Positive
Anti-nuclear antibody	Positive
Anti-single stranded DNA	Positive
Anti-extractable nuclear Antigen	Negative
Anti-histone antibodies	Positve

What is the most likely diagnosis?

A. Polymorphous Light Eruption
B. Dermatomyositis
C. Drug induced lupus erythematous
D. Rheumatoid arthritis
E. Systemic lupus erythematosus

C. Drug induced lupus erythematous

A polymorphous rash would expect to be erythematous and less associated with systemic symptoms. Dermatomyositis may have a positive ENA (anti-Jo1) with heliotrope rash and Gottrons papulses. There also may be associated muscle weakness. Rheumatoid arthritis would expect to show arthritis and is less associated with a rash and the antibody picture. SLE is more associated with double stranded DNA and ENA.

Drug induced lupus can be caused by a variety of drugs but most commonly antihypertensives and antifungals. Specific examples include isoniazid, hydralazine, procainamide, diltiazem and phenytoin. Anti-histone antibodies are associated with drug-induced lupus in most cases.

Question 28 of 79

An 18 year-old girl with neuropsychiatric Wilsons disease is commenced on chelation therapy.

One month later she presents with fatigue, generalised arthralgia, and an erythematous rash affecting the face, back of the neck, shoulders, and arms.

Which test is most specific for the suspected diagnosis?

A. Anti-centromere antibodies
B. Anti-Scl70 antibodies
C. Anti-mitochondrial antibodies
D. Anti-histone antibodies
E. Anti-dsDNA antibodies

D. Anti-histone antibodies

This is drug-induced lupus as a result of penicillamine chelation therapy.

Anti-histone antibodies are specific for drug-induced lupus.

Other unusual side-effects of penicillamine include membranous glomerulonephritis and a myasthenia-like syndrome.

Other causes of drug-induced lupus include procainamide, minocycline, hydralazine, and isoniazid. Anti-centromere antibodies are found in limited systemic sclerosis.

Anti-Scl70 antibodies are found in diffuse systemic sclerosis.

Anti-mitochondrial antibodies are specific for primary biliary cirrhosis.

Anti-dsDNA antibodies are specific for systemic lupus erythematosus.

Question 29 of 79

A 52-year-old man presents with lethargy and reduced sensation in both feet. He reports a 2 month history of fevers and 4kg weight loss. He also reports intermittent testicular pain.

On examination there is livedo reticularis on both legs and reduced light touch and pain sensation on both feet.

Blood tests reveal:

Hb	116 g/l	Na +	137 mmol/l	Bilirubin	18 µmol/l
Platelets	487	K +	4.8 mmol/l	ALP	92 u/l
WBC	8.3	Urea	12.8 mmol/l	ALT	102 u/l
Neuts	6.3	Creatinine	182 µmol/l	γGT	16 u/l
MCV	89 fL	ESR	78mm/hr	Albumin	35 g/l

What is the most likely diagnosis?

A. Cryoglobulinaemia
B. Granulomatosis with polyangiitis
C. Eosinophilic granulomatosis with polyangiitis
D. Polyarteritis nodosa
E. Microscopic polyangiitis

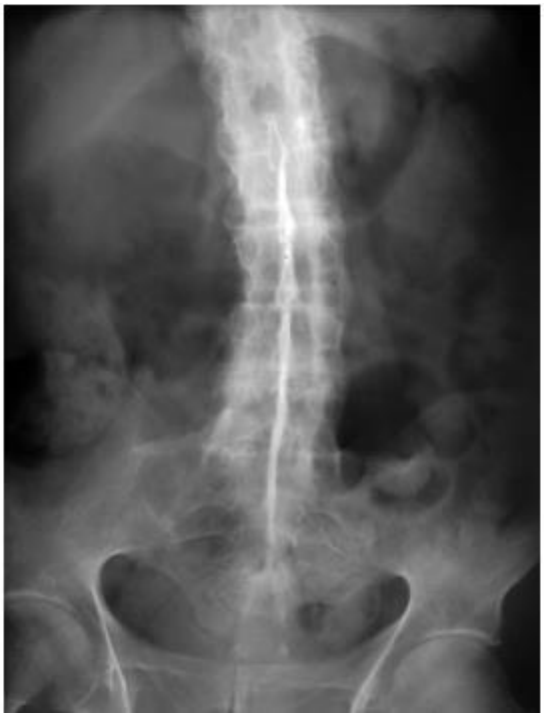
D. Polyarteritis nodosa

Fever, lethargy, neuropathy, testicular pain and renal dysfunction are consistent with polyarteritis nodosa (PAN).

All systemic vasculitides can present with fever and lethargy, however polyarteritis nodosa is the systemic vasculitis most frequently associated with testicular involvement.

Question 30 of 79

A 40-year-old man is investigated for back pain. For the past few months he has been troubled with pain in his lower back which is typically worse in the morning and better by the end of the day. There is some radiation of pain to the right buttock but no leg pains. An x-ray of his lumbar spine is shown below



What is the most likely cause of his back pain?

A. Lumbar disc prolapse at multiple levels
B. Osteopetrosis
C. Calcification of the vertebral artery
D. Spinal stenosis
E. Ankylosing spondylitis

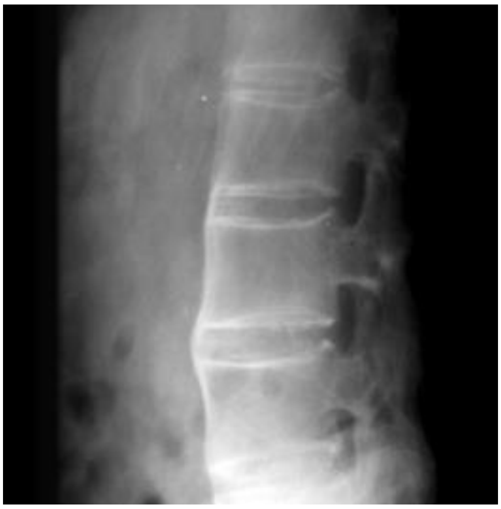
E. Ankylosing spondylitis

This image shows the typical appearance of bamboo spine with a single central radiodense line related to ossification of supraspinous and interspinous ligaments which is called dagger sign. Ankylosing is detectable in both sacroiliac joints.

Note the history of morning pain is typical for an inflammatory arthritis such as ankylosing spondylitis.

Question 31 of 79

A 26-year-old woman presents with a four month history of back pain. The pain is located around the lower lumbar vertebrae and spreads to both buttocks. Ibuprofen and walking seem to improve the pain. A lumbar spine film is requested:



What is the most likely cause of this patients back pain?

A. Marble bone disease
B. Discitis
C. Ankylosing spondylitis
D. Facet-joint dysfunction
E. Rheumatoid arthritis

C. Ankylosing spondylitis

Ankylosing spondylitis with well formed syndesmophytes are seen on the lumbar spine film.

The first thing to address is the sex of the patient. Of course ankylosing spondylitis is more common in men but the male-to-female ratio is only 3:1. This means it is reasonable to be asked about female patients in questions, particularly if there is accompanying 'hard evidence' such as x rays.

Marble bone disease (osteopetrosis) results in dense, thick bones that are prone to fracture. Syndesmophytes are not a feature.

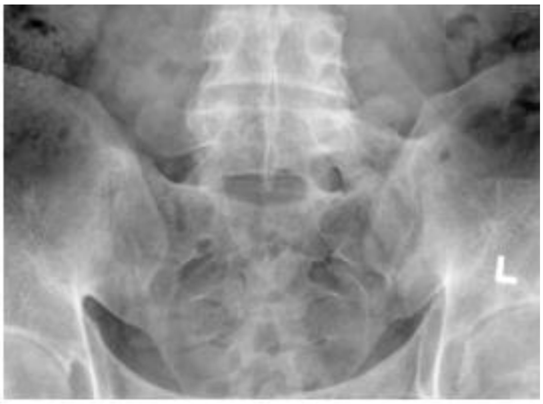
Facet-joint dysfunction is a common cause of back pain but it would not explain the x-ray findings.

Rheumatoid arthritis of course does not commonly present with back pain. The following x-ray changes are typically seen:

- loss of joint space
- juxta-articular osteoporosis
- soft-tissue swelling
- periarticular erosions
- subluxation

Question 32 of 79

A 56-year-old man presents with buttock pain. This has been present for many years but has recently become worse. The pain is usually worse in the early part of the day and often eases by the late afternoon. An x-ray is requested:



What is the most likely underlying diagnosis?

A. Ankylosing spondylitis
B. Alkaptonuria
C. Multiple myeloma
D. Peripheral arterial disease
E. Bilateral hip osteoarthritis

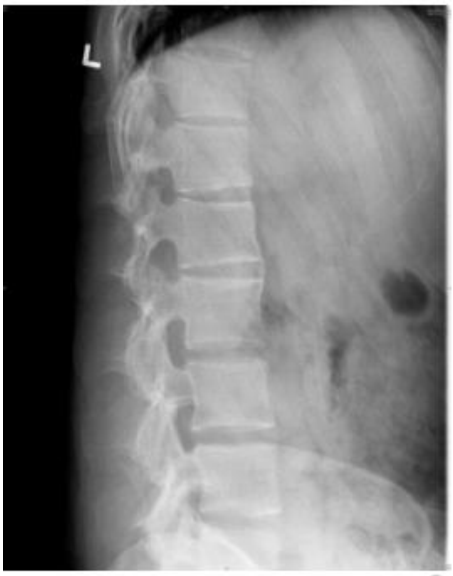
A. Ankylosing spondylitis

Fusion of bilateral sacroiliac joints consistent with ankylosing spondylitis. Sacroiliitis may present as sclerosis of joint margins which can be asymmetrical at early stage of disease, but is bilateral and symmetrical in late disease.

Pain that is worse in the morning is consistent with an inflammatory condition such as ankylosing spondylitis.

Question 33 of 79

A 36-year-old man presents with progressive lower back pain for the past six months. The pain is worse in the mornings and tends to ease with exercise and the passage of the day. He has tried paracetamol but this does not fully controlled his pain. An x-ray of his spine is shown below:



What is the most appropriate first-line treatment

A. Methotrexate
B. Sulfasalazine
C. Naproxen
D. Vitamin D supplementation
E. Infliximab

C. Naproxen

The x-ray shows syndesmophytes and squaring of vertebral bodies consistent with a diagnosis of ankylosing spondylitis.

NSAIDs are the first-line drug treatment in an ankylosing spondylitis. Regular exercise is also very important. The role of anti-TNF therapies is increasing but they are not currently first-line and the 2008 NICE guidelines specifically advise against using infliximab.

Question 34 of 79

A 78-year-old man is investigated for headaches. A routine blood screen is normal other than an elevated ALP. A skull x-ray is ordered:

What is the most likely diagnosis?

A. Myeloma
B. Cervical spondylosis
C. Pituitary tumour
D. Calcified temporal arteritis
E. Paget's disease of the bone

E. Paget's disease of the bone

This is a very stereotypical question - you should always think Paget's disease if shown a skull x-ray in an exam.

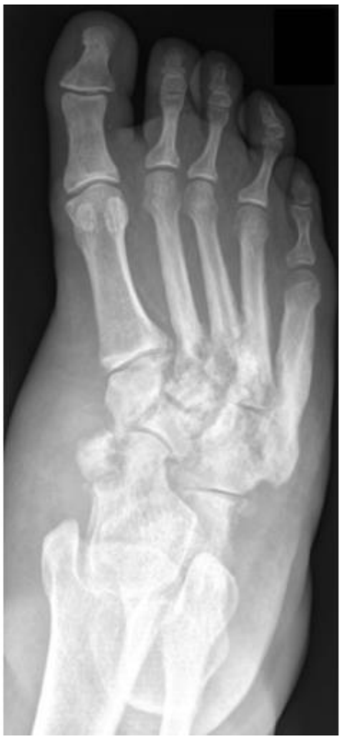
The radiograph demonstrates marked thickening of the calvarium. There are also ill-defined sclerotic and lucent areas throughout. These features are consistent with Paget's disease.

Question 35 of 79

An 85-year-old woman presents with a long history of poorly controlled type 2 diabetes mellitus presents to her GP complaining of a swollen right foot. She describes it as a 'gammy' foot and says she is always tripping over it. The pain is described as being 2 out of 10. The patient also describes reduced sensation up to her ankles.

On examination she has reduced sensation in both feet. The right midfoot is swollen, warm and slightly erythematous but there is no ulcer present. The dorsalis pedis pulse is difficult to feel on the right hand side.

An x-ray is requested:



What is the most likely diagnosis?

A. Septic arthritis of the 1st metatarsophalangeal joint
B. Osteomyelitis
C. Charcot joint
D. Critical ischaemia of the right foot secondary to peripheral arterial disease
E. Gout

C. Charcot joint

The x-ray shows extensive bone remodeling / fragmentation involving the midfoot. In combination with the presence of a swollen, red, warm joint in a patient with a history of poorly controlled diabetes is highly suggestive of a Charcot's joint.

The x-ray findings are not consistent with osteomyelitis and questions would often give other clues such as an overlying ulcer, which is not present in this case.

Question 36 of 79

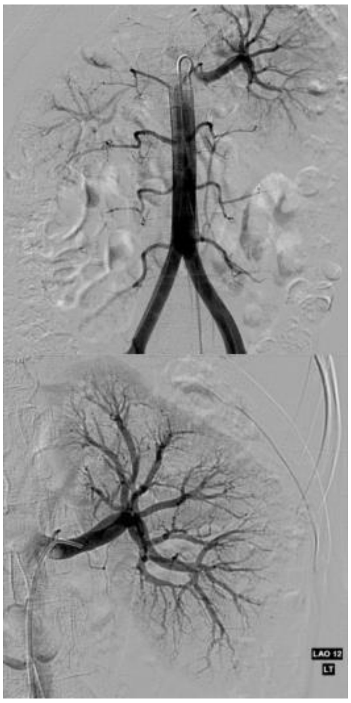
A 23-year-old woman is investigated for abdominal pain and haematuria. She started developing abdominal pain around 2 months ago and it is generally worse after eating. She also reports arthralgia, weight loss of 3kg and general lethargy.

On examination blood pressure is 150/102 mmHg, pulse 84/min and temperature 37.6ºC. Dipstick examination of her urine shows blood+++.

Bloods show the following:

Na	142 mmol/l
K	4.9 mmol/l
Urea	8.9 mmol/l
Creatinine	123 µmol/l
CRP	73 mg/l

An angiogram is performed:



What is the most likely diagnosis?

A. Renal cell cancer
B. Polyarteritis nodosa
C. Henoch-Schonlein purpura
D. Antiphospholipid syndrome
E. Granulomatosis with polyangiitis

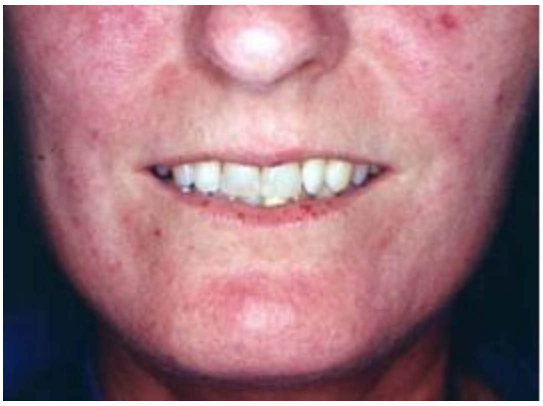
B. Polyarteritis nodosa

The combination of systemic symptoms, fever, hypertension and renal failure are supportive of polyarteritis nodosa. The abdominal pain is due to the arteries supplying the gut being affected.

Both kidneys demonstrate beading and numerous microaneurysms affecting the intrarenal vessels. Similar changes are seen affecting the intrahepatic vessels with a few small microaneurysms noted. The proximal branches of the SMA appears normal; however there are no normal straight arteries from the jejunal arteries and lack of normal anastomotic arcades and loops. This is associated with multiple microaneurysms

Question 37 of 79

This 60-year-old woman who is being treated for heartburn comes for review. She has developed some spots on her lips:



What is the most likely diagnosis?

- A. CREST syndrome
- B. Oesophageal cancer
- C. Vitamin C deficiency
- D. Peutz-Jeghers syndrome
- E. Iron-deficiency anaemia

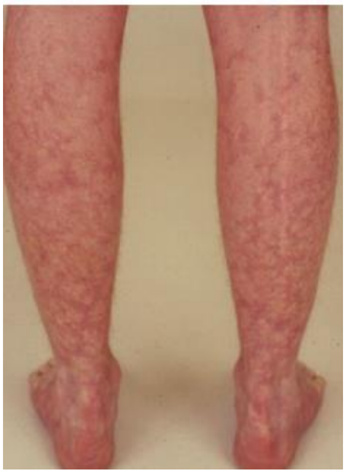
A. CREST syndrome

The heartburn may be explained by oesophageal dysmotility, a feature of CREST syndrome. The lesions on her lips are telangiectasia. She also has the typical tightening of the facial skin seen in patients with systemic sclerosis.

Question 38 of 79

A 52-year-old man presents with feeling generally unwell. For the past few weeks he has felt lethargic, lost weight and had troublesome testicular pain. Over the past week he has also noticed a 'weakness at the ankle' on the right side.

On examination blood pressure is 164/96 mmHg, pulse 86/min, temperature 37.3ºC. Examination of the cardiovascular system is unremarkable. Foot drop is noted on the right side consistent with a common peroneal nerve palsy. A rash is also noted on his legs:



A urine dipstick shows blood++.

What is the most likely diagnosis?

- A. Henoch-Schonlein purpura
- B. Wegener's granulomatosis
- C. Polyarteritis nodosa
- D. Amyloidosis
- E. Behcet's syndrome

C. Polyarteritis nodosa

The correct answer is **Polyarteritis nodosa**. This patient presents with a combination of constitutional symptoms (lethargy and weight loss), testicular pain, hypertension, common peroneal nerve palsy (right-sided foot drop), and livedo reticularis on the legs. These findings are consistent with polyarteritis nodosa (PAN), a systemic necrotizing vasculitis that affects mediumsized arteries. PAN can cause multiorgan involvement, leading to a variety of clinical presentations. In this case, the patient's hypertension may be due to renal artery involvement, while the testicular pain could result from inflammation of the testicular artery. The common peroneal nerve palsy is likely secondary to ischemia caused by vasculitic changes in vasa nervorum.

Henoch-Schonlein purpura is an incorrect answer because this condition typically presents in children and is characterized by palpable purpura, arthritis or arthralgia, abdominal pain, and renal involvement. While there is some overlap with the presented symptoms, Henoch-Schonlein purpura would not explain the patient's testicular pain or common peroneal nerve palsy.

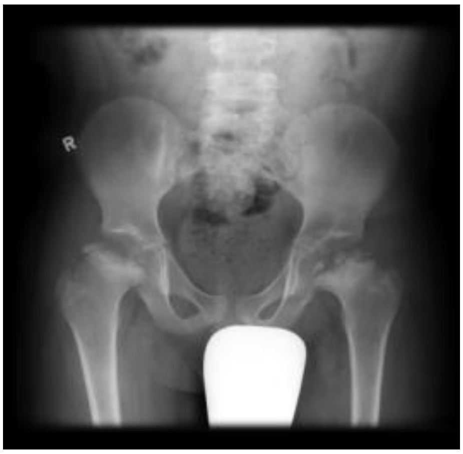
Wegener's granulomatosis, now known as granulomatosis with polyangiitis (GPA), is another form of vasculitis that primarily affects small- to medium-sized vessels. It classically presents with upper and lower respiratory tract symptoms and glomerulonephritis. Although it can potentially cause similar symptoms as PAN, GPA typically involves granulomatous inflammation which isn't mentioned in this scenario.

Amyloidosis is a group of diseases characterized by extracellular deposition of misfolded proteins called amyloid fibrils. While amyloidosis can cause multisystem involvement leading to diverse clinical manifestations such as nephrotic syndrome, cardiomyopathy, and peripheral neuropathy, the presence of livedo reticularis and testicular pain are not typical features of this disease.

Behcet's syndrome is a rare multisystem inflammatory disorder characterized by recurrent oral and genital ulcers, uveitis, and skin lesions. Although it can cause systemic vasculitis affecting both arteries and veins, the patient's presentation with hypertension, common peroneal nerve palsy, and testicular pain is less suggestive of Behcet's syndrome compared to polyarteritis nodosa.

Question 39 of 79

A 8-year-old boy is complains of progressively worsening pain in both groin areas. He has no past medical history of note and his immunisations are up-to-date. There is no recent history of trauma. On examination he walks with a limp. An x-ray is requested:



What is the most likely diagnosis?

- A. Developmental dysplasia of the hip
- B. Slipped upper femoral epiphysis
- C. Osteosarcoma
- D. Acute lymphoblastic leukaemia
- E. Perthes disease

E. Perthes disease

The correct answer is **Perthes disease**. Perthes disease, also known as Legg-Calve-Perthes disease, is a childhood condition that affects the hip joint. It occurs when blood supply to the femoral head is temporarily interrupted, leading to avascular necrosis of the bone. This results in pain and limping, which are common symptoms seen in this patient. The x-ray image provided shows characteristic changes of Perthes disease, with flattening and irregularity of the femoral head.

Developmental dysplasia of the hip is an incorrect option because it typically presents earlier in life and is associated with hip instability or dislocation rather than pain and limping. The x-ray findings in developmental dysplasia of the hip would show shallow acetabulum and potential dislocation or subluxation of the femoral head.

Slipped upper femoral epiphysis (SUFE) is also an incorrect choice. SUFE usually occurs during adolescence and presents with acute or chronic pain in the groin, thigh, or knee. The x-ray findings would demonstrate displacement of the metaphysis from the epiphysis along the growth plate. In this case, there is no evidence of such displacement on x-ray.

Osteosarcoma is a type of bone cancer that can cause pain and swelling around a joint but it's less likely to be the cause for this patient's symptoms given his age and lack of other signs like swelling or systemic symptoms. Additionally, osteosarcoma would present with lytic or sclerotic lesions on x-ray which are not evident in this case.

Finally, **acute lymphoblastic leukaemia** (ALL) can sometimes present with bone pain due to infiltration by leukaemic cells; however, ALL would be unlikely to cause localized bilateral groin pain without other systemic symptoms such as fever, fatigue, or weight loss. The x-ray findings in ALL would typically show diffuse osteopenia and metaphyseal bands, which are not present in this case.

Question 40 of 79

A 52-year-old female presents to rheumatology outpatient clinic with three months history of severe pain on her hands. She has no significant past medical history. On examination, there are swelling and erythema of the first, second and third metacarpophalangeal joints on both hands. She is diagnosed with rheumatoid arthritis.

CRP	34mg/L

What treatment should be started?

- A. Methotrexate + infliximab
- B. Infliximab
- C. Methotrexate + prednisolone + sulfasalazine
- D. Methotrexate + prednisolone
- E. Azathioprine

D. Methotrexate + prednisolone

In 2018 NICE updated their rheumatoid arthritis guidelines. They now recommend diseasemodifying antirheumatic drug (DMARD) monotherapy with a short-course of bridging prednisolone. In the past dual DMARD therapy was advocated as the initial step.

Question 41 of 79

A 52-year-old female has presented to your neurology clinic reporting difficulty in lifting her arms during exercises at the gym over the past two months. She is distressed by her symptoms and is tearful, mentioning that she is also using significant amounts of make-up to cover a new purple rash and swelling around her eyelids. She reports no past medical history no recent trauma and is normally fit and well. She has had a dry cough for the past 5 months that she puts down to her previous social smoking when she would smoke up to 2 cigarettes whilst going out with friends every 2 weeks.

On examination, you note a limited range of passive movement in both shoulders and hips secondary to tender deltoids and hip flexors. Examination of power demonstrates 4- out of 5 symmetrically in hip flexion and shoulder abduction. An elliptical erythematous rash is present around her eyes, the skin around her fingers appear tough bilaterally. Auscultation of her chest reveals bibasal fine inspiratory crackles and normal heart sounds. Observations show she is currently has a low-grade temperature of 37.7 degrees. A chest radiograph demonstrates bilateral fibrotic changes.

Her admission blood tests are as follows:

Hb	121 g/l
Platelets	590 * 10 /l9
WBC	12.3 * 10 /l9
ESR	20 mm/hr
Creatine kinase	3000 u/l
LDH	250 u/l

What is the most likely unifying diagnosis?

- A. Inclusion body myositis
- B. Systemic sclerosis
- C. Dermatomyositis
- D. Polymyalgia rheumatica

E. Fibromyalgia
F. Dermatomyositis

C. Dermatomyositis

The patient has presented with a syndrome of proximal myopathy associated with a violaceous rash on her eyelid and mechanics hands, in addition to possible interstitial lung disease. The most suspicious diagnosis is an inflammatory myositis. Inclusion body myositis is a diagnosis of exclusion and is normally isolated to proximal and less commonly distal, bulbar and facial muscles. Systemic sclerosis is a possible diagnosis and can produce inflammatory myositis similar to polymyositis. However, cranial and peripheral neuropathies are more common. The distinctive cutaneous features in this patient are strongly suggestive of dermatomyositis: the violaceous eyelid rash and oedema is a heliotropic rash associated with dermatomyositis. Other cutaneous findings include Gottron's papules (violaceous papules on the extensor surfaces of fingers), shawl and V sign (photosensitive hyperpigmentation around the shoulders and upper chest), mechanics hands and periungual erythema. Note the normal ESR result, which is often not elevated in dermatomyositis patients.

Question 42 of 79

A 63-year-old female presents to gastroenterology outpatient clinic with a four-week history of gastric reflux, which has not improved despite being prescribed both ranitidine and omeprazole by her GP. She is awaiting an urgent OGD to investigate symptoms further. She reports having lost 7kg in weight over the past 6 months and is also distressed by appearances of white hard lumps appearing on her fingertips. On examination, you note cool peripheries and dry mucous membranes, left thumb calcinosis surrounded by shiny skin up to her wrist joint and wrinkling of skin around her mouth. Her blood tests are as follows demonstrate she is positive for anticentromere antibodies.

What is the most likely diagnosis?

A. Diffuse cutaneous systemic sclerosis
B. Systemic sclerosis sine scleroderma
C. Zollinger-Ellison syndrome
D. Limited cutaneous systemic sclerosis
E. Raynaud's syndrome

D. Limited cutaneous systemic sclerosis

Systemic sclerosis (SS) is a fairly clear answer when presented with a combination of sclerodactyly, calcinosis, perioral puckering and gastro-oesophageal reflux symptoms in a middle-aged female. The main differentials are whether this represents diffuse cutaneous, limited cutaneous or systemic sclerosis sine scleroderma. The latter describes patients with systemic involvement and possible Raynaud's phenomenon in the absence of other cutaneous manifestations with detection of SS autoantibodies. SS can be described generally as diffuse when skin proximal to the distal forearm is involved, such as the elbow, thorax or abdomen. Note that both limited and diffuse cutaneous SS has extracutaneous involvement: however, patients with diffuse cutaneous SS are more likely to develop significant renal, lung and cardiac disease.

Autoantibodies are useful in confirming the subtype of SS and predict extracutaneous involvement but negative results do not rule out SS. Anti-centromere antibodies are associated with limited cutaneous SS1, anti-Scl 70 with diffuse SS and lung involvement , anti-RNA polymerase III to those at high risk of scleroderma renal crisis , anti-U3-RNP to those at high risk of pulmonary hypertension and anti-PM-Scl to those at high risk of SS associated myositis .

Question 43 of 79

A 27-year-old male presents after recently returning from Bangladesh with 2 weeks of daily spiking fever, a new rash on his foot and pain on bending his knees or closing his hands. He also reports lumps and bumps on his neck that he thinks are new. He denies any cough or weight loss. He has no other past medical history and is unaware of any unwell family members. On examination, his temperature is 39.2 degrees. You note a maculopapular rash on his left sole and face. His knees and wrists are swollen and tender. His chest and cardiovascular examination are unremarkable, his abdomen is soft. However, you note a 12cm splenomegaly. His serum tests demonstrate:

Hb	127 g/l
Platelets	450 * 10 /l9
WBC	17.0 * 10 /l9
Neuts	11.0 * 10 /l9

Na^+	138 mmol/l
K^+	3.5 mmol/l
Urea	7.8 mmol/l
Creatinine	70 µmol/l

CRP	30 mg/l
Ferritin	2000 µg/l
ALP	250 u/l
ALT	160 u/l
ANA	negative
dsDNA	negative

His chest radiograph appears unremarkable with no focal consolidation. A first induced sputum is negative for acid-fast bacilli.

What is the most likely diagnosis?

A. Miliary tuberculosis
B. Adult onset Stills disease
C. Reactive arthritis post-travellers diarrhoe
D. Porphyria cutanea tarda
E. Systemic lupus erythematosus

B. Adult onset Stills disease

Adult onset Stills disease (ASD) is a systemic inflammatory condition of unknown aetiology but often thought to be secondary to an infectious trigger on a background of genetic predisposition. It classically presents in young adults under 40 years old, peaking between 15-25 years, with a daily fever and a new non-pruritic rash. The most sensitive diagnostic classification is the Yamaguchi criteria . defined by a patient presenting with all major criteria and at least two minor criteria.

Major criteria:

- Fever greater than 39 degrees over one week
- Arthralgia or arthritis lasting two weeks or longer
- Non-pruritic or maculopapular rash, salmon coloured, found on trunk or extremities, particularly during febrile episodes
- Leucocytosis >10,000/microL

Minor criteria:

- Sore throat, lymphadenopathy, hepatomegaly or splenomegaly, abnormal liver function tests, negative ANA or rheumatoid factor

A number of features in the history and examination should point you towards ASD. The nature of the fever is quotidian: ASD fevers tend to spike daily or twice daily. Secondly, the location of the rash is also characteristic, most commonly a salmon coloured rash in the trunk or the soles, palms and face. Knees, wrist and ankles are the most common sites for arthritis and arthralgias. Ferritin rises are characteristic of ASD, observed in up to 70% of patients . Mild elevations in ALT and ALP are also characteristic of ASD. However, the patient does not describe a sore throat, which is often severe and non-suppurative in up to 70% of ASD patients. Treatment of ASD involves systemic immunosuppression, initially with prednisolone followed by DMARDs.

The main differential to consider is TB. A two-week history is short for miliary TB and particularly unusual for someone without chest signs. It typically is the result of a pulmonary focus eroding the pulmonary vein, leading to systemic dissemination. A negative induced sputum also points against miliary Tb but does not fully exclude it. SLE could also fit with the clinical picture but again, less likely with negative dsDNA and ANA. There is no suggestion that the patient has recently had GI symptoms. Reactive arthritis, formerly known as Reiter's syndrome, does not account for the splenomegaly or serum abnormalities.

Question 44 of 79

An 88-year-old female is admitted to hospital after recurrent mechanical falls. Her past medical history includes an anterior resection for sigmoid carcinoma, type 2 diabetes mellitus and gout.

Three days into her admission, she was treated for hospital-acquired pneumonia with three days of intravenous tazocin. One week into her admission, she developed a swollen inflamed 2nd MTP joint and colchicine was started.

After becoming medically stable 10 days into admission and awaiting a package of care at home, nursing staff report diarrhoea, with type 7 stool up to 7 times a day. She has no laxatives prescribed. One set of stool cultures were sent within 15 minutes of the last episode, which have proved negative for *Clostridium difficile* toxin and, MC+S and norovirus.

What is the most likely cause of her diarrhoea?

A. Clostridium difficile
B. Norovirus
C. Colchicine
D. Recurrence of colon carcinoma
E. Tazocin

C. Colchicine

Colchicine is a classic cause of diarrhoea, particularly in the elderly and in higher doses, leading to the old adage of 'you run before you walk!' Antibiotics can induce diarrhoea without infection, particularly those acting on anaerobes . It is unusual in this timeframe, having diarrhoea 4 days after the antibiotics finished. However, the most serious cause in this scenario is whether tazocin has resulted in pseudomembranous colitis, resulting in *Clostridium difficile* infection. In this case, negative samples for antigens and toxins are likely to be a true negative result. The main concern regards samples that were not transported to the lab promptly (within 2 hours), resulting in the breakdown of *Clostridium difficile* toxin, hence a false negative result. There is little to suggest cancer recurrence or an outbreak of norovirus.

Question 45 of 79

A 28-year-old woman who is 20 weeks pregnant is referred to you by her GP. She has a 2-month history of arthralgia, myalgia, and fatigue. She had initially put this down to pregnancy but was finding it increasingly difficult to do her job as a health care assistant in a local nursing home. She denied any shortness of breath, swallowing difficulties or alopecia.

She had asthma since childhood but was relatively well controlled on inhaled salbutamol as required and beclomethasone 400 micrograms twice daily.

She was a smoker of 10 cigarettes per day and had not drunk any alcohol since learning she was pregnant. She lives with her husband and 2-year-old son. Her mother has a history of rheumatoid arthritis.

Her observations show a blood pressure of 138/86 mmHg and a heart rate of 92 beats per minute. Urinalysis showed a trace of protein.

On examination there was tenderness of the 2nd and 3rd metacarpophalangeal (MCP) joints bilaterally and both wrists but no evidence of active synovitis. There are several painless mouth ulcers. You notice a few bruises on her arms but no other evidence of a rash. Her chest was clear and heart sounds were normal. Neurological examination was normal including full visual fields and eye movements.

Her bloods showed the following:

Haemoglobin	108 g/L
White Cell Count	9.2 x 10 /L
Platelets	103 x 10 /L

Neutrophils	6.02 x 10 /L
Lymphocytes	0.80 x 10 /L
Eosinophils	0.90 x 10 /L
ESR	29 mm/h

Urea	6.9 mmol/L
Creatinine	118 micromol/L
CRP	11 mg/L
Alkaline Phosphatase	87 iu/L
ALT	42 iu/L
Albumin	32 g/L

ANA	1: 320
dsDNA	24
Anti -Ro	Positive
Anti -La	Positive
Rheumatoid Factor	Positive
Anti CCP	Negative
Antiphospholipid antibody	negative

Given the most likely diagnosis, what complication needs to be discussed with her?

A. Post partum haemorrhage
B. Congenital heart block
C. Deep vein thrombosis
D. Pre-eclampsia
E. Scleritis

B. Congenital heart block

This question tests your knowledge of the diagnosis of Systemic Lupus Erythematosus (SLE). Diagnosis is based on the American College of Rheumatology (ACR) criteria written in 1982 and revised in 1997:

Four or more of the 11 criteria need to be fulfilled to be able to diagnose SLE. Note that while fatigue is a common feature it is not used in the diagnostic criteria. In this case, the criteria are oral ulcers + arthritis + positive dsDNA + the presence of ANA (while there is lymphopenia this is a single test result only)

In this case, there are sicca symptoms and Anti-Ro and -La antibodies suggesting an overlap with Sjogren's syndrome. Rheumatoid factor is positive in approximately 40% of SLE patients. The absence of anti-CCP should point you away from rheumatoid arthritis.

The point to make with this question is to test the candidate's knowledge of diagnosis of connective tissue diseases and their associated complications. Anti-Ro antibodies can cross the placenta and can lead to neonatal lupus and congenital heart block of the newborn, which can require pacing at birth. Miscarriage is another common complication of SLE. These can occur beyond the first trimester.

While postpartum haemorrhage, pre-eclampsia and deep vein thrombosis are complications of pregnancy that will need to be discussed, the presence of SLE does not increase the risk of either in this scenario. If the antiphospholipid antibody or lupus anticoagulant were positive then there is an increased risk of arterial or venous thrombosis, in which case you might consider anticoagulation but obviously not with warfarin in pregnancy.

Question 46 of 79

You are the medical doctor on an acute medical admissions unit. A 56-year old female with hypertension, pulmonary fibrosis and a recent diagnosis of Raynaud's phenomenon presents with generally feeling unwell. On further questioning she also reports dysphagia for the past few months for which she is awaiting investigations under the gastroenterology team at your hospital. She is currently only on amlodipine 5mg od.

Her observations are: temperature 36.4°C, pulse 88/min, blood pressure 172/88 mmHg, respiratory rate 14/min, sats 100% on room air. Her chest is clear and abdomen soft, non-tender. Blood tests reveal an acute kidney injury with: sodium 141 mmol/l, potassium 4.6 mmol/l, urea 27 mmol/l, creatinine 320 µmol/l (her GP notes state she had a normal renal function from a routine blood test 1 month ago).

What is the most appropriate treatment at this stage?

A. Fluids
B. Stat 5mg amlodipine
C. Stat angiotensin-converting enzyme inhibitor (ACE-i)
D. Haemodialysis
E. Haemofiltration

C. Stat angiotensin-converting enzyme inhibitor (ACE-i)

This lady has features of diffuse cutaneous systemic sclerosis - she has Raynaud's phenomenon, pulmonary fibrosis and dysphagia. Her current presentation is that of scleroderma renal crisis. This is a medical emergency and treatment should be administered as soon as possible. The most appropriate treatment initially would be an ACE-i with a consideration for dialysis and renal transplantation if required.

Question 47 of 79

A 68-year-old lady attends for review in the oncology clinic. She has advanced oestrogen receptor positive, HER2 negative breast cancer, with metastasis to her ribs, thoracic vertebrae and right humerus. She previously underwent right mastectomy and first line chemotherapy but has declined further chemotherapy.

She has had back and rib pain which was was improved by external beam radiotherapy. She was started on alendronate to help prevent pathological fractures but has since suffered nausea, severe acid reflux and epigastric discomfort not helped by a proton pump inhibitor. Her alendronate was stopped and risedronate was trialled but resulted insimilar effects and so was also discontinued.

What is the most appropriate medication to prescribe this lady to help prevent skeletal related events?

A. Denosumab
B. Lapatinib
C. Letrozole
D. Strontium
E. Trastuzumab

A. Denosumab

This lady has bone metastases so is at risk of pathological fracture. The first choice agent would be a bisphosphonate such as alendronate. However, this lady is unable to take bisphosphonates due to oesophageal irritation and nausea are common side effects.

Denosumab is a monoclonal antibody which binds to and inhibits RANKL on the surface of osteoclasts, preventing the break down of bone. It is approved by NICE for women with osteoporosis or with advanced breast cancer and bone metastases, who have been intolerant to bisphosphonates.

Strontium ranelate is recommended for primary prevention of osteoporotic fractures in at risk women who have been intolerant of bisohosphonates. However, it has not been shown to reduce skeletal related events in bone metastases.

Lapatinib is a tyrosine kinase inhibitor of the HER2 and epidermal growth factor receptors and is part therapy to treat HER2 positive breast cancer.

Letrozole is an aromatise inhibitor used to treat oestrogen receptor positive breast cancer. Although it is generally effective against these types of cancer, it has not been shown in trials to reduce skeletal related events in patients with this cancer type.

Trastuzumab is a monoclonal antibody against the HER2 receptor and is part of therapy to treat HER2 positive breast cancer.

References:

National Institute for Health and Care Excellence. Denosumab for the prevention of skeletalrelated events in adults with bone metastases from solid tumours. NICE technology appraisal guidance [TA265] (2012)

National Institute for Health and Care Excellence. Alendronate, etidronate, risedronate, raloxifene and strontium ranelate for the primary prevention of osteoporotic fragility fractures in postmenopausal women. NICE technology appraisal guidance 160 (2011)

Question 48 of 79

A 38-year-old male plumber is referred to the medical assessment unit by his GP due to reduced oxygen saturations. He has had mild asthma since childhood but no other medical history of note. His medications are a salbutamol inhaler when required and co-codamol for long standing back pain. On examination he is found to have an early diastolic murmur but no other abnormalities are detected. He goes on to have a chest x-ray which demonstrates apical interstitial shadowing. He undergoes pulmonary function tests which are as follows:

FEV1	1.9L	(Predicted 2.1-3.1)
FVC	2.2	(Predicted 3.0-4.4)
TLC	4.5	(Predicted 5.0-7.5)
Transfer factor (DLCO)	Low	

What is the most likely diagnosis?

A. Ankylosing spondylitis
B. Asbestosis
C. Extrinsic allergic alveolitis
D. Churg-Strauss syndrome
E. Sarcoidosis

A. Ankylosing spondylitis

There are a couple of red-herrings in this question designed to lead candidates astray. The fact that he is a plumber could be linked to a diagnosis of asbestosis, whilst his prior history of asthma may be suggestive of Churg-Strauss syndrome. However the fact that this patient's CXR shows apical interstitial shadowing, combined with the restrictive pattern of his pulmonary function tests indicate that he is suffering with apical lung fibrosis. This narrows the potential answers down to ankylosing spondylitis, sarcoidosis or extrinsic allergic alveolitis. The history of back pain and the finding of an early diastolic murmur (suggestive of aortic regurgitation) confirm ankylosing spondylitis as the most likely diagnosis.

Question 49 of 79

A 68-year-old gentleman presents acutely with a hot, red, swollen and painful right big toe. This has happened twice before, some years ago, and he has never sought medical attention. He has a raised serum urate level and joint fluid analysis demonstrates negatively birefringent needleshaped crystals. Once symptoms settle he is started on allopurinol but develops a severe hypersensitivity reaction to this.

Which agent should be tried next as a long-term treatment option?

A. Colchicine
B. Naproxen
C. Febuxostat
D. Methotrexate
E. Prednisolone

C. Febuxostat

Febuxostat is recommended as an option for the management of chronic hyperuricaemia in gout only for patients who are intolerant of allopurinol or for whom allopurinol is contra-indicated. For the purposes of this guidance, intolerance of allopurinol is defined as adverse effects that are sufficiently severe to warrant discontinuation, or to prevent full dose escalation for optimal effectiveness.

Watch out for severe hypersensitivity reactions (including Stevens-Johnson) with febuxostat, which would be a reason to discontinue this agent.

Question 50 of 79

A 45-year-old woman was referred to Rheumatology clinic after experiencing widespread aches and pains felt throughout her body. The pains were felt particularly in her arms and legs in addition to significant pain throughout the patient's spinal column. The patient could not recall a precise onset of her symptoms but she felt they had been present for at least 12 months, possibly longer. In addition, the patient reported on-going feelings of tiredness and lethargy. Despite going to bed around 10 pm each evening, the patient reported waking in the morning still feeling exhausted. She denied any history of hot or tender joints, skin rashes, hair loss, swallowing difficulties or dry eyes. The patient's appetite was described as normal for her with no significant change in weight.

There was no previous past medical history and the patient took no regular medications except for a non-prescription multi-vitamin. Family history was remarkable for hypothyroidism affecting her mother and elder sister. The patient worked as an accountant and lived with her two teenage children. She had separated from her ex-husband 18 months previously.

The examination did not demonstrate any evidence of active synovitis of the hands or feet with no other inflamed or deformed joints. Palpation of the muscles of the upper arms and legs as well as the paraspinal muscles was exquisitely tender. Neurological examination of the arms and legs was unremarkable. The cardiovascular and respiratory examination was unremarkable with no skin rashes.

During clinic interaction, the patient appeared tired and stressed but had a good rapport and maintained good eye contact. She denied any significant low mood but was anxious that her symptoms represented a serious underlying illness.

Investigations requested following clinic are listed below.

Haemoglobin	129 g / L
White cell count	7.2 * 10 /l
Platelets	332 * 10 /l
Mean cell volume	87 fL
Sodium	140 mmol / L
Potassium	3.6 mmol / L
Urea	3.5 mmol / L
Creatinine	68 micromol / L
Erythrocyte sedimentation rate	11 mm / h
Rheumatoid factor	Negative
Anti-nuclear antigen	Weak positive
B12	324 pmol / L (reference 74-516)
Folate -	30 nmol / L (reference 7-36)
Serum immunoglobulin	Normal electrophoresis strip
Thyroid stimulating hormone	0.9 microU / mL (reference 0.4-5.0)

X-rays of hands: some minor degenerative change in right index proximal interphalangeal joint but otherwise unremarkable with no boney erosion or deformity

What is the cause of the patient's pain?

A. Fibromyalgia
B. Systemic lupus erythematous
C. Chronic regional pain syndrome
D. Generalised anxiety disorder
E. Depression

A. Fibromyalgia

The patient has chronic widespread pain (>3 months) associated with lethargy, non-refreshing sleep and multiple tender points on palpation. Basic blood tests are essentially normal and there is no history or examination to suggest connective tissue disease or other pathology. This presentation is consistent with fibromyalgia, the diagnostic label used to describe chronic widespread pain associated with multiple muscular tender points or associated symptoms of fatigue, non-refreshing sleep or cognitive dysfunction.

Please note that many healthy individuals have weakly positive anti-nuclear antigen results and this does not imply a diagnosis of systemic lupus erythematous in the absence of symptoms and signs of the disease. It may be that requesting immunological tests was inappropriate in this patient given the lack of clinical evidence of connective tissue disease.

Chronic regional pain syndrome is associated with persistent burning pain in one limb, usually after a minor injury. The brief mental state examination documented does not suggest evidence of significant depression or generalised anxiety.

Question 51 of 79

A 72-year-old woman attends the clinic with a 6-month history of weakness and muscle pain. On examination, you note a proximal muscle weakness of the upper (MRC grade 4/5) and lower (MRC grade 3/5) limbs. A systematic enquiry and examination were otherwise unremarkable.

Blood results are as follows:

Hb	110 g/L	Male: (135-180)Female: (115 - 160)
Platelets	310 * 10 /L $_9$	(150 - 400)
WBC	8.2 * 10 /L $_9$	(4.0 - 11.0)
Na $_+$	135 mmol/L	(135 - 145)
K $_+$	4.2 mmol/L	(3.5 - 5.0)
Urea	6.1 mmol/L	(2.0 - 7.0)
Creatinine	88 µmol/L	(55 - 120)
CRP	2 mg/L	(< 5)

What antibody is most sensitive (rather than specific) to the underlying condition?

A. Anti dsDNA antibody
B. Anti-Jo-1 antibody
C. Anti-PL-7 antibody
D. Anti-Smith antibody
E. Antinuclear antibody

E. Antinuclear antibody

Proximal muscle weakness + raised CK + no rash → ?polymyositis

The patient most likely has a diagnosis of polymyositis as indicated by proximal muscle weakness and myalgia.

Antinuclear antibody (ANA) is correct. Although ANA is a relatively sensitive test, with the majority of patients (around 80%) being positive, it is not specific to polymyositis.

Anti-Jo-1 antibody is incorrect. Although this antibody is highly specific to polymyositis, it is not very sensitive.

Anti dsDNA is incorrect. The anti-double stranded DNA antibodies (anti-dsDNA) are considered a specific marker for systemic lupus erythematosus (SLE). Although muscle weakness can occur in SLE, the absence of other diagnostic criteria of SLE (e.g. oral ulcers, fever, and neurological dysfunction) makes it a less likely diagnosis.

Anti-PL-7 antibody is incorrect. Although this can be positive in dermatomyositis, it is not sensitive nor specific to this condition.

Anti-Smith is incorrect. Anti-Smith antibody is another highly specific antibody for SLE.

Question 52 of 79

A 72-year-old woman attends rheumatology clinic for review of her osteoporosis treatment. She had been diagnosed with osteoporosis five years previously on the basis of a DEXA scan (see results below). At that time, the DEXA scan had been arranged by her GP due to a strong family history of osteoporosis (maternal hip fracture) and the patient having received multiple courses of corticosteroids as treatment for asthma. The patient has never sustained a fracture of her hip, wrist or vertebrae. Following the initial diagnosis, the patient had been treated with alendronic acid 70 mg weekly. She had not experienced any adverse effects from this medication although reported finding the need to drink copious water with her dose onerous.

The patient's past medical history was significant for asthma, although the patient reported that this was now much better controlled than previously and she had not required any corticosteroid treatment in several years. She denied any history of thyroid disease or rheumatoid arthritis. The patient had never smoked and very rarely consumed any alcohol.

Details from the patient examination in clinic and selected results from her DEXA scans are given below.

Height	160 cm
Weight	65 kg
Femoral neck BMD (5 years previously)	T -2.6 g / cm2
Femoral neck BMD (present day)	T -1.9 g / cm2
FRAX 10-year probability of major osteoporotic fracture	18 %
FRAX 10-year probability of hip fracture	6.8 %

What is the most appropriate management of the patient's osteoporosis?

A. Discontinue alendronic acid and initiate treatment with denosumab

B. Hold further osteoporosis treatment with repeat DEXA scan in two years

C. Continue treatment with alendronic acid with repeat DEXA scan in five years

D. Continue treatment with alendronic acid with repeat DEXA scan in two years

E. Hold further osteoporosis treatment with repeat DEXA scan in five years

B. Hold further osteoporosis treatment with repeat DEXA scan in two years

At the present time it is uncertain how long patients should take bisphosphonates. Recent concerns about side effects of long-term bisphosphonate treatment (for example, atypical femur fractures) have led to recommendations about the use of treatment breaks ('drug holidays') in certain patients.

The patient in this example meets the recommended criteria for a treatment break. In particular, her age (< 75 years), femoral neck bone mineral density > -2.5 and lack of history of fragility fracture are all favourable. A repeat DEXA scan is recommended after two years or in the event of fragility fracture to review need for further treatment.

It is recommended to use the WHO Fracture Risk Assessment Tool (FRAX) to estimate a patient's 10-year risk of fragility fracture. This can be combined National Osteoporosis Guideline Group (NOGG) guidance to inform treatment decisions. In this example, the patient's FRAX score would place her below the treatment threshold of NOGG guidance. It should be noted, that despite the recommendation to use FRAX score and NOGG guidance in this situation, neither tools are validated for patients taking bisphosphonates.

Parenteral treatments for osteoporosis such as denosumab should be reserved for individuals in whom bisphosphonate treatment has failure, or who are intolerant of bisphosphonates.

Question 53 of 79

A 58-year-old man who has no fixed abode comes to the Emergency department because he is unable to walk. He has a history of alcoholism and type 2 diabetes. His main complaint is that his shoes have worn out and because of loss of sensation he didn't notice that he had stepped on a nail. In total the lesion on his right foot has been present for approximately 3 weeks.

Which of the following is the next step in evaluating his foot injury?

A. Inflammatory markers
B. MRI foot
C. Plain x-ray foot
D. USS foot
E. Wound swab

C. Plain x-ray foot

The key next step here is to gather useful information about the extent of any foot infection. By 3 weeks post injury, changes consistent with osteomyelitis should be visible on plain x-ray. These may include soft tissue swelling, bone demineralisation, cortical irregularity, and an elevated periosteum.

Many patients progress from a plain x-ray on to MRI imaging of the foot for further evaluation of the extent of infection and to guide potential operative approaches for debridement. Inflammatory markers are a very non-specific marker of infection and ultrasound of the foot is only useful to visualise soft tissue swelling or collection of pus / fluid. A wound swab is likely to show a range of bacteria, this is what drives selection of a broader spectrum antibiotic such as coamoxiclav in the diabetic population.

Question 54 of 79

A 32-year-old woman with a history of systemic lupus erythematosus and stable joint disease currently managed with hydroxychloroquine comes to the nephrology clinic. She complains of worsening peripheral oedema. Examination reveals a blood pressure of 159/88 mmHg, her pulse is 78 beats per minute and regular. There is pitting oedema of both lower limbs.

investigations

Na^+	141 mmol/l
K^+	5.1 mmol/l
Urea	8.1 mmol/l
Creatinine	159 µmol/l
Creatinine one month earlier	132 µmol/l
Urinary protein	5 g/24hrs

Which of the following is the most appropriate intervention with respect to long-term renal outcomes?

A. Cyclophosphamide
B. Methotrexate
C. Methylprednisolone
D. Mycophenolate
E. Rituximab

D. Mycophenolate

Mycophenolate mofetil (MMF) is a prodrug of mycophenolic acid (MPA), an inhibitor of inosine-5'- monophosphate dehydrogenase. MPA depletes guanosine nucleotides preferentially in T and B lymphocytes and inhibits their proliferation. In proteinuric renal disease related to systemic lupus erythematosus, it is the intervention of choice.

Cyclophosphamide is not recommended for lupus nephritis because renal outcomes are similar to those for mycophenolate, with higher rates of infection. Although corticosteroids are used in conjunction with second-line agents, it is mycophenolate which is more important with respect to long-term renal outcomes. Anti-B cell agents such as rituximab are used in the treatment of lupus but are normally added to therapy with a conventional second line agent such as mycophenolate. Methotrexate is used primarily in the treatment of rheumatoid arthritis.

Question 55 of 79

A 62-year-old man with a diagnosis of Paget's disease is seen in clinic with a two month history of worsening bone pain, mainly in his left leg. His medications include paracetamol, ibuprofen, and alendronate.

Examination reveals marked deformity of the long bones, particularly the left tibia. Blood tests:

Calcium	2.40 mmol/L (2.25-2.5)
Albumin	37g/L (34-54)
Corrected calcium	2.50 mmol/L (2.25-2.5)
Alkaline phosphatase	484 U/L (45-105)
Alanine transaminase	27 U/L (5-35)

What is the next stage in the treatment of this patient?

A. Cholecalciferol
B. Surgery
C. Calcitonin
D. Hearing aid
E. Prednisolone

C. Calcitonin

Paget's disease is characterised by abnormal bone remodelling, particularly in the skull and long bones. The characteristic blood test results include an elevated alkaline phosphatase, with otherwise normal liver function tests (as alkaline phosphatase is also found in bone). A raised calcium may only be seen if there is associated immobility.

Analgesics and non-steroidal inflammatory drugs are initially used to manage pain, with treatment escalated to bisphosphonates and calcitonin in refractory cases.

Question 56 of 79

A 35-year-old lady with diffuse systemic sclerosis attends the rheumatology clinic. She has had worsening arthralgia over the last 2 months, mainly in the hands and feet. She does not complain of any other symptoms.

On examination her blood pressure is 161/94 mmHg, her heart rate is 90 beats per minute and her oxygen saturations are 96% on room air. She has sclerodactyly and tender small joints of the hands and feet with mild swelling. The hands are pale and cool. Her chest is clear.

Her blood tests are as follows:

Hb	110 g/l	Na^+	136 mmol/l	Bilirubin	5 µmol/l
Platelets	$210 * 10^9$/l	K^+	4.7 mmol/l	ALP	90 u/l
WBC	$10 * 10^9$/l	Urea	5 mmol/l	ALT	21 u/l
Neuts	$7 * 10^9$/l	Creatinine	89 µmol/l	γGT	30 u/l
Lymphs	$2.5 * 10^9$/l	ESR	99 mm/h	Albumin	32 g/l

Which drug should be used with caution in this patient?

A. Tacrolimus
B. Azathioprine
C. Methotrexate
D. Mycophenolate mofetil
E. Prednisolone

E. Prednisolone

Steroid use is known to precipitate scleroderma renal crisis and this is a patient who already has hypertension. Azathioprine, mycophenolate mofetil, tacrolimus and methotrexate are all immunosuppressive agents which may be used in rheumatological conditions, though methotrexate may cause additional pulmonary fibrosis.

Question 57 of 79

A 65-year-old man was referred to rheumatology for advice regarding the management of his gout. The patient had suffered intermittent episodes of inflammation of the first metatarsophalangeal joint of both feet over the past ten years. The frequency of these episodes had been increasing, with 6 episodes in the past year. In addition, the patient's right knee had recently become inflamed with microscopy of synovial aspirate demonstrating needle shaped crystals with negative birefringence.

Colchicine and NSAIDs had been used effectively to provide symptomatic relief to the patient during an acute attack. Allopurinol had been previously trialled as prophylaxis at a dose of 200 mg daily, although was stopped after the patient's renal function was noted to have deteriorated after allopurinol was initiated. Lifestyle modifications have also been attempted.

Other medical problems included type 2 diabetes, hypertension, hypercholesterolaemia and chronic renal failure. Regular medications were ramipril, metformin and simvastatin.

On examination the patient was noted to be obese without evidence of current joint inflammation. Tophi were noted on examination of the patient's ears. Blood tests taken prior to clinic attendance are listed below.

Hb	16.5 g/dl
Platelets	150 * 10 /l9
WBC	8.6 * 10 /l9

Na^+	137 mmol/l
K^+	4.7 mmol/l
Urea	11.2 mmol/l
Creatinine	190 µmol/l
eGFR	45 ml/min
Calcium (adjusted)	2.3 mmol/l
Urate	395 µmol/l

What is the best strategy for gout prophylaxis in this patient?

A. Prednisolone 10 mg daily
B. Colchicine
C. Febuxostat
D. Naproxen
E. Reduced dose allopurinol

C. Febuxostat

Urate lowering therapy is indicated in recurrent attacks of acute gout, although there are no firm guidelines as to when to initiate therapy. Target serum uric acid levels are usually taken as less than 360 micromol / L. Febuxostat is a non-purine xanthine oxidase inhibitor approved by NICE for use in individuals, such as this patient, who are intolerant of allopurinol or in whom allopurinol is contra-indicated.

Prednisolone, colchicine and naproxen are all used in the acute treatment of gout. Extended use of colchicine and NSAIDs can be considered to reduce the risk of gout relapse during the instigation of allopurinol therapy but have no role as prophylactic therapy in isolation.

Allopurinol can cause renal, hepatic and severe skin reactions (allopurinol hypersensitivity syndrome) and is best avoided in this patient given previous worsening of renal function with a relatively low dose of allopurinol.

Question 58 of 79

A 45-year-old female presents with severe lower back pain. She states that the pain started a couple of months ago and has progressively got worse. She has also been experiencing fevers, rigors and weight loss. On examination she has severe tenderness on palpation over the L4 vertebrae. There is no associated neurological signs.

Blood results are as follows:

Hb	105 g/l	Na^+	140 mmol/l
Platelets	542 * 10 /l 9	K^+	3.8 mmol/l
WBC	20.2 * 10 /l 9	Urea	8.5 mmol/l
Neuts	15.4 * 10 /l 9	Creatinine	92 µmol/l
Lymphs	2.2 * 10 /l 9	CRP	288 mg/l

What investigation is most likely to confirm the diagnosis?

A. Plane film lumbar x-ray
B. MRI
C. CT with bone window
D. White cell scan
E. Blood culture

B. MRI

An MRI scan of the spine is the most appropriate investigation to diagnose infected discitis as it has the greatest sensitivity

The patient has systemic signs indicating infection, together with point tenderness over a vertebrae, making discitis the most likely diagnosis. Discitis, or vertebral osteomyelitis, is defined as infection of vertebral bones with extension into intervertebral discs. The clinical presentation of discitis can be variable from acute back pain and fevers to slowly progressive back pain over a number of months.

More than 50% of discitis cases are due to Staphylococcus aureus. Infection may also be due to a wide range of other organisms including gram negatives, Streptococci and Tuberculosis. Mixed infection is also possible. Three sets of blood cultures should be taken before anti-microbials are given. However, blood cultures are only positive in approximately 50-70% of patients with discitis.

MRI spine is the most sensitive diagnostic test. A whole spine MRI should be performed as discitis frequently occurs at multiple levels. MRI is the imaging modality of choice due to its very high sensitivity and specificity. It is also useful in differentiating between pyogenic, tuberculous, and fungal infections, and a neoplastic process.

A bone scan and white cell (WBC) scan may be used to demonstrate increased uptake at the site of infection, and are more sensitive than plain film and CT, but lack specificity.

CT guided biopsy should be performed in all patients prior to initiation of antibiotics unless there is a positive blood culture which fits the clinical picture or the patient is septic/unstable.

Question 59 of 79

A 76-year-old man presents to his GP with a 3-month history of generalised weakness and fatigue. He is otherwise well with a past medical history of osteoarthritis, gout and type 2 diabetes mellitus (T2DM). His medications include paracetamol and metformin. He is an ex-smoker of 20 cigarettes per day.

On examination, he has relative weakness of shoulder abduction and adduction and needs to use his arms to get up out of the chair, though distal power is maintained. His eye movements are normal and reflexes are preserved throughout, sensation is also normal. There is no obvious rash or joint swelling.

Blood tests show:

Hb	121 g/L	Male: (135-180)Female: (115 - 160)
Platelets	231 * 10 /L 9	(150 - 400)
WBC	9.4 * 10 /L 9	(4.0 - 11.0)

Na $^+$	143 mmol/L	(135 - 145)
K $^+$	4.8 mmol/L	(3.5 - 5.0)
Bicarbonate	28 mmol/L	(22 - 29)
Urea	6.5 mmol/L	(2.0 - 7.0)
Creatinine	136 µmol/L	(55 - 120)

Thyroid stimulating hormone (TSH)	2.3 mU/L	(0.5-5.5)
Free thyroxine (T4)	11.7 pmol/L	(9.0 - 18)
Creatine kinase	632 U/L	(35 - 250)

CRP	43 mg/L	(< 5)

What is the likely underlying diagnosis?

A. Dermatomyositis
B. Lambert Eaton myasthenic syndrome
C. Myasthenia gravis
D. Polymyalgia rheumatica
E. Polymyositis

E. Polymyositis

Proximal muscle weakness + raised CK + no rash → ?polymyositis

This patient has proximal muscle weakness without pain and raised creatinine kinase (CK), which is in keeping with **polymyositis**.

In **Lambert-Eaton Myasthenic Syndrome** you may see proximal muscle weakness associated with ocular muscle weakness and ptosis. It typically improves with repeated muscle use and is associated with small-cell lung cancer. It is not however associated with a raised CK.

Patients with **dermatomyositis** may present similarly but would also have skin changes such as a photosensitive facial rash and/or Gottron's papules which distinguishes the diagnosis from polymyositis.

In **Myasthenia Gravis** you would expect fatigable weakness, typically of the ocular muscles leading to double vision and ptosis. Proximal limb muscles, respiratory muscles and bulbar function may also be affected. However, you would not expect a raised CK.

In **polymyalgia rheumatica** patients may have proximal muscle pain, typically hip and shoulder pain, with fatigue and raised inflammatory markers. You would not expect to have a raised CK.

Question 60 of 79

A 70-year-old man with rheumatoid arthritis presents for his monthly monitoring bloods because he is on methotrexate. He takes methotrexate 20mg once a week with folic Acid 5mg once weekly on a different day. He currently feels well in himself and his arthritis is well controlled. He does not drink alcohol or smoke. His monitoring blood tests come back as follows:

Hb	140 g/l	Na $^+$	139 mmol/l	Bilirubin	14 µmol/l
Platelets	240 * 10 /l 9	K $^+$	4.2 mmol/l	ALP	100 u/l
WBC	7 * 10 /l 9	Urea	5 mmol/l	ALT	80 u/l
Neuts	4.5 * 10 /l 9	Creatinine	87 µmol/l		

What is the correct course of action?

A. Stop methotrexate
B. Reduce methotrexate dose to 10mg once weekly
C. Switch methotrexate to sulfasalazine
D. Continue on current dose with repeat bloods in one month
E. Stop methotrexate and urgent liver USS

D.Continue on current dose with repeat bloods in one month

There is no need to stop methotrexate unless the alanine transaminase (ALT) or aspartate transaminase (AST) doubles according to the BSR guidelines. The patient should continue to have monthly blood test monitoring in the mean time.

Question 61 of 79

A 54-year-old patient is referred to rheumatology with reports of 12 months of dry eyes and dry mouth that she finds very troublesome. There are no other symptoms of note. She has a past medical history of coeliac disease and is compliant with a gluten-free diet.

On examination, she has dry oral mucosa. Chest auscultation is normal. There are is no nail fold vasculitis. There is no cervical, axillary or inguinal lymphadenopathy.

Blood tests:

Hb	136 g/L	Male: (135-180)Female: (115 - 160)
Platelets	189 * 10 /L 9	(150 - 400)
WBC	5.2 * 10 /L 9	(4.0 - 11.0)
Na $^+$	137 mmol/L	(135 - 145)

K +	4.2 mmol/L	(3.5 - 5.0)
Urea	4.2 mmol/L	(2.0 - 7.0)
Creatinine	88 µmol/L	(55 - 120)
CRP	4 mg/L	(< 5)
ESR	88 mm/Hr	(0-20)
Antinuclear antibody	positive (1:1280)	

What is the most definitive way of confirming the likely diagnosis?

A. Extractable nuclear antigen
B. Measurement of salivary flow
C. Salivary gland biopsy
D. Schirmer's test
E. Slit lamp examination

C. Salivary gland biopsy

Salivary gland biopsy is the most definitive way of confirming the diagnosis of primary Sjogren's syndrome - sections will show a typical lymphocytic infiltrate

Salivary gland biopsy is correct. This patient presents with sicca symptoms (dry eyes and dry mouth), raised ESR and a strongly positive antinuclear antibody. There are no symptoms of another connective tissue disease. This suggests a diagnosis of primary Sjogren's syndrome. The most definitive way of confirming the diagnosis is to do a salivary gland biopsy. This will show focal lymphocytic sialadenitis.

Extractable nuclear antigen is incorrect. Typically, if there is a positive antinuclear antibody result, then the next step is to try to subclassify the serology further by testing the ENA. Anti-Ro and antiLa antibodies are associated with Sjogren's syndrome and may support the diagnosis. However, similar to the other tests above, they are not as specific for the diagnosis as a lip biopsy and therefore not as definitive.

Measurement of salivary flow is incorrect. A whole saliva unstimulated flow rate of <= 0.1mL/minute suggests salivary gland hypofunction and can be used to aid a diagnosis of Sjogren's syndrome. However, it is not as specific for the diagnosis as a positive salivary gland biopsy.

Schirmer's test is incorrect. This can be used to aid the diagnosis of Sjogren's syndrome. It involves placing a strip of filter paper inside the lower eyelid. The eyes are shut for 5 minutes. The paper is removed and the amount of moisture is calculated. Less than 5mm in 5 minutes is considered an abnormal result. It is not as specific for the diagnosis of primary Sjogren's syndrome as proving focal lymphocytic sialadenitis pathologically.

Slit lamp examination is incorrect. An ophthalmology review may elicit severe dryness and ocular inflammation. This may suggest Sjogren's syndrome. However, there will be other differentials that can cause these features and a biopsy is more specific for the diagnosis.

Question 62 of 79

A 47-year-old woman was admitted with a 24-hour history of acute breathlessness. She had a three-month history of progressive weakness, 8 kg weight loss, and progressive dysphagia. She was a non-smoker. On examination, her temperature was 38.4°C, pulse 96 beats per minute, and blood pressure 136/65 mmHg. Auscultation revealed coarse inspiratory crackles at the right base and mid zone. She had grade 4/5 weakness of the proximal muscles of the upper and lower limbs. There were no other abnormalities.

Investigations:

Hb	130g/l (115-165)
Platelets	238 * 10 /l (150-400)9
WBC	16 * 10 /l (4.0-11.0)9
CRP	130mg/l (<10)
Na +	140mmol/l (135-145)
K +	3.7mmol/l (3.5-4.5)
Urea	7.5mmol/l (5.0-9.0)
Creatinine	98µmol/l (60-11)
Creatine kinase	5230U/l (24-170)

Which of the following is most likely to be helpful in making a specific diagnosis?

A. Anti-double-stranded DNA antibodies
B. Anti-synthetase antibodies
C. Anti-nuclear antibodies
D. Anti-smooth muscle antibodies
E. Rheumatoid factor

B. Anti-synthetase antibodies

Anti-synthetase antibodies are specifically associated with polymyositis

The diagnosis is in this scenario is aspiration pneumonia secondary to dysphagia caused by polymyositis. It is an idiopathic inflammatory myopathy characterized by symmetrical, proximal muscle weakness and elevated skeletal muscle enzyme levels (creatine kinase). A number of autoantibodies are seen with polymyositis; however, many of these are not disease-specific. Rheumatoid factor is seen in at least 50% of cases of polymyositis, whilst anti-nuclear antibodies (ANA) are seen in approximately 30% of cases. Anti-double-stranded DNA antibodies are more specific for systemic lupus erythematosus (SLE). Anti-smooth muscle antibodies are seen in autoimmune hepatitis. Antibodies in myositis can be divided into those which are specific for myositis, and those which are associated with myositis.

Myositis-specific antibodies are targeted against three types of proteins:

- Transfer ribonucleic acid (tRNA) synthetases: whilst a number of antibodies have been described, the main one is the Anti-Jo-1 antibody. Patients who are Anti-Jo-1 positive tend to have interstitial lung disease, arthritis (non-deforming) and fevers.
- Nuclear Mi-2 protein: these are seen in approximately 20% of patients with myositis, and are more specific for dermatomyositis.
- Signal recognition peptide (SRP): anti-SRP antibodies are seen in approximately 5% of patients with polymyositis, and are associated with poor response to treatment and a poor prognosis.

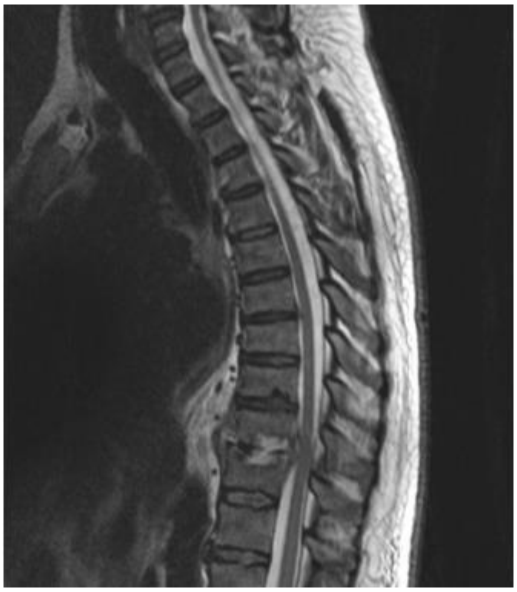
Myositis-associated antibodies are seen in up to 50% of patients with myositis. They are typically seen in other connective tissue diseases. Examples include anti-PM/Scl antibodies (seen in polymyositis/systemic sclerosis overlap disease), and anti-Ku antibodies, seen in overlap myositis/connective tissue diseases

Question 63 of 79

A 71-year-old man is being treated for urosepsis. For the past seven days, he has experienced significant back pain which is exacerbated when mobilising with the physiotherapy team. He has occasionally suffered from back pain in the past however reports that his pain at present is far more severe than anything he is used to.

He has a past medical history of type 2 diabetes and adenocarcinoma of the colon for which he is awaiting definitive surgical treatment.

An MRI spine (T2) is performed and displayed below:



What is the most likely diagnosis?

A. Bony metastases
B. Disc prolapse
C. Discitis
D. Osteoarthritis
E. Vertebral fracture

C. Discitis

This man's MRI shows a high signal intensity mass within the T10-11 disc space which is indicative of discitis. He is at a significantly increased risk of developing this condition given his ongoing systemic infection and immunocompromise (type 2 diabetes and active cancer).

Bony metastases would be of particular concern given this man's adenocarcinoma of the colon however the MRI images do not show any bony pathology. Instead, the radiological abnormality relates to the T10-11 intervertebral disc. Metastases are therefore an incorrect answer.

Disc prolapse is a differential diagnosis here given this man's ongoing physiotherapy. The mild impingement of the spinal cord from the intervertebral disc here is in keeping with prolapse however the increased intensity signal and inflammation is characteristic of discitis which is, therefore, the correct answer.

Due to the acute onset of symptoms and the MRI indicative of discitis, osteoarthritis of the spine is not the most likely diagnosis here.

The MRI shows an intervertebral disc pathology but no vertebral fractures. This is therefore also incorrect.

Question 64 of 79

A 43-year-old patient presents to the emergency department with one day of fever, abdominal pain, joint pain and chest pain. This is the third time this has occurred. He has no past medical history and does not take any regular medications. He is of Turkish descent.

Observations:

- Temperature 38.4
- Blood pressure 120/66 mmHg
- Respiratory rate 18/minute
- Oxygen saturations 96% on room air
- Heart rate 88 beats per minute

On examination, there is generalized abdominal tenderness. Synovitis is detectable at the left ankle. There is a pericardial rub.

Blood tests:

Hb	138 g/L	Male: (135-180)Female: (115 - 160)
Platelets	189 * 10 /L 9	(150 - 400)
WBC	4.5 * 10 /L 9	(4.0 - 11.0)
Na +	137 mmol/L	(135 - 145)
K +	4.2 mmol/L	(3.5 - 5.0)
Urea	5.2 mmol/L	(2.0 - 7.0)
Creatinine	88 µmol/L	(55 - 120)
CRP	52 mg/L	(< 5)
Antinuclear antibody	negative	(negative)

What is the most appropriate medication to administer?

A. Anakinra
B. Co-amoxiclav
C. Colchicine
D. Hydroxychloroquine
E. Prednisolone

C. Colchicine

Colchicine may be helpful in Familial Mediterranean Fever

Colchicine is the correct answer. This patient presents with recurrent episodes of fever, abdominal pain, chest pain and joint pain. Clinical examination suggests arthritis, peritonitis and pericarditis. Additionally, he is of Turkish descent. Taken together, this suggests a diagnosis of Familial Mediterranean Fever (FMF). The first-line treatment of FMF is colchicine. It can abort and prevent attacks.

Anakinra is incorrect. This is an IL-1 inhibitor biologic medication. There is some literature to suggest this can be effective in refractory FMF but it is not first-line therapy.

Co-amoxiclav is incorrect. The recurrent constellation of arthritis, pericarditis, peritonitis and fever is more suggestive of FMF than an infection.

Hydroxychloroquine is incorrect. This medication can be used to treat systemic lupus erythematosus. While SLE can cause pericarditis, arthritis, fever and in rare cases, peritonitis, the antinuclear antibody is negative, making this diagnosis very unlikely.

Prednisolone is incorrect. This can be used to treat protracted febrile myalgia in FMF but is not first-line therapy.

Question 65 of 79

A 55-year-old woman presents for review. Her mother has just been discharged after suffering a hip fracture. She is concerned that she may have 'inherited' osteoporosis and is asking what she should do. She has no significant past medical history of note, takes no regular medication and has never sustained any fractures. She smokes around 20 cigarettes per day and drinks about 3-4 units of alcohol per day.

What is the most appropriate course of action?

A. Arrange bone mineral density measurement (DEXA scan)
B. Reassure her that assessment of fragility fracture risk does not need to be done until 65years
C. Refer her to the genetics team for a risk assessment
D. Start first-line bone protection (i.e. ensure calcium/vitamin D replete + oralbisphosphonate)
E. Use the FRAX tool

E. Use the FRAX tool

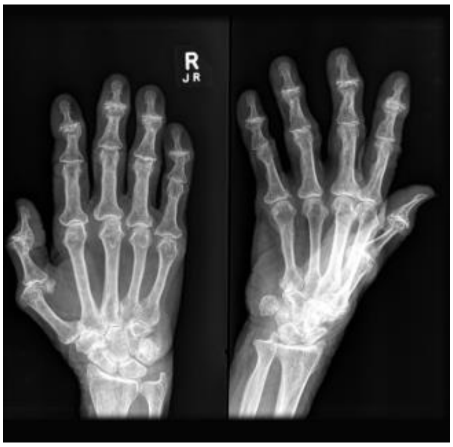
This lady has a number of risk factors for developing osteoporosis:

- positive family history
- smoking
- excess alcohol intake

She should therefore have an immediate FRAX assessment, rather than waiting until 65 years as we would for women without any relevant risk factors

Question 66 of 79

A 79-year-old woman complains of pain in her hands. An x-ray is ordered:



Based on the x-ray findings, what is the most likely diagnosis?

A. Gout
B. Primary hyperparathyroidism
C. Rheumatoid arthritis
D. Osteoarthritis
E. Paget's disease

D. Osteoarthritis

The distribution of joint problems (mainly distal interphalangeal joints and carpometacarpal joints) and changes seen (loss of joint space, subchondral sclerosis) points to a diagnosis of osteoarthritis.

Question 67 of 79

A 58-year-old man presents with malaise and fever. He states that he has been feeling unwell for several months. His main complaints are intermittent swelling and pain of his right ear, painful red eyes, and arthralgia. He has no past medical history and takes no regular medicines.

On examination, you note right auricular swelling, bilateral anterior uveitis, and a symmetrical small joint polyarthritis.

Hb	115 g/l	Na^+	138 mmol/l
Platelets	330 * 10 /l 9	K^+	4.2 mmol/l
WBC	13.1 * 10 /l 9	Urea	6.2 mmol/l
Neuts	10.4 * 10 /l 9	Creatinine	95 µmol/l
Lymphs	2.5 * 10 /l 9	CRP	132 mg/l
Eosin	0.6 * 10 /l 9	pANCA	negative
C3	normal	C4	normal
ANA	negative	Anti Sm	negative
RhF	positive	Anti CCP	negative

What is the most likely diagnosis?

A. Eosinophilic granulomatosis with polyangiitis
B. Granulomatosis with polyangiitis
C. Systemic lupus erythematosus
D. Rheumatoid arthritis
E. Relapsing polychondritis

E. Relapsing polychondritis

Relapsing polychondritis is a multi-systemic condition which most commonly causes relapsing episodes of auricular chondritis

Relapsing polychondritis is a multi-systemic condition characterised by repeated episodes of inflammation and deterioration of cartilage. It is often a painful disease which can cause joint deformity and be life-threatening if the respiratory tract, heart valves, or blood vessels are affected. The exact mechanism is poorly understood, but it is thought to be related to an immunemediated attack on cartilage proteins.

The history in this case is suggestive of relapsing polychondritis, however the clinical features are non-specific and can occur due to many of the other options. The laboratory date is therefore of great use in narrowing down the differential diagnosis.

A normal C3 and C4, and negative ANA, make SLE unlikely.

A negative pANCA and cANCA makes granulomatosis with polyangitis and eosinophilic granulomatosis less likely.

A negative anti-CCP makes rheumatoid arthritis less likely. Although RhF is positive, this is very non-specific and is positive in many other diseases and in the healthy general population.

Question 68 of 79

A 54-year-old man presents to Gastroenterology outpatient clinic, for review of his Crohn's disease which was diagnosed 3 years ago. His other past medical history includes ischaemic heart disease, hypercholesterolaemia and gout. He has been suffering from a few months of increased diarrhoea and abdominal pain, and you feel he would benefit from starting azathioprine.

Which medication is it important to ensure he is not taking before commencing azathioprine?

A. Ramipril
B. Allopurinol
C. Losartan
D. Aspirin
E. Simvastatin

B. Allopurinol

Azathioprine and allopurinol have a severe interaction causing bone marrow suppression

Allopurinol inhibits the enzyme xanthine oxidase (XO). This enzyme is also required to inactive 6- mercaptopurine (the active agent from azathioprine). When XO is inhibited, this causes an increase in 6-mercaptopurine levels, which are then shunted down a different metabolic pathway, leading to higher levels of 6-thioguanine metabolites. There are incorporated into the DNA of white blood cells, leading to reduced activation and reduced replication potential.

Coadministration of azathioprine and allopurinol requires dose reductions and extra monitoring for life threatening agranulocytosis. The other medications listed do not interact with azathioprine and require no extra monitoring.

Question 69 of 79

A 45-year-old gentleman presents to the emergency department with proximal muscle weakness of increasing intensity over the last few days. Initially he put it down to restarting gym exercises, however this is progressively getting worse.

On examination you note a slim gentleman who has difficulty in rising from the chair, and that appears to be in pain on examination of the deltoids and quadriceps. He has low grade pyrexia (37.3), however he exhibits no rashes or evidence of photosensitivity.

His bloods are as follow:

Hb	134 g/l	Na +	140 mmol/l	Bilirubin	18 µmol/l
Platelets	355 * 10 /l 9	K +	3.7 mmol/l	ALP	86 u/l
WBC	9.3 * 10 /l 9	Urea	7.7 mmol/l	ALT	101 u/l
Neuts	5.5 * 10 /l 9	Creatinine	87 µmol/l	γGT	110 u/l
Lymphs	3.3 * 10 /l 9	Albumin	32 g/l		
Eosin	0.14 * 10 /l 9	CRP	125 mg/l		
CK	2158 IU/L				

Which investigations will confirm this patient's diagnosis:

A. Anti-Jo1 antibody screen
B. Electromyography
C. Ultrasound of muscles affected
D. Muscle biopsy
E. Magnetic Resonance Imaging

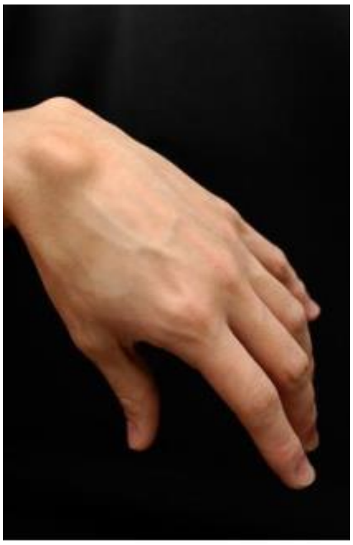
D. Muscle biopsy

Muscle biopsy is the gold standard investigation for the diagnosis of polymyositis

The history and examination findings, alongside the rise in Creatinine Kinase in this gentleman should alert us towards the potential diagnosis of Polymyositis. All of the options provided above are useful in the diagnosis of polymyositis, which makes this question slightly tricky. Anti-Jo-1 antibodies are characteristically associated with polymyositis and indeed they are likely to point us to the right direction; however given the fact that it is an Anti-nuclear antibody subtype it can be raised in other systemic inflammatory condition such as Systemic Lupus Erythematosus and Mixed Connective Tissue disorder. Electromyography can be used to distinguish between myopathic and neuropathic causes of weakness and isolate the muscle group involved; however it is fairly nonspecific. Magnetic Resonance Imaging (MRI) is nowadays commonly employed, having the benefit of being non-invasive and able to display large muscle groups, it has a high sensitivity however it's non specific and cannot necessarily differentiate the changes of inflammatory myopathy. Ultrasound of muscles has now been surpassed by the use of MRI; this leaves us with muscle biopsy. Even though it is less commonly used today due to its invasive nature and difficulty in accessing the appropriate muscle group, it remains the most sensitive and specific test in identifying polymyositis histopathologically, provided that the right area of muscle is biopsied.

Question 70 of 79

A 41-year-old woman presents with a lump over the wrist. It has been present for approximately 6 months and is not painful. She does not experience any symptoms from it and has no significant past medical history. On examination, the lump appears as shown below:



Given the likely diagnosis, what is the most appropriate next step?

A. Aspiration
B. Reassure and monitor
C. Surgical referral
D. Ultrasound
E. Wrist X-ray

B. Reassure and monitor

This appearance of this lesion is highly suggestive of a ganglion. These are simple cysts and are the most common benign lesions of the hand and wrist. Typically, they appear as seen here, over the dorsal aspect of the wrist. Onset is usually insidious. They often cause no symptoms and patients can simply be **reassured and advised to monitor**.

Aspiration is incorrect. This can be both diagnostic, if uncertain, and therapeutic. In this case, there is no indication for aspiration as the patient is not experiencing any symptoms.

A **surgical referral** is unwarranted here. This is typically employed when symptoms are severe or there are neurovascular manifestations.

Ultrasound scanning is not necessary here. The photo very clearly shows a ganglion cyst and the patient is not experiencing any symptoms.

Similarly, **X-ray** is unwarranted. The photo, and history, allow us to be almost certain of the diagnosis and so imaging is not required.

Question 71 of 79

A 46-year-old lady complains of proximal muscle weakness over 6 months. History is otherwise unremarkable. She has no past medical history and is on no medications. She does not drink alcohol. On examination, power is 4/5 proximally in both arms and legs, otherwise unremarkable. Blood investigations are normal except for an elevated creatinine kinase level at 900 U/L. Electromyography demonstrates myopathic features. You order a muscle biopsy to help differentiate which myopathy is present. The results show endomysial lymphocytic infiltrates that invade nonnecrotic muscle fibres.

What is the most likely diagnosis?

A. Dermatomyositis
B. Inclusion body myositis
C. Systemic lupus erythromatosis
D. Lung cancer
E. Polymyositis

E. Polymyositis

Proximal muscle weakness is a nonspecific complaint and the elevated CK with this presentation suggests a myopathy, of which the common are inflammatory (polymyositis, dermatomyositis, and inclusion body myositis), toxic myopathies (e.g. statin- or alcohol-induced), and inherited (Duchenne/Becker muscular dystrophy, myotonic dystrophy). There is no history of toxic agents here and the history is not lifelong to suggest an inherited causes. There are no skin changes to suggest dermatomyositis. The diagnostic difficulty may be between inclusion body myositis and polymyositis. The former tends to affect wrists and fingers more than the latter. Where there is diagnostic difficulty clinically, a biopsy may help make a diagnosis:

Endomysial lymphocytic infiltrates that invade nonnecrotic muscle fibres = polymyositis Perimysial inflammation of lymphocytes and parafascicular atrophy = dermatomyositis Inflammatory infiltrates and inclusions within muscle fibres = inclusion body myositis. Polymyositis responds to immunosuppression.

It is likely she has a polymyositis and coincidental carpal tunnel syndrome rather than an inclusion body myositis.

Question 72 of 79

9. A 39 year old woman presents to her GP with symptoms of dysuria and increased urinary

frequency for the past three days. She also complains of lower abdominal pain but has no overt

signs of systemic sepsis. Examination is entirely normal aside from mild suprapubic pain. Urine dip

correlates with a diagnosis of urinary tract infection with positive nitrites, leukocytes, blood and protein. The sample is sent for culture. The patients medical history is significant only for rheumatoid arthritis for which she takes methotrexate, folic acid, ibuprofen and omeprazole.

Which one of the following antibiotics is contraindicated in this patient?

A. Co-amoxiclav

B. Ciprofloxacin
C. Cefpodoxime
D. Nitrofurantoin
E. Trimethoprim

E. Trimethoprim

The concurrent use of methotrexate and trimethoprim containing antibiotics may cause bone marrow suppression and severe or fatal pancytopaenia

Trimethoprim is a common bacteriostatic antibiotic used in the treatment of uncomplicated urinary tract infections. It is bacteriostatic rather than bactericidal in that it inhibits bacterial replication rather induces bacterial death. Replication inhibition is achieved by interfering with the generation of thymidine, an essential amino acid base in DNA, by inhibiting the action of the enzyme dihydrofolate reductase which is an essential component of the thymidine metabolic pathway, using folate as a primary substrate. Trimethoprim may also be combined with sulphonamide drugs such as sulphamethoxazole to create antibiotics such as co-trimoxazole; these drugs work synergistically by inhibiting alternative enzymes in the thymidine synthesis pathway.

Although trimethoprim is a relatively safe and effective drug in the treatment of urinary tract infections it does have some contraindications due to its mechanism of action. Due to its antagonism of folate metabolism, it should not be used in pregnancy, particularly the first trimester due to the risk of neural tube defects. Another effect of inhibiting folate metabolism is the impairment of metabolically active tissues such as bone marrow or cancerous cells. This effect is exploited in chemotherapy agents such as methotrexate. This drug also inhibits folate metabolism to prevent tumour growth; it can also be used in treating rheumatoid arthritis. Concomitant use of methotrexate and trimethoprim can lead to severe and potentially fatal bone marrow suppression with pancytopaenia. This likelihood is increased if the trimethoprim is coupled with a sulphonamide drug, and also in the elderly.

All the other drugs listed above may be used in conjunction with folate inhibiting chemotherapeutic agents, although penicillin containing agents and quinolone antibiotics can cause a reduction in metabolism of methotrexate and the full blood count should be monitored in prolonged courses. Similarly methotrexate and nitrofurantoin used together can cause hepatotoxicity and liver function should be closely monitored. The safest choice of drug above is cefpodoxime, an oral third generation cephalosporin, which has no significant clinical interactions with the other drugs this patient is taking.

Question 73 of 79

A 40-year-old man is stable on warfarin therapy for the treatment of atrial fibrillation. Whilst on a stag party in Spain he develops scrotal pain and itching. Whilst in Spain, he is treated with a course of ciprofloxacin for a presumed urinary tract infection. Two weeks later he develops a hot, red, swollen and painful knee and both elbows become inflamed. On examination in the emergency department you identify localised tenderness of the knee and painful movement in all directions. The knee is red and hot. Both elbows are mildly warm, with painful movement on flexion and extension. His conjunctiva are also red. He is afebrile. Examination of his external genitalia is essentially normal however there is evidence of excoriation around the scrotum.

What is the cause of his knee pain?

A. Reactive arthritis
B. Still's disease
C. Septic arthritis
D. Gout
E. Haemarthrosis

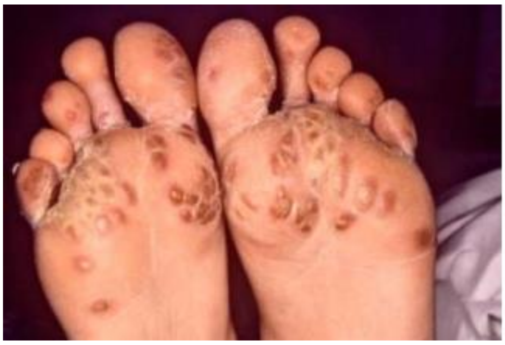
A. Reactive arthritis

Reactive arthritis affects the joints, eyes and genitourinary/gastrointestinal system. It has been associated with gastrointestinal (GI) infections including Shigella, Salmonella, Campylobacter, and other organisms, as well as with genitourinary (GU) infections (especially with Chlamydia trachomatis). The clinical triad commonly occurs 1-4 weeks following exposure.

Ciprofloxacin interacts with warfarin causing prolonged INR which is a risk factor for haemarthrosis, but the history of conjunctivitis and GU symptoms does not support this.

Question 74 of 79

A 34-year-old man presents for review. He has been generally unwell since an episode of diarrhoea four weeks ago, with joint pains, pain on passing water and a rash on the soles of his feet:



What does this rash likely represent?

A. Pompholyx
B. HIV-associated dermopathy
C. Plantar pustular psoriasis
D. Mosaic warts
E. Keratoderma blennorrhagica

E. Keratoderma blennorrhagica

The correct answer is keratoderma blennorrhagica. Keratoderma blennorrhagica is a skin condition associated with reactive arthritis, which is an autoimmune response to an infection in another part of the body. The patient's history of diarrhoea, joint pains, and pain on passing water suggests that he may have reactive arthritis. The rash on the soles of his feet supports this diagnosis as keratoderma blennorrhagica typically presents as brownish-red hyperkeratotic plaques on the soles and occasionally palms.

Pompholyx is an incorrect answer because it presents as small, intensely itchy vesicles on the fingers, palms, and soles. It does not match the description or appearance of the rash seen in this case.

HIV-associated dermopathy is also incorrect because it usually presents as a widespread pruritic rash involving multiple body sites rather than being confined to the soles. Moreover, there are no other clinical features mentioned in the case that would suggest HIV infection.

Plantar pustular psoriasis can be ruled out because although it affects the soles of feet and can cause discomfort during walking, it typically presents with pustules and scale formation which are not observed in this case.

Finally, mosaic warts are an incorrect answer because they present as grouped verrucae (warts) with a rough surface on weight-bearing areas of the foot. These warts are caused by human papillomavirus (HPV) infection and do not fit with the clinical picture described for this patient.

Question 75 of 79

A 52-year-old woman presents with several months' history of fatigue and dry eyes. She also describes a significantly dry mouth, to the point that she sometimes finds it difficult to eat food. She has no significant past medical history.

On examination, the eyes appear mildly red. The tongue appears dry. Given the suspected diagnosis, the doctor requests an initial blood test:

Antinuclear antibodies	1:1600	(<1:280)

What is the most definitive test to confirm the likely diagnosis?

A. Anti-La antibody titre
B. Anti-Ro antibody titre
C. Salivary gland biopsy
D. Schirmer's test
E. Sialometry

C. Salivary gland biopsy

Salivary gland biopsy is the most definitive way of confirming the diagnosis of primary Sjogren's syndrome - sections will show a typical lymphocytic infiltrate

The diagnosis here is that of Sjogren's syndrome, an autoimmune disorder affecting exocrine glands and resulting in dry mucosal surfaces. The high antinuclear antibody titre is non-specific, but reinforces the diagnosis of an autoimmune condition and is present in 70% of patients. Of the options listed above, the most definitive diagnostic tool is **salivary gland biopsy**. This will show a focal lymphocytic infiltration.

Anti-La antibodies are found in 30% of patients with primary Sjogren's syndrome. This is not particularly specific or sensitive for the condition, and certainly not as definitive a test as biopsy.

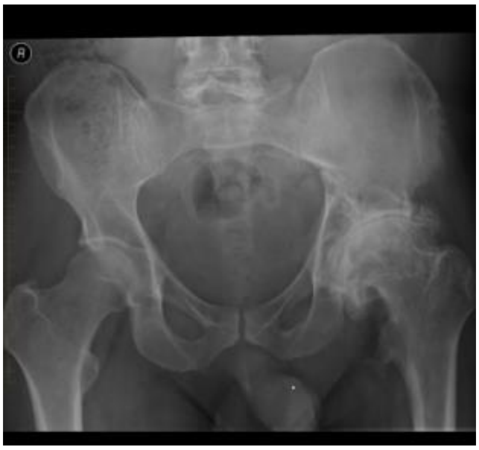
Similarly, **anti-Ro antibodies** are not entirely specific or sensitive for Sjogren's syndrome. They are found in 70% of patients, rather than the 30% seen with anti-La, but are still not as definitive as biopsy.

Schirmer's test is a quantitative measurement of tears using filter paper near the conjunctival sac. It is useful to aid diagnosis but is not as definitive as salivary gland biopsy.

Sialometry measures unstimulated salivary flow for 15 minutes into a calibrated tube. This is useful to aid diagnosis but does not differentiate between causes of dry mouth, of which Sjogren's syndrome is just one cause.

Question 76 of 79

An 80-year-old man presents is taken to the Emergency Department after falling at home. His daughter notes that he fell onto his left side. An x-ray is taken of the pelvis:



What is the diagnosis?

A. Paget's disease of the bone
B. Severe osteoarthritis
C. Left intertrochanteric fracture
D. Left subcapital fracture
E. Myeloma

B. Severe osteoarthritis

This x-ray shows advanced osteoarthritic changes at the left hip joint; loss of joint space and subchondral sclerosis are prominent.

Question 77 of 79

An 80-year-old retired GP with no past medical history presents to the hospital with a 6-month history of muscle aches and weakness. She also has difficulty swallowing and has had 3 courses of antibiotics for a presumed chest infection in the last 3 months. In the last 2 days she has been struggling to cope at home and has had two falls.

Blood tests show :

Erythrocyte Sedimentation Rate (ESR) 60 mm/hour g/l
Creatinine Kinase 8000 U/L

Which of the following investigations would be LEAST helpful in the workup?

A. Autoimmune profile
B. FBC
C. AST and ALT

D. Urine myoglobin
E. Renal biopsy

E. Renal biopsy

This lady seems to have polymyositis. An autoimmune profile is useful as ANA is positive in onethird of patients. Anti-jo antibodies are positive in 20% of patients and indicate a poorer prognosis with interstitial lung disease. A full blood count may show leukocytosis or thrombocytosis. AST/ALT are both muscle enzymes that will be elevated. Creatinine kinase will also be elevated along with urine myoglobin. In some cases, the patient may develop renal impairment from rhabdomyolysis and thus U+Es should be monitored. A renal biopsy would not be diagnostically helpful in this situation.

Question 78 of 79

A 56-year-old man is seen in the emergency department with a 2-week history of worsening fatigue and exertional shortness of breath. He denies any recent infective symptoms or syncopal episodes. His past medical history includes Crohn's disease, for which he was recently started on azathioprine, type 2 diabetes, epilepsy, and gout.

On examination, he is pale. Chest sounds are clear and heart sounds are normal. He has a soft and non-tender abdomen.

Laboratory tests:

Hb	81 g/L	(135 - 180)
Platelets	66 * 10 /L 9	(150 - 400)
WBC	1.1 * 10 /L 9	(4.0 - 11.0)
Neuts	0.3 * 10 /L 9	(2.0 - 7.0)
Na +	136 mmol/L	(135 - 145)
K +	4.1 mmol/L	(3.5 - 5.0)
Urea	3.1 mmol/L	(2.0 - 7.0)
Creatinine	83 µmol/L	(55 - 120)

Given this patient's presentation, what medication should be stopped?

A. Allopurinol
B. Gliclazide
C. Lansoprazole
D. Metformin
E. Sodium valproate

A. Allopurinol

Azathioprine and allopurinol have a severe interaction causing bone marrow suppression

This patient's blood results show pancytopenia with low haemoglobin, platelets and white cell count. He is also experiencing symptoms of anaemia with increasing lethargy and exertional shortness of breath. When investigating a patient with symptomatic pancytopenia, it is important to exclude any iatrogenic causes, such as medication. This patient has recently been started on azathioprine, an immunosuppressant medication. Of the drugs listed in this question, azathioprine must not be given in conjunction with **allopurinol** as the 2 drugs cause an increased risk in haematological toxicity, such as that seen in this patient.

Gliclazide is a sulfonylurea used in the treatment of diabetes mellitus. The most important side effect to note is the risk of hypoglycaemia.

Lansoprazole is a proton pump inhibitor used in the treatment of gastro-oesophageal reflux disease. Side effects include electrolyte imbalances such as hypomagnesaemia and an increase in the risk of *Clostridium difficile* infection.

Metformin is used in the treatment of diabetes mellitus. Gastrointestinal side effects are the most common including diarrhoea and abdominal pain. It does not interact with azathioprine.

Sodium valproate is an anti-epileptic medication that has a number of side effects including pancreatitis, weight gain, alopecia and hepatic toxicity. Rarely, it can cause agranulocytosis. However, it does not interact with azathioprine and would not be contraindicated in this patient.

Question 79 of 79

A 65-year-old lady is admitted urgently to hospital after her General Practitioner noted an abnormality in routine bloods. She is known to suffer from rheumatoid arthritis. Current medications include methotrexate 15 mg once per week, folic acid 5 mg (6 days of the week), ramipril 2.5 mg OD, atorvastatin 40 mg OD. She was recently commenced on antibiotics for a urinary tract infection. The bloods are the following:

Hb	68 g/l
Platelets	65 * 10 /l9
WBC	2.1 * 10 /l9
Neutrophils	0.5 * 10 /l9
Lymphocytes	0.7 * 10 l9
Monocytes	0.1 * 10 l9

Which antibiotic prescribed for this lady could explain this picture?

A. Nitrofurantoin
B. Trimethoprim
C. Co-amoxiclav
D. Cefalexin
E. Ciclosporin

B. Trimethoprim

The concurrent use of methotrexate and trimethoprim containing antibiotics may cause bone marrow suppression and severe or fatal pancytopaenia

Methotrexate is a folic acid antagonist affecting the cell cycle. Trimethoprim is also a folic acid antagonist. When combined together the risk of myelosuppression is increased, given also the fact that trimethoprim reduces the renal excretion rate of methotrexate.

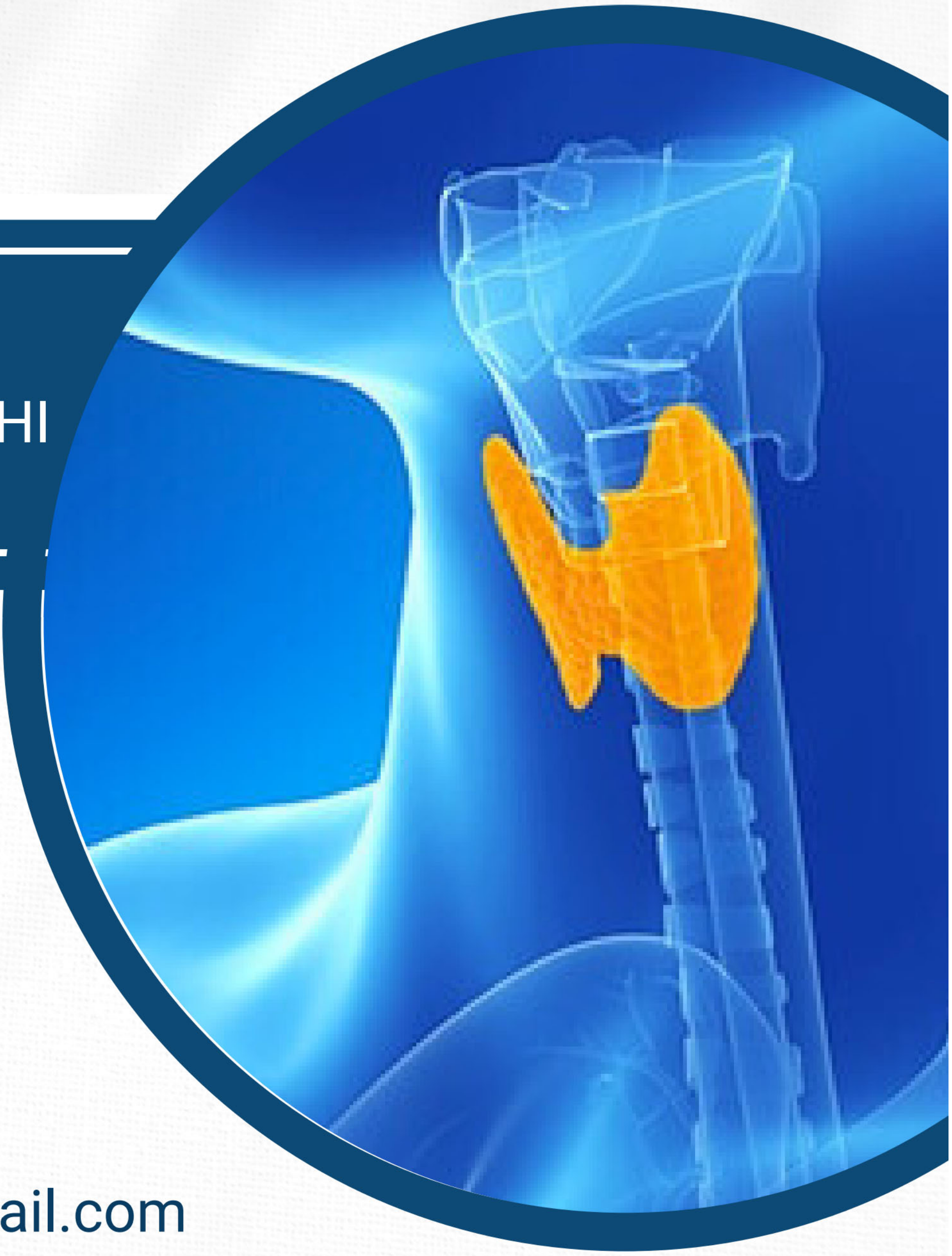
Alrajhi Jad

PASSMEDICINE

ENDOCRINOLOGY

Internal medicine

EDITED BY
DR. JAMIL ALRAJHI



2024

773-555-493
jameelalrajhi@gmail.com

PASSMEDICINE PART 2
ENDOCRINE
2024

Question 1 of 348

A 48-year-old woman presents with a 2-month history of fatigue, anorexia and nausea. She has also noticed some darkening of the skin on her hands. Prior to this, she was fit and well, with no significant past medical history. She has a strong family history of Hashimoto's thyroiditis.

On examination, there is some evidence of wasting of the face, and noticeable hyperpigmentation of the palmar creases. Her blood pressure is 105/75 mmHg. Blood tests are taken:

Na +	134 mmol/L	(135 - 145)
K +	5.3 mmol/L	(3.5 - 5.0)
Urea	6.8 mmol/L	(2.0 - 7.0)
Creatinine	76 µmol/L	(55 - 120)
Calcium	2.43 mmol/L	(2.1-2.6)
TSH	1.2 mU/L	(0.5-5.5)
Free T4	13.3 pmol/L	(9.0 - 18)

An initial test is conducted to confirm the likely diagnosis.

Which subsequent test can be performed to distinguish a primary cause from a secondary cause?

A. High-dose dexamethasone suppression test
B. Long Synacthen test
C. Low-dose dexamethasone suppression test
D. Short Synacthen test
E. Urinary metanephrines

B. Long Synacthen test

The long Synacthen test can be used to distinguish primary adrenal failure from secondary adrenal failure

The diagnosis here is that of adrenal insufficiency. The history of fatigue, anorexia and nausea points towards this, along with evidence of hyperpigmentation of the palmar creases, hypotension, hyponatraemia and hyperkalaemia. A primary failure is known as Addison's disease. To make the initial diagnosis of adrenal failure, a short Synacthen test should be used - cortisol will fail to rise. However, the question is asking specifically how to differentiate primary from secondary adrenal failure, secondary being a failure of the pituitary to make sufficient ACTH. For this, the long Synacthen test should be used - a higher dose is given and then cortisol levels measured over a longer time period. With primary failure, the adrenals will still be unable to produce cortisol, but with secondary failure, the exogenous ACTH will eventually push the adrenals to produce cortisol.

The high-dose dexamethasone suppression test is used to differentiate primary and secondary causes of Cushing's syndrome, not adrenal insufficiency. This would have instead presented with weight gain and hypokalemia, not the other way around.

The low-dose dexamethasone suppression test is used first-line to make the initial diagnosis of Cushing's. It does not play a role here.

The short Synacthen test is used to initially diagnose adrenal insufficiency. It does not, however, differentiate between primary and secondary causes - the long test does this.

Urinary metanephrines are used to diagnose pheochromocytoma. In this condition, there is increased secretion of catecholamines and patients present with hypertension, headaches, sweating and palpitations.

Question 2 of 348

A 55-year-old female presents with light-headedness and abdominal pain. She has a past medical history of asthma for which she takes regular beclometasone and as required salbutamol. On examination her blood pressure is 95/75 mmHg and heart rate 115 beats per minute.

Blood results are as follows:

Hb	135 g/l	Na +	129 mmol/l
Platelets	352 * 10 /l 9	K +	5.2 mmol/l
WBC	14.2 * 10 /l 9	Urea	10.2 mmol/l
Neuts	10.3 * 10 /l 9	Creatinine	115 µmol/l
Lymphs	2.2 * 10 /l 9	CRP	8 mg/l

A short Synacthen test is performed:

Time (minutes)	0	30	60
Cortisol (nmol/l)	150	165	212

A long Synacthen test is then performed:

Time (hours)	1	2	8	24
Cortisol (nmol/l)	202	420	820	1626

What is the most likely cause?

A. Adrenal Cushing's syndrome
B. Secondary adrenal insufficiency
C. Primary adrenal insufficiency (Addison's disease)
D. Cushing's disease
E. Iatrogenic adrenal insufficiency

B. Secondary adrenal insufficiency

The long Synacthen test can be used to distinguish primary adrenal failure from secondary adrenal failure

The clinical and biochemical results are suggestive of adrenal insufficiency. Biochemical results which suggest adrenal insufficiency include hyponatraemia, hyperkalaemia, a normal anion gap metabolic acidosis and hypoglycaemia.

The short Synacthen test demonstrates failure of cortisol to rise which confirms the diagnosis of adrenal insufficiency. A normal response is defined as a 30 minute serum cortisol concentration greater than 420 nmol/L.

The next step is to localise the lesion. This can be achieved by measurement of ACTH or by performing the long Synacthen test. Interpretation of this test is governed by the following:

1. In primary adrenal failure: we would not expect there to be a significant rise in cortisol during the long Synacthen rest since the adrenal glands are intrinsically dysfunctional
2. In secondary adrenal failure: chronically low levels of ACTH due to pituitary failure result in atrophy of the adrenal glands. Prolonged stimulation of the adrenal glands by ACTH in the long Synacthen test results in a degree of recovery by the adrenal glands resulting in a significant rise in cortisol. A response that rises gradually to a peak at 24 hours occurs in secondary adrenal failure. This pattern of results also occurs due to prolonged corticosteroid therapy use.

An important point is to remember that in some cases of long-standing adrenal atrophy due to secondary adrenal insufficiency, the adrenal glands will not respond even after 24 hours and will require several daily doses of depot Synacthen before an adrenal response is seen. The majority of these cases should be identifiable by measurement of plasma ACTH, which would be expected to be very low (in contrast to primary adrenal insufficiency where ACTH levels are very high).

In this case, the long Synacthen test demonstrates a cortisol which rises gradually to a peak at 24 hours confirming the diagnosis of secondary adrenal failure. The differential is now between iatrogenic adrenal insufficiency and secondary adrenal insufficiency due to pituitary disease. The patient is on inhaled steroids for asthma, however this is highly unlikely to cause adrenal insufficiency unless the patient was on a liver enzyme inhibitor (e.g. ketoconazole). Therefore the most likely diagnosis is secondary adrenal insufficiency.

Question 3 of 348

A 28-year-old woman is due to be discharged from the hospital. She was admitted four days ago following a month of feeling unwell with vomiting, postural dizziness and weight loss. Following a Synacthen test, she was diagnosed with Addison's disease. She was started on treatment with hydrocortisone three times a day, as well as fludrocortisone on a daily basis. She has had no other medical problems or treatments in the past. She has been taking hydrocortisone 10mg at 09:00 and then 5mg at 12:00 and 15:00. On discharge she informs the medical team that she sometimes does shift-work.

What is the most appropriate advise to give regarding steroid dosing for night shifts?

A. Take doses at 09:00, 12:00 and 15:00 regardless of shift patterns
B. Adjust to take first dose at waking, then doses at three hoursand six hours from starting
C. On shift days take doses at 09:00, 12:00 and 15:00, but also takean additional 10mg at starting shift
D. On shift days take doses at 09:00, but then take second dose at starting shift and third dose six hours into shift
E. On shift days omit taking steroids

B. Adjust to take first dose at waking, then doses at three hoursand six hours from starting

For patients on steroid replacement when working shift work, doses should be taken from when waking

Patients with Addison's disease need both glucocorticoid and mineralocorticoid replacement. Cortisol is highly linked to diurnal rhythm - which is why cortisol naturally peaks in the morning and is at its nadir between midnight and 04:00. Replacement should aim to match the natural rhythm as much as possible; dosing is normally split between three doses, first on waking, then at midday and early afternoon. When a patient shifts their daytime routine, such as working on night shifts or travelling, the patient should be advised to take their morning dose on waking and maintain the timing from there. Patients should not be advised to omit doses, and encouraged to always ensure they are well supplied.

Question 4 of 348

A 35-year-old woman presents with weight gain for the last couple of years. She feels that most weight has been gained on her face and abdomen. On further questioning, her periods have been irregular for some time. She was diagnosed with type 2 diabetes mellitus last year. The patient drinks approximately 15 units of alcohol per day.

On examination, she has violaceous striae across the abdomen. Proximal muscle weakness is also evident when asking the patient to stand. Blood tests are taken:

Na +	143 mmol/L	(135 - 145)
K +	2.8 mmol/L	(3.5 - 5.0)

The team suspects that the patient's alcohol use may be the underlying cause of her presentation, rather than an endogenous cause, and want to differentiate the two.

What is the most appropriate investigation?

A. High-dose dexamethasone suppression test
B. Insulin stress test
C. Low-dose dexamethasone suppression test
D. Petrosal sinus sampling
E. Short synacthen test

B. Insulin stress test

The insulin tolerance test can be used to distinguish Cushing's syndrome from pseudo-Cushing's

This patient may have pseudo-Cushing's syndrome as a result of alcohol use, as opposed to true Cushing's syndrome. Patients with pseudo-Cushing's syndrome display clinical and biochemical features similar to those seen in Cushing's syndrome, such as the scenario here, but the presentation is caused by alcohol abuse, rather than an endogenous source or steroid use. Other causes of pseudo-Cushing's include severe depression, eating disorders, and chronic illness. Of the options listed above, the **insulin stress test** is used to differentiate between true Cushing's and pseudo-Cushing's.

The **high-dose dexamethasone suppression test** is used to differentiate pituitary from ectopic sources of Cushing's syndrome. It is not useful in pseudo-Cushing's.

The **low-dose dexamethasone suppression test** is used as a first-line test for patients with suspected Cushing's syndrome. It will not help to differentiate between Cushing's and pseudo-Cushing's.

Petrosal sinus sampling is performed for patients with confirmed ACTH-dependent Cushing's syndrome without an obvious pituitary lesion on MRI. Blood is sampled peripherally and from the inferior petrosal sinuses and the ratio of ACTH is measured. It is not useful to distinguish Cushing's from pseudo-Cushing's.

The **short synacthen test** is not relevant here. It is involved in the diagnosis of Addison's disease, rather than Cushing's syndrome.

Question 5 of 348

A 34-year-old woman has been referred to your clinic with weight gain and irregular periods.

Observations: heart rate 95bpm, blood pressure 155/92 mmHg.

Hb	110 g/L	Male: (135-180)Female: (115 - 160)
MCV	112 fl	(80-100)
Platelets	199 * 10 /L 9	(150 - 400)
WBC	5.5 * 10 /L 9	(4.0 - 11.0)
Bilirubin	17 µmol/L	(3 - 17)
ALP	138 u/L	(30 - 100)
ALT	55 u/L	(3 - 40)
9AM Cortisol	461 nmol/L	(140-690)
Midnight Cortisol	154 nmol/L	(140-690)
Cortisol following insulin stress test	645 nmol/L	(>170 rise compared to 9 AM cortisol)
Urine free cortisol	802 nmol/24h	(<120)
ACTH	13.3 pmol/L	(2.2-13.3)

MRI head: unremarkable intracranial appearances.

CT thorax abdomen pelvis: fatty changes within the liver with parenchymal heterogeneity, pelvic viscera unremarkable, clear lung fields.

What is the most likely diagnosis?

A. Cushing's disease
B. Cushing's syndrome
C. Exogenous steroid use
D. Hypothyroidism
E. Pseudo-Cushing's

E. Pseudo-Cushing's

The insulin tolerance test can be used to distinguish Cushing's syndrome from pseudo-Cushing's

In this case, we have a patient with cushingoid features - weight gain, hypertension, and irregular periods. Her blood tests show macrocytic anaemia and abnormal liver function tests which could be in keeping with alcohol excess. A CT scan showing fatty liver changes adds weight to this assumption. Imaging has not shown any obvious adenoma, ovarian cysts, or gland hypertrophy.

Pseudo-Cushing's is the correct answer. Pseudo-Cushing's is also known as 'physiological hypercortisolism'. This can be a result of pregnancy, polycystic ovarian syndrome, chronic alcohol abuse, depression, and severe physical stress (e.g. infection, surgery). In these cases, there is physiological over-activity of the hypothalamic-pituitary-adrenal axis in response to these states. It is difficult to differentiate it from Cushing's syndrome. Features of pseudo-Cushing's include; normal or mildly raised serum cortisol, maintained diurnal variation in serum cortisol, a raised urine free cortisol, and a rise in cortisol levels of >170nmol following an insulin stress test. Some Cushing's symptoms such as striae, easy bruising, and proximal myopathy are not usually seen in pseudoCushing's.

Cushing's disease is a particular type of Cushing's syndrome where a pituitary tumour causes high levels of cortisol due to increased ACTH secretion. This can be differentiated from adrenal Cushing's by checking ACTH levels (these would be high in Cushing's disease), finding a pituitary adenoma on MRI brain, CRH stimulation testing (in Cushing's disease you would see a high ACTH level), and inferior petrosal sinus sampling (high levels of ACTH in these samples compared to serum). In this case, Cushing's disease is not the correct answer as MRI scanning did not show a pituitary lesion, we would also expect to see a higher ACTH level.

Cushing's syndrome is a syndrome of cortisol excess. Unlike pseudoCushing's, this is not in response to a particular physiological state but is due to an abnormality in the hypothalamic-pituitary-adrenal axis. In Cushing's syndrome, you would expect to see high serum cortisol and high urine free cortisol, but generally to a higher degree than seen in this case. There would also be a loss of diurnal variation and the rise in cortisol in response to the insulin stress test would be blunted as the sustained hypercortisolism suppressed CRH secretion.

Exogenous steroid use is an important differential, good history taking is extremely important. In exogenous steroid use, we would expect to see low ACTH and low cortisol levels.

Hypothyroidism may present with the symptoms this patient has, but would not explain the abnormal urine free cortisol.

Question 6 of 348

A 37-year-old man presents to the endocrinology clinic for review. He was started on hydrocortisone for Addison's disease six months ago following a history of feeling generally unwell, postural dizziness and vomiting. Following starting hydrocortisone, he felt much better. He was originally started on 10mg in the morning and 5mg at lunchtime and 5mg in the early afternoon. This was further increased to 10mg, 10mg and 5mg for the morning, lunchtime, and afternoon, respectively. The patient feels that all of the original symptoms have resolved, but he has noticed swelling of his ankles. On examination, he has non-pitting oedema of his legs. He has a heart rate of 78bpm, blood pressure of 165/102mmHg, saturations of 98% on room air and a respiratory rate of 14 breaths per minute. His previous blood pressure recorded in clinic had been 105/61mmHg He has no other medical problems, and takes no other tablets apart from paracetamol and ibuprofen when needed, especially for headaches.

What is the most appropriate action?

A. Start amlodipine

B. Start ACE inhibitor
C. Reduce dose of hydrocortisone
D. Increase dose of hydrocortisone
E. Start beta-blocker

C. Reduce dose of hydrocortisone

Features of over-replacement of corticosteroids require dose adjustment

This is a patient on treatment for Addison's disease who has developed evidence of over-replacement. These features can be any of Cushing's disease; such as hypertension for this patient. The increase in blood pressure with steroids implies that this is not essential hypertension but rather secondary hypertension. This implies that he is having a too much steroid replacement and hydrocortisone dose should be reduced. Features of over-replacement include hypertension, thin skin, striae, easy bruising, hyperglycaemia and electrolyte abnormalities. Features of under-replacement include fatigue, postural hypotension, weight loss and salt craving.

Question 7 of 348

A 24-year-old woman is reviewed in the emergency department. She has presented with vomiting and feeling 'light-headed'. She is known to have a history of Addison's disease and has been unable to take her normal dose of hydrocortisone and fludrocortisone over the last five days due to nausea and vomiting. The past medical history includes hypothyroidism and vitiligo.

Her normal medication is:

- hydrocortisone 10mg, 10mg and 5mg at morning, lunchtime and afternoon
- fludrocortisone 100 micrograms
- levothyroxine 100 micrograms

So far she has been given hydrocortisone 100mg IV and 2L of IV fluids, as well as 10mg of metoclopramide IV. Her nausea has settled but she is still struggling to eat or drink. Her systolic blood pressure has increased from 82mmHg to 110mmHg. Capillary glucose is 8.5mmol/l.

She is prescribed regular IV hydrocortisone and anti-emetics.

What further prescription would be appropriate at this stage?

A. Start oral hydrocortisone
B. Immediate fludrocortisone
C. Start insulin on a sliding scale
D. Further IV fluids
E. Start IV antibiotics

D. Further IV fluids

In an adrenal crisis, hydrocortisone is needed at high dose and fludrocortisone can be omitted as hydrocortisone has mineralocorticoid activity

This is a patient with known Addison's disease who presented unwell having been unable to take regular hydrocortisone doses. The key management is immediate IV hydrocortisone with plenty of IV fluids. When the patient is out of the immediate management stage, feeling better, and able to tolerate oral intake then hydrocortisone can be restarted orally. As this patient has not managed oral intake yet, prescribing oral hydrocortisone may lead to further missed doses. When the patient is having high dose IV hydrocortisone, there is significant mineralocorticoid activity, meaning that fludrocortisone is not needed. She is unlikely to need insulin at all, unless there is evidence of type 1 diabetes mellitus, which can be associated with Addison's disease.

Question 8 of 348

A 43-year-old woman with a background of anaemia and epilepsy is referred to the endocrinology clinic with fatigue, weight loss, abdominal pain, nausea, and vomiting. She does not take any steroids. On examination, there is no evidence of any abnormal pigmentation. A series of cortisol measurements are taken after 250µg of synacthen injection at 9 am:

Baseline cortisol	190 nmol/L	(>180 nmol/L)
Cortisol at 30min	390 nmol/L	(>420 nmol/L)
Cortisol at 60min	520 nmol/L	(>420 nmol/L)

What is the most appropriate next investigation?

A. Insulin tolerance test
B. Long synacthen test
C. Oral glucose tolerance test
D. Overnight dexamethasone suppression test
E. Water deprivation test

B. Long synacthen test

The long Synacthen test can be used to distinguish primary adrenal failure from secondary adrenal failure

This patient has signs of adrenal failure and undergoes a short synacthen test.

Generally, a cortisol of >420nmol/L at 30 or 60 minutes post-synacthen indicates an adequate adrenal response. The test in this patient shows a borderline response. Although this makes primary adrenal failure less likely, the short synacthen test is less sensitive to secondary adrenal failure. It is important to investigate further when results are borderline and there is a strong clinical history. Generally, secondary adrenal insufficiency presents similarly to Addison's disease but with less hypovolaemia and no pigmentation.

A long synacthen test (where samples are taken at 1, 4, 8, and 24 hours) can be used to distinguish Addison's from secondary causes of adrenal insufficiency such as steroid use, panhypopituitarism, and isolated failure of adrenocorticotrophic hormone (ACTH).

The insulin tolerance test to the point of extreme hypoglycemia is considered the gold standard for assessing the integrity of the hypothalamic-pituitary-adrenal axis. It can be used in the diagnosis of panhypopituitarism. However, this test is dangerous and contraindicated in those with epilepsy or ischaemic heart disease.

The oral glucose tolerance test determines how quickly glucose is cleared from the blood. It is usually used to test for diabetes, insulin resistance, or impaired beta-cell function. It can also be used alongside insulin-like growth factor 1 (IGF-1) levels to diagnose acromegaly.

The overnight dexamethasone suppression test is useful in identifying adrenal hyperfunction (Cushing's syndrome) or failure (Addison's). It is unlikely to be useful after a negative short synacthen test, which is the gold standard for diagnosis of Addison's.

The water deprivation test is used in the diagnosis of diabetes insipidus.

Question 9 of 348

A 32-year-old patient with Addison's disease comes for review into the endocrinology clinic. He was diagnosed by Addison's disease nine months ago and has since then been started on 20mg of hydrocortisone taken in divided doses and 50micrograms of fludrocortisone. He complains of feeling intermittently tired but has otherwise is well. He has a history of hypothyroidism and takes 100 micrograms of levothyroxine for this. He has no other medical problems and feels well in his mood. His most recent TSH was done one month ago, and showed his thyroid function to be well within control.

What investigation could be offered to assess if the dose of hydrocortisone represents accurate glucocorticoid replacement?

A. Dexamethasone suppression test
B. Cortisol day curve
C. Synacthen test
D. Morning serum cortisol
E. Urinary electrolytes

B. Cortisol day curve

A cortisol curve can be used to assess how appropriate dosing of glucocorticoid steroids in Addison's disease patients is

A cortisol day curve allows serial measurements to be taken to monitor how well cortisol is being replaced in patient with Addison's disease as well other causes of adrenal insufficiency such as panhypopituitarism. Morning serum cortisol is an appropriate investigation as first line testing for Addisons's disease, with Synacthen testing to confirm that diagnosis. Dexamethasone suppression testing can confirm Cushing's syndrome. Urinary electrolytes would be appropriate in the diagnosis of SIADH.

Question 10 of 348

A 29-year-old man with known Addison's disease contacts his endocrinologist. He has signed up for a marathon and is wondering what to do with hydrocortisone and fludrocortisone for the event. He is normally fit and well without any other medical problems. His diagnosis was established five years ago and since then he has had two admissions due to an inability of taking hydrocortisone and now has emergency doses of IM hydrocortisone.

What is the most appropriate advice to give?

A. Take doses as normal for the marathon
B. Double the hydrocortisone dose but not the fludrocortisone dose for the marathon
C. Double the fludrocortisone dose but not hydrocortisone dose for the marathon
D. Double both the fludrocortisone dose and the hydrocortisone dose for the marathon
E. Omit both medications for the marathon

D. Double both the fludrocortisone dose and the hydrocortisonedose for the marathon

A patient with Addisons' disease who undertakes significantly strenuous activity should double the dose of glucocorticoid and mineralocorticoids

This is a patient with Addison's disease who is planning significantly strenuous activity. In such a situation, both fludrocortisone dose and the hydrocortisone dose should be doubled, with advice to increase fluid intake as well. If the patient was just on hydrocortisone, then no additional fludrocortisone would be needed.

Glucocorticoid therapy should ideally mimic endogenous cortisol rhythm with the lowest level at time of falling asleep and highest at waking. Treatment should aim to control symptoms at the smallest dose possible. Hydrocortisone has a short half-life but excellent bioavailibility.

Mineralocorticoid therapy will be eventually required in adrenal insufficiency to counter intravascular volume depletion. It is important in the presence of increased fluid loss that the mineralocorticoid dose is adjusted. This is why doubling of the dose is advised.

If less strenuous activity, such as a long hike, was planned than increasing the dose of hydrocortisone by 5-10mg would be reasonable, without any change in fludrocortisone. This change would also apply for any day that increased activity is planned for. They should also be advised to increase fluid intake in the proportion to the increase in hydrocortisone dose.

If a patient is taking an alternative steroid regime, such as dexamethasone or prednisolone, then endocrinology advise would be needed.

Omitting any medications could be dangerous and precipitate an Addisonian crisis.

Question 11 of 348

A 42-year-old woman presented to ambulatory care clinic with a three-month history of increasing tiredness, lethargy, and poor concentration. She had lost weight because of a reduction in appetite due to nonspecific abdominal pain. The pain was not altered by eating, movement, or related to having her bowels open. There was no associated vomiting or diarrhoea. She recently had to return early from holiday in Mexico because she felt so unwell. On examination, she was slim and tanned from her recent holiday. Her temperature was 36.9°C, pulse 89 beats per minute, blood pressure 90/60 mmHg, and respiratory rate 18 breaths per minute. Cardiovascular and respiratory examination was normal. Her abdomen was soft and non-tender.

Investigations:

Hb	128g/l (115-165)
WBC	10.2 * 10 /l (4.0-11.0)9

Na +	128mmol/l (137-144)
K +	5.6mmol/l (3.5-4.9)
Urea	10.8mmol/l (2.5-7.0)
Creatinine	98µmol/l (60-110)
Albumin	38g/l (37-49)
Alkaline phosphatase	126U/l (45-105)
ALT	112U/l (5-35)
Bilirubin	12µmol/l (1-22)
Alkaline phosphatase	126U/l (45-105)
Amylase	826U/l (60-180)
Ca +	2.84mmol/l (2.20-2.60)

What is the next most appropriate investigation for diagnosis to be made?

A. Dexamethasone suppression test
B. Short synacthen test
C. Protein electrophoresis
D. Thyroid function test
E. Urine and plasma osmolality

B. Short synacthen test

Primary hypoadrenalism is diagnosed by a short synacthen test and a failure to increase cortisol levels to above 500nmol/L

The scenario depicts primary hypoadrenalism. The classic electrolyte disturbance in primary hypoadrenalism (Addison's disease) is hyponatraemia (in about 90%) and hyperkalaemia (about 65%). Hyperkalaemia occurs because of the loss of aldosterone production. Hyponatraemia is partly due to sodium depletion but also vasopressin levels are elevated, resulting in an increase in free water retention. Reversible changes to liver enzymes may also occur. Hypercalcaemia (6%) may also occur. This is partly due to a reduction in calcium removal by the kidney and an increase in mobilization of calcium from bone stores. TSH can be elevated in primary hypoadrenalism due to a loss of feedback on the pituitary gland.

There are no features of hypercortisolism to necessitate a dexamethasone suppression test. Urine and plasma osmolalities maybe useful when evaluating hyponatraemia of unknown cause but on their own not useful in making specific diagnosis. Multiple myeloma is unlikely in the above scenario.

Question 12 of 348

A 28-year-old woman presents with daily intractable vomiting. She is 10 weeks pregnant with 3+ ketones in her urine. Initially, she tried cyclizine from her general practitioner with little success. You suspect hyperemesis gravidarum.

Apart from rehydration and anti-emetics, what also should be prescribed?

A. Thiamine
B. Carbohydrate replacement with 5% dextrose
C. Niacin
D. Vitamin B12
E. Folate

A. Thiamine

Nausea and vomiting are both common in pregnancy, affecting 50-80% of pregnant women. Hyperemesis gravidarum occurs in 0.3-3% of pregnancies. It is a diagnosis of exclusion and other causes of vomiting should be considered: urinary tract infection, endocrine causes (thyrotoxicosis, diabetic ketoacidosis etc), surgical causes and drugs such as iron supplements.

The Royal College of Obstetrics and Gynaecology (RCOG) guidelines state:

Thiamine supplementation (either oral or intravenous) should be given to all women admitted with prolonged vomiting, especially before administration of dextrose or parenteral nutrition.

A 5% dextrose intravenous infusion may precipitate Wernicke's encephalopathy. In addition, patients are frequently hyponatraemic and therefore sodium chloride 0.9% is most appropriate (+/- potassium replacement).

Folic acid (400 micrograms) daily is recommended for all women prior to conception and up to the 13th week of pregnancy. Women who are deemed higher risk of spina bifida are advised to take 5mg daily. This is to prevent neural tube defects but has no role in hyperemesis gravidarum.

Vitamin B12 deficiency is not of clinical concern in this scenario.

Niacin deficiency leads to pellagra. Hyperemesis gravidarum is a self-limiting condition associated with pregnancy and therefore niacin deficiency is not of clinical concern here.

Question 13 of 348

A 19-year-old man is reviewed prior to discharge. He presented with vomiting and abdominal pain and was found to have diabetic ketoacidosis. He was managed as an inpatient for five days before being well enough for discharge. He is also diagnosed with type 1 diabetes mellitus on this admission as a cause of the diabetic ketoacidosis. He has been educated by the diabetic nurse on how to manage his diabetes and insulin at home, but he is concerned about what his target plasma glucose should be after eating.

What is the recommended target after eating to be achieved by home monitoring?

A. 3-6mmol/litre
B. 5-10mmol/litre
C. 5-9mmol/litre
D. 7-12mmol/litre
E. 2-9 mmol/litre

C. 5-9mmol/litre

The correct answer is 5-9mmol/litre. NICE recommends that people with type 1 diabetes should aim for 5-7mmol/litre on waking, 4-7mmol/litre before meals and 5-9mmol/litre 90 minutes after eating. Frequent testing is very important in patients starting with insulin therapy to avoid both high and low sugar levels.

Question 14 of 348

A 65-year-old man visits his GP practice for review. He has a history of type 2 diabetes mellitus (T2DM) and hypertension.

His current medications are metformin 1 g twice daily and ramipril 10 mg once daily.

Na +	137 mmol/L	(135 - 145)
K +	4.8 mmol/L	(3.5 - 5.0)
Bicarbonate	24 mmol/L	(22 - 29)
Urea	8.2 mmol/L	(2.0 - 7.0)
Creatinine	152 µmol/L	(55 - 120)
Glucose	12 mmol/L	(4-7)
HbA1C	74 mmol/mol	(< 42)
Urine ACR	91 mg/mmol	

What additional medication should be started for this man?

A. Dapagliflozin
B. Gliclazide
C. Insulin
D. Pioglitazone
E. Semaglutide

A. Dapagliflozin

SGLT-2 inhibitors should be used in addition to metformin as initial therapy for T2DM if CVD, high-risk of CVD or chronic heart failure

This patient has poorly controlled type 2 diabetes mellitus (T2DM), possibly complicated by diabetic nephropathy, as indicated by elevated blood glucose levels, HbA1c, and urinary albumin-creatinine ratio. Given these factors, additional therapy for his T2DM is warranted. He has hypertension, so he is at high risk of cardiovascular disease; therefore, starting him on a sodium-glucose co-transporter 2 (SGLT-2) inhibitor, such as **dapagliflozin**, is advisable. NICE recommends offering SGLT-2 inhibitors in addition to metformin for patients with a high risk of cardiovascular disease, such as in this case.

NICE also recommends an SGLT2 inhibitor (in addition to the ARB or ACE inhibitor) if the urine albumin-creatinine ratio (ACR) is over 30 mg/mmol, such as in this case, and they meet the criteria in the marketing authorisation (including relevant estimated glomerular filtration rate [eGFR] thresholds). SGLT-2 inhibitors reduce the risk of CKD progression, mortality and cardiovascular events in adults with type 2 diabetes and CKD.

Gliclazide, a sulphonylurea, is an effective therapy for achieving glycaemic control in T2DM. However, sulphonylureas do not provide cardiovascular mortality benefits (unlike SGLT-2 inhibitors) and are weight-neutral (unlike metformin, which aids weight loss). They also carry a risk of hypoglycaemia.

As the patient is currently on only a single agent for T2DM, escalating to **insulin** therapy is not indicated at this stage. Insulin therapy would increase the risk of hypoglycaemia and induce weight gain. Given he is at high risk of cardiovascular disease, SGLT-2 inhibitors are recommended. Additionally, NICE advises trying dual oral diabetic medications before considering insulin initiation.

Pioglitazone, a PPAR-γ agonist that improves insulin sensitivity, can be considered if dual therapy with metformin and SGLT-2 is ineffective or contraindicated. However, as he is at high risk of cardiovascular disease (history of hypertension) and only takes metformin, SGLT-2 inhibitors are the preferred 2nd line treatment over pioglitazone.

GLP-1 agonists, such as **semaglutide**, are gaining recognition for their effectiveness in weight loss and have promising early results in reducing cardiovascular adverse events. GLP-1 agonists in T2DM are considered when triple therapy with metformin and two other oral antidiabetic drugs is ineffective, not tolerated, or contraindicated in the context of obesity when criteria are met. This patient is only in metformin, and it is early to consider GLP-1 in such a case.

Question 15 of 348

A 22-year-old woman presents with persistent nausea and vomiting. She is 16 weeks gestation. She has no significant past medical history and takes no regular medicines. Observations are within normal limits.

Blood results are as follows:

Hb	115 g/L	Male: (135-180)Female: (115 - 160)
Platelets	192 * 10 /L 9	(150 - 400)
WBC	10.2 * 10 /L 9	(4.0 - 11.0)
Na +	138 mmol/L	(135 - 145)
K +	3.5 mmol/L	(3.5 - 5.0)
Urea	4.2 mmol/L	(2.0 - 7.0)
Creatinine	56 µmol/L	(55 - 120)
CRP	2 mg/L	(< 5)

What treatment is indicated?

A. Cyclizine
B. Domperidone
C. Metoclopramide
D. Ondansetron
E. Pyridoxine

A. Cyclizine

Antihistamines are first-line in the management of nausea & vomiting in pregnancy/hyperemesis gravidarum

Cyclizine is correct. The royal college of Obstetrics and Gynaecology (RCOG) guidelines recommend antihistamines as the first-line agents for nausea and vomiting in pregnancy.

Domperidone is incorrect. This drug is a second-line agent. The risk of extrapyramidal side effects is less compared to metoclopramide.

Metoclopramide is incorrect. This drug is a second-line agent. Metoclopramide can cause extrapyramidal side effects, particularly in young females (<25 years old).

Ondansetron is incorrect. This drug is a second-line agent. The use of ondansetron during the first trimester is associated with a small increased risk of the baby having a cleft lip/palate.

Pyridoxine is incorrect. Pyridoxine can be used in conjunction with doxylamine, however, pyridoxine monotherapy is specifically not recommended in the RCOG guidelines.

Question 16 of 348

A 31-year-old woman is admitted with nausea and vomiting. She is 10 weeks into her first pregnancy. She reports vomiting multiple times daily and has been unable to tolerate oral anti-emetics or fluids for over 24 hours. You suspect hyperemesis gravidarum and commence intravenous fluids.

What treatment is recommended first-line?

A. Cyclizine
B. Haloperidol
C. Metoclopramide
D. Ondansetron
E. Pyridoxine

A. Cyclizine

Antihistamines are first-line in the management of nausea & vomiting in pregnancy/hyperemesis gravidarum

The correct answer is **cyclizine**. Anti-histamines such as cyclizine or promethazine are recommended first-line for hyperemesis gravidarum.

Haloperidol is incorrect. Haloperidol is an anti-psychotic but is associated with anti-emetic effects. However, it is not recommended in RCOG guidance. It has more of a role in managing nausea and vomiting in palliative care.

Metoclopramide is incorrect. This is a prokinetic agent with useful antiemetic properties. It carries risks of extrapyramidal side effects such as dystonia so isn't recommended first-line or for long courses.

Ondansetron is incorrect. This is an option if first-line treatments are not effective or not tolerated. However, there is a slightly increased risk of cleft lip if used in the first trimester. For this reason, it requires specific counselling and should not be used first-line.

Pyridoxine is incorrect. Pyridoxine has been used in hyperemesis in some cases, often in combination with doxylamine, particularly outside the UK. However, it is not yet recommended in RCOG guidelines.

Question 17 of 348

A 34-year-old man presents to the endocrine clinic for review. He was diagnosed with Addison's disease a year ago, and has found that his treatment of hydrocortisone dosing has restored him to normal function. He no longer feels tired, has regained the weight he lost before becoming unwell, and feels mentally much better. He is now planning a trip to South Africa and was advised to seek advise before travelling. He has already secured a good supply of his hydrocortisone and has obtained medical insurance.

What measures should be taken before he travels?

A. Advise that malaria prophylaxis is contraindicated whilst taking hydrocortisone
B. Issue supply of IM hydrocortisone
C. Advise against travelling to any areas with TB
D. Advise that he should not fly
E. Issue supply of prednisolone

C. Issue supply of IM hydrocortisone

Patients with Addison's should be given a hydrocortisone injection kit for adrenal crises

All patients with Addison's disease should have a supply of IM hydrocortisone and be advised to use this if unable to take oral hydrocortisone or vomiting. This can prevent Addisonian crisis and is especially important when the patient is travelling. It can also be important to supply the patient with the necessary documentation as they will be travelling with needles.

Hydrocortisone does not preclude anti-malarial treatment, and there is no reason that the patient would be unable to fly. It is important to advise the patient to visit a travel clinic to review the level of malaria in the area that they are travelling to, and also to review vaccination status and the need for additional vaccinations.

Question 18 of 348

A 34-year-old woman calls the endocrinology nurse for advice. She has been vomiting for the last 24 hours and has been unable to take her regular medications. She has a past medical history of Addison's disease. She normally takes oral hydrocortisone and fludrocortisone. Apart from vomiting and reduced oral intake she denies any symptoms. Specifically, she has not been dizzy on standing, had any blackouts or any diarrhoea. She has also been having a normal temperature. She has IM hydrocortisone at home.

What is the most appropriate advice to give in regards to her hydrocortisone?

A. Advise her to come to the emergency department
B. Advise her to take the IM hydrocortisone at come to the emergency department for review
C. Advise her to not take any hydrocortisone until the vomiting settles
D. Adviser her to take IM hydrocortisone only if starting to feel fainto r experiencing postural symptoms
E. Advise her to double the dose of oral hydrocortisone

A. Advise her to take the IM hydrocortisone at come to the emergency department for review

A person with Addisons' who vomits should take IM hydrocortisone until vomiting stops

This is a patient with Addison's disease who is unable to take her regular oral hydrocortisone and is therefore at risk of developing an Addisonian crisis. If unable to take the normal oral hydrocortisone then the patient should be advised to take IM hydrocortisone to avoid this. This is why all patients with Addison's disease should have IM hydrocortisone for these situations.

Given that she has been vomiting for the past 24 hours it is appropriate for her to attend the emergency department for assessment and possible IV fluids, IV hydrocortisone.

Question 19 of 348

A 28-year-old lady has noticed over the last year that she does not tolerate cold weather well. She is fatigued and her partner notices that she is also low in mood despite having no obvious triggers. Physical examination is unremarkable. Her electrocardiogram (ECG) demonstrates a sinus rhythm at 43 beats per minute. She has a background of type 1 diabetes mellitus for which she takes insulin. She also has coeliac disease. Her blood results are shown below:

Hb	136 g/l
MCV	103 fL
Na	133 mmol/l
K	4 mmol/l
Urea	3.5 mmol/l
Creatinine	70 µmol/l
Glycosylated haemoglobin (HbA1c)	51 mmol/mol (6.8%)
TSH	9.2 mIU/L (reference range 0.3-4.0 mIU/L
T3	2 pmol/L (reference range 3-9 pmol/L)
T4	5 pmol/L (reference range 9-25 pmol/L)

What is the next best step in her management?

A. Commence levothyroxine
B. Commence carbimazole
C. Ultrasound scan thyroid
D. MRI pituitary
E. Short synacthen test

E. Short synacthen test

The short synacthen test is the best test to diagnose Addison's disease

Every time you see type 1 diabetes, pernicious anaemia, rheumatoid arthritis, coeliac, or indeed ANY of the autoimmune conditions in the past medical history of a patient in the exam engage autoimmune mode in your brain. Once you have done that, you will be specifically looking not to miss other coexistent autoimmune conditions they may be trying to hide from you behind a cryptic clinical sign or some subtle blood abnormality. In this example, where she has type 1 diabetes, thinking like that then gives you the diagnosis of an autoimmune hypothyroid cause (Hashimoto's). If you think you have made a diagnosis of Hashimoto's, you should routinely next always look to rule out Addison's, even if the sodium is normal (which it isn't in this example: a subtle blood abnormality). Addison's (just like other autoimmune conditions) may coexist with Hashimoto's. However, the danger of missing Addison's in Hashimoto's is that it sits there masked by the hypothyroid. If you miss it and treat the hypothyroid blindly first without covering the Addison's, you will unmask the Addison's and the patient will come back in an adrenal crisis a few days later.

Question 20 of 348

A 48-year-old woman is referred to the endocrinology clinic with a 6- week history of weight gain and fatigue.

Her observations are as follows:

- Temperature 36.5ºC
- Heart rate 77 beats/min
- Blood pressure 171/97mmHg
- Respiratory rate 16 breaths/min
- Oxygen saturations 98% on air

Laboratory tests:

Hb	136 g/L	(115 - 160)
Platelets	288 * 10 /L 9	(150 - 400)
WBC	9.4 * 10 /L 9	(4.0 - 11.0)
Na +	142 mmol/L	(135 - 145)
K +	3.4 mmol/L	(3.5 - 5.0)
Urea	4.1 mmol/L	(2.0 - 7.0)
Creatinine	112 µmol/L	(55 - 120)
Glucose 16 mmol/L	(4 - 7)	

Low-dose dexamethasone suppression test:

Cortisol	250 nmol/L	(119 - 618)

What is the next best investigation?

A. High-dose dexamethasone suppression test
B. Insulin tolerance test
C. Petrosal sinus ACTH sampling
D. Pituitary MRI
E. Urinary cortisol

A. High-dose dexamethasone suppression test

The high-dose dexamethasone suppression test is useful for distinguishing between ACTH dependent (e.g. pituitary source) and non-ACTH dependent (e.g. ectopic and adrenal source) causes of Cushings's syndrome

This patient has Cushing's syndrome. Due to increased glucocorticoid production, symptoms include weight gain and hyperglycaemia as seen in this patient. Hypertension is another common finding also seen here. Laboratory tests may reveal hypokalaemic metabolic acidosis. The best test to diagnose Cushing's syndrome is the low-dose dexamethasone test. This has been done for this patient and shows a cortisol level that has not been suppressed despite exogenous steroid use. Once Cushing's syndrome has been confirmed, additional localisation tests need to be completed. One of these includes the high-dose dexamethasone suppression test which is used to discriminate between Cushing's disease (i.e. pituitary adenoma) and ectopic ACTH production. Suppressed cortisol levels following high doses of glucocorticoids confirms a pituitary cause, as opposed to normal levels of cortisol that suggest an adrenal cause.

The insulin stress test is used to differentiate Cushing's syndrome from pseudo-Cushing's. Pseudo-Cushing's presents similarly to Cushing's syndrome; however, rather than being caused by excessive corticosteroid levels, it is due to alcohol excess or severe depression. There is nothing in this patient's history to suggest depression or alcohol excess. Furthermore, the positive finding following the low-dose dexamethasone suppression test points away from a diagnosis of pseudo-Cushing's.

Petrosal sinus ACTH sampling is another investigation that can be used to help distinguish between a pituitary or adrenal cause of Cushing's syndrome. However, it is an invasive investigation and easier tests such as the high-dose dexamethasone suppression test should be considered first.

A pituitary MRI can be used to diagnose a pituitary cause of Cushing's syndrome. However, it is not the next recommended investigation. Rather, a high-dose dexamethasone suppression test is easier and cheaper to complete and should be completed next.

Urinary cortisol is used as an initial investigation in the diagnosis of Cushing's syndrome. It is not a specific localisation test.

Question 21 of 348

A 45-year-old man presents to the endocrine clinic for review. He has had 3 stones in weight gain over the past 6 months and his GP is concerned about a possible diagnosis of Cushing's syndrome and has checked an initial 24hr urinary free cortisol which is elevated. He is hypertensive with a blood pressure of 155/90 mmHg, his pulse is 75 beats per minute and regular. His body mass index is 35 kg/m² and there are obvious abdominal striae.

Which of the following would be most suggestive of an adrenal adenoma producing cortisol?

A. Normal 9am serum cortisol
B. Raised urinary free cortisol on repeat testing
C. Serum cortisol of 220 mmol/l at 9am after an overnightdexamethasone suppression test
D. Serum potassium of 2.4 mmol/l
E. Undetectable levels of ACTH

E. Undetectable levels of ACTH

The key fact is that an adrenal adenoma producing cortisol would suppress the release of ACTH. For this reason, if ACTH is undetectable then an adrenal tumour is the most likely cause. In this case, a CT abdomen would be the obvious next step.

Normal 9 am serum cortisol would make a diagnosis of Cushing's less likely, and a raised urinary free cortisol on repeat testing is not specific for the underlying cause. Failure to suppress after overnight dexamethasone suppression test merely confirms a diagnosis of Cushing's. Serum potassium levels as low as 2.4 mmol/l would be very unusual in a case of Cushing's.

Question 22 of 348

A 34-year-old man of ethnic Indian origin is reviewed in endocrinology clinic. He has type 1 diabetes. He has a twice-daily mixed insulin regime but has poor diabetic control with elevated HbA1c and high blood glucose. He wants to improve his diabetic control but is concerned about increasing his insulin dose or frequency as he is already overweight with a body mass index (BMI) of 29kg/m .

Apart from increasing insulin, are there any other medical management options to better control his diabetes?

A. No further medical treatment
B. Metformin
C. Gliclazide
D. Acarbose
E. Pioglitazone

B. Metformin

The correct answer is metformin. This is a patient with poorly controlled type 1 diabetes. Of importance to this case, he is also overweight and is of ethnic Indian origin. Increasing insulin doses is seldom popular due to the side effects. NICE recommends that in overweight people of Indian origin metformin is an alternative to increasing insulin and may be helpful, especially as it would not lead to further weight increase.

Question 23 of 348

A 34-year-old woman is seen by her GP for the annual review of her type 1 diabetes. Her most recent HbA1c is 58 mmol/mol and the only problem she has noticed are severe hypoglycaemic episodes during the night around 2-4am, that she noticed she was getting while working night shifts for a local superstore. However, by breakfast time, her blood glucose levels often rise to around 15 mmol/mol. Her insulin regimen is currently a twice daily mixed insulin.

What is the most appropriate change to her current insulin treatment?

A. Move to a basal bolus of insulin
B. Take 1 dextrose tablet at 9pm
C. Reduce nocturnal insulin dose
D. Reduce morning insulin dose
E. Add gliclazide

A. Move to a basal bolus of insulin

This patient is experiencing severe nocturnal hypoglycaemic episodes followed by rises in blood glucose by breakfast time. The most appropriate change to the insulin treatment regimen is changing to a basal bolus regime, with one daily injection of background long-acting insulin and three short acting injections of insulin.

Question 24 of 348

You are asked to review a 62-year-old Caucasian man who is an inpatient on the medical admissions unit. He is currently being treated for a left lower lobe community acquired pneumonia. You note consumed alcohol excessively prior to admission but has been abstinent for the last four days.

During this admission it has been noted that serial bloods glucose measurements have been elevated and subsequently a new diagnosis type two diabetes has been made. The admission consultant noted Cushingoid featured and requested an overnight low dose dexamethasone suppression test. The results are as follows:

8am Cortisol after 1mg dexamethasone at 11pm the previous day	438 nmol/L
Reference range for serum cortisol	170-540 nmol/L

What is most appropriate next step in the investigation of this gentleman?

A. Serum ACTH measurement
B. Midnight serum cortisol
C. Inferior petrosal sinus sampling post CRH administration
D. High dose dexamethasone suppression test
E. MRI pituitary

B. Midnight serum cortisol

Pseudo-Cushing's syndrome is common in those with excessive alcohol consumption. The physical signs resemble true Cushing's syndrome however the aetiology is idiopathic rather than dysfunction of the hypothalamic-pituitary axis. It is therefore important to exclude pseudo Cushing's prior to further investigation.

The hallmark of true Cushing's syndrome is lack of diurnal variation in serum cortisol. However, in pseudo-Cushing's diurnal variation is normally maintained. Those with pseudo-Cushing's will have elevated 24 hour urinary cortisol and will also fail to suppress serum cortisol with a low dose dexamethasone suppression test. The correct approach in this case is therefore to measure a midnight cortisol prior to proceeding to further investigation.

Question 25 of 348

A 53-year-old female presents with 48 hours of general malaise. 30 years ago, she underwent a resection of a pituitary mass and has since been compliant on desmopressin, levothyroxine and hydrocortisone, up until her last dose earlier in the morning. She has no other past medical history. Her husband reports the patient to have had reduced oral intake for the past 2 days while she has been unwell. She has no reported head injuries, rigors or pyrexia. On examination, her GCS is E3 V2 M5. She is cool peripherally and a temperature demonstrates 33.4 degrees under her tongue. Her spot blood glucose is 2.2 mmol/l. Her blood pressure is 86/50 mmhg heart rate 110/min and sinus rhythm. Blood tests demonstrate a sodium of 158 mmol/l and potassium of 4.2 mmol/l.

What is your first action(s)?

A. Send thyroid function test
B. Administer IV liothyronine
C. Send random cortisol
D. Administer IV hydrocortisone
E. CT head, blood culture, urine dip, HbA1c

D. Administer IV hydrocortisone

In the context of a sudden decline in any patient with long-term steroid use with hypotension, hypothermia and hypoglycaemia, you must treat presumptively for an Addisonian crisis. It must be remembered while the classic biochemical presentation of hyponatraemia and hyperkalaemia is frequently quoted in textbooks, this is frequently not the case in real life. While she may indeed be thyroid deficient, neither thyroid function tests nor intravenous free T3 is the most immediate treatment. A random cortisol, in the context of recent hydrocortisone in the community, is uninterpretable.

Question 26 of 348

A 32-year-old patient is due to be discharged from hospital. He has had his third admission for DKA in the last two years since being diagnosed with type 1 diabetes mellitus. He has come off his fixed-rate IV insulin and started to eat and drink again, and has restarted his long-acting insulin detemir (Levemir) and short-acting insulin aspart (Novorapid) as his pre-admission plan. His DKA was most likely provoked by the patient drinking alcohol and not taking his insulin. This is similar to previous admissions, but he only drinks two bottles of beer a week, but occasionally binge drinks. He has no other co-morbidities and takes no other medications.

What is the most appropriate suggestion to avoid further admissions with DKA?

A. Start metformin
B. Change insulin detemir (Levemir) to insulin degludec (Tresiba)
C. Change insulin detemir (Levemir) to insulin aspart biphasic(Novomix 30)
D. Increase dose in insulin aspart (Novorapid) by 20%
E. Advise reduction in carbohydrates in diet

B. Change insulin detemir (Levemir) to insulin degludec (Tresiba)

A patient with recurrent admissions for DKA can be started on degludec to reduce readmission rate

This is a patient with type 1 diabetes with recurrent DKA secondary to a combination of missing doses of insulin and binge drinking alcohol. The most appropriate and likely to be successful strategy is to change Levemir to Degludec. Degludec has a much higher half-life than Levemir and therefore maintains a basal insulin level when the patient omits or forgets doses. This can prevent DKA. Metformin is not indicated for this patient. Insulin aspart biphasic (Novomix 30) would allow reduction in the number of insulin injections. Increasing the dose of insulin aspart (Novorapid) is unlikely to prvent DKA for this patient as the fundamental issue is missing doses, and the short-term nature of the insulin aspart (Novorapid) is unlikely to provide any insulin cover for further omitted doses. Patients with type 1 diabetes can also benefit from DAFNE, a course which helps patients adjust their insulin doses to what they are eating and the patient should be offered this as an educational tool as well to improve control.

Question 27 of 348

An 18-year-old man presents to the clinic with poor development of secondary sexual characteristics. On examination, you note a lack of testicle development and sparse axillary and pubic hair.

Blood results are as follows:

Testosterone	2 nmol/L	(6 -27)
FSH	4.6 IU/L	(1.8 - 22.5)
LH	2.8 IU/L	(1.2 - 103)

What is the most likely diagnosis?

A. Androgen insensitivity syndrome
B. Kallmann's syndrome
C. Klinefelter syndrome
D. Mullerian agenesis
E. Turner's syndrome

B. Kallmann's syndrome

Kallman's syndrome - LH & FSH low-normal and testosterone is low

Kallmann's syndrome is correct. Kallmann's syndrome is a recognised cause of delayed puberty secondary to hypogonadotropic hypogonadism. The biochemical profile is in keeping with hypogonadotropic hypogonadism due to the presence of low testosterone and inappropriately normal LH and FSH. Kallmann's syndrome is the only listed condition which results in hypogonadotropic hypogonadism.

Androgen insensitivity syndrome is correct. Androgen insensitivity syndrome in its complete form is a disorder of hormone resistance characterised by a female phenotype in a genetic male due to the resistance to the effector functions of testosterone. The hormonal profile findings include very high testosterone, elevated LH, and a relatively normal FSH. Thus the patient's phenotypical sex and hormonal profile exclude this diagnosis.

Klinefelter syndrome is incorrect. Klinefelter syndrome is an aneuploid genetic condition where a male has an additional copy of the X chromosome. The primary features are infertility and small, poorly functioning testicles. This condition results in primary gonadal failure, and thus a compensatory elevation in LH and FSH.

Mullerian agenesis is incorrect. Mullerian agenesis is a congenital malformation characterised by a failure of the Mullerian ducts to develop, resulting in a missing uterus and variable degrees of vaginal hypoplasia of its upper portion. Given that the patient is phenotypically male, this diagnosis is unlikely.

Turner's syndrome is incorrect. Turner's syndrome can cause primary ovarian failure due to gonadal dysgenesis. This would present with low sex hormone levels, and markedly raised LH and FSH levels. It only affects females. Thus the patient's phenotypic sex, and biochemical results are not in keeping with this diagnosis.

Question 28 of 348

A 35-year-old woman presents to the emergency department with headaches. These have been happening intermittently for the last 3 months but this one is particularly painful. They are associated with sweating and palpitations. She has found that headaches are often triggered after drinking coffee.

Neurological examination is normal. Observations are heart rate 119/min, blood pressure 186/108mmHg, respiratory rate 18/min, oxygen saturation 99%, temperature 37.3ºC.

What is the most appropriate management?

A. Glyceryl trinitrate intravenous infusion
B. Labetalol intravenous bolus injection
C. Labetalol intravenous infusion
D. Phenoxybenzamine intravenous infusion
E. Phenoxybenzamine orally

E. Phenoxybenzamine orally

PHaeochromocytoma - give PHenoxybenzamine before betablockers

The combination of headache, tachycardia, and diaphoresis alongside significant hypertension is suggestive of phaeochromocytoma. This is additionally supported by the intermittent nature of the symptoms. There are several recognised triggers for 'attacks', including dietary factors such as cheese, tomatoes, and coffee.

Both alpha-blockers and beta-blockers are used in the management of phaeochromocytoma. Alpha-blockers oppose catecholamine-induced vasoconstriction. An alpha-blockade must be performed first with the alpha-blocker phenoxybenzamine. This medication only has oral forms available and licenced in the BNF and therefore **phenoxybenzamine orally** is the correct answer. If the beta-receptors are blocked first this leads to a loss of beta-mediated vasodilation and the subsequent unopposed alpha-adrenergic receptor stimulation can precipitate extreme hypertension.

A **glyceryl trinitrate intravenous infusion** can be used for treating significant hypertension. In phaeochromocytoma, hypertension is primarily driven by the activation of the sympathetic nervous system through the release of catecholamines acting on alpha receptors. Therefore, the most appropriate and effective way to counter this is through an alpha-blocker.

Labetalol is a beta-blocker. Although beta-blockers play a role in the management of phaeochromocytoma, it is important to give alpha-blockers first as mentioned above. Therefore, both **labetalol intravenous bolus injection** and **labetalol intravenous infusion** are incorrect.

Phenoxybenzamine intravenous infusion is incorrect, only oral forms of this medication are available and licenced in the BNF.

Question 29 of 348

A 45-year-old woman presents to the emergency department feeling very hot, weight loss and nausea. She has a past medical history of cardiomyopathy for which she an ICD inserted and takes amiodarone due to recurrent episodes of ventricular tachycardia. Blood tests show an undetectable thyroid stimulating hormone. She also has rheumatoid arthritis. She has previously had an angiogram showing no evidence of coronary artery disease. She takes methotrexate, aspirin, paracetamol, omeprazole, warfarin and bisoprolol. She is suspected of having acute thyrotoxicosis.

What is the most appropriate action in regards to her current medication in addition to stopping amiodarone?

A. Stop aspirin
B. Stop omeprazole
C. Stop bisoprolol
D. Stop warfarin
E. Stop paracetamol

A. Stop aspirin

In acute thyrotoxicosis, stop aspirin as it can worsen the storm by displacing T4 from thyroid binding globulin

This patient with acute thyrotoxicosis should have amiodarone, the likely cause, of her hyperthyroidism stopped. In addition, aspirin should be stopped. Aspirin binds to thyroxine-binding globulin and displaces bound T4, thereby increasing the levels of free T4.

Question 30 of 348

A 54-year-old woman is referred to cardiology clinic due to echocardiogram findings consistent with cardiomyopathy. She underwent the echocardiogram after developing shortness of breath and there were concerns about underlying heart failure. She has a past medical history of hypertension, palpitations and anxiety. She has been troubled by tremors and increasing weight loss, sweating and heat intolerance over the last three months and has been diagnosed with hyperthyroidism and started on carbimazole.

What is the likely outcome of her cardiac condition?

A. Likely to develop permanent heart failure
B. Likely to develop pulmonary hypertension
C. Likely to develop aortic regurgitation
D. Likely to have resolution of symptoms but not of cardiomyopathy
E. Likely to have resolution of symptoms and of cardiomyopathy

E. Likely to have resolution of symptoms and of cardiomyopathy

Thyrotoxicosis is associated with reversible cardiomyopathy

The patient has thyrotoxicosis. The hyperthyroidism can cause cardiac complication by a rate-related mechanism. Due to this, patients can develop tachycardia, palpitations, AF and rate-related cardiac failure, as well as cardiomyopathy. All of these problems are likely to resolve when the thyrotoxicosis resolves, and this patient has been diagnosed and treatment has been started with carbimazole.

Question 31 of 348

A 35-year-old female presents with weight loss and palpitations and fatigue. Thyroid function tests were performed as part of a workup for her symptoms:

TSH	<0.01mU/L	0.4-4.0mU/L
T4	15.3pmol/L	9.0-25.0pmol/L

What is the next most appropriate investigation to perform in this patient?

A. Urinary pregnancy test
B. Plasma metanephrines
C. T3 levels
D. Thyroid antibodies
E. 24 hour ECG

C. T3 levels

T3 levels should be performed where tests show normal T4 with suppressed TSH

T3 thyrotoxicosis should always be considered in patients with suppressed TSH and normal T4 levels, especially when patients are symptomatic.

Pregnancy can cause raised total thyroxine but normal free T4 and T3 and would not suppress TSH. Thyroid antibodies would be useful to consider once the diagnosis has been confirmed. Plasma metanephrines should be performed to investigate if an underlying pheochromocytoma is suspected and ECG may be indicated to investigate palpitations but would not explain the whole presentation.

Question 32 of 348

A 54-year-old man presented to endocrine clinic with gradually worsening low mood, malaise, and reduced exercise capacity. . He was operated on previously for a non-functioning pituitary adenoma (NFPA) resulting in partial anterior hypopituitarism, and had been on thyroxine and hydrocortisone replacement therapy. On examination, he had evidence of central adiposity. He was known to have dyslipidaemia, epilepsy and ischaemic heart disease (IHD) and so an insulin tolerance test was contraindicated.

Investigations

9a.m cortisol	415nmol/l
IGF-1	8nmol/l (16 - 118)
FT4	15.4nmol/l (11.5 - 22.7)
TSH	0.03mU/l (0.35 - 5.5)

Which one of the following tests is most appropriate in his case to confirm the diagnosis of adult GH deficiency?

A. Domperidone test
B. Growth hormone levels
C. Growth hormone releasing hormone (GHRH)-arginine stimulation test
D. Insulin-like growth factor- binding protein measurement
E. Glucose tolerance test

C. Growth hormone releasing hormone (GHRH)-arginine stimulation test

Arginine-GHRH stimulation test is employed to assess GH secretion in patients with ischemic heart disease or seizures where insulin tolerance test will be inappropriate

The clinical features of growth hormone deficiency (GHD) are nonspecific and include lethargy, low mood, poor quality of life, loss of muscle mass, and central adiposity. A low IGF-1 in such a clinical context may point towards GHD, which needs to be confirmed with dynamic tests for GH secretion. Insulin tolerance test (ITT) is considered the gold standard for assessing GH secretion, although it needs to be conducted carefully in a closely-monitored space due to the risks associated with hypoglycaemia.

ITT is contra-indicated in patients with seizures or ischaemic heart disease. As a result, an alternative test such as arginine-GHRH stimulation test is employed (as in this clinical scenario where the patient has IHD). About 30-40% of patients with GHD may have a normal IGF-1 level. IGF-1 levels are influenced by age, time of onset of GHD, and degree of hypopituitarism. Domperidone test is used in the evaluation of hyperprolactinemia and macroprolactinaemia associated disorders while glucose tolerance test with measurement of IGF-1 levels is used in the evaluation of suspected acromegaly.

Question 33 of 348

A 16-year-old male attends the clinic with steatorrhoea and poor growth. He has a family history of abetalipoproteinemia. He is on no regular medications. On examination you note ataxia and reduced visual acuity (6/12 in the right eye and 6/15 in the left eye).

Investigations are as follows:

Hb	110 g/l	Na +	136 mmol/l
Platelets	380 * 10 /l 9	K +	3.9 mmol/l
WBC	10.1 * 10 /l 9	Urea	6.6 mmol/l
Neuts	6.8 * 10 /l 9	Creatinine	48 µmol/l
Blood film	Acanthocytosis		

What management will you advise?

A. Reassurance
B. Simvastatin
C. Dietary restriction of fats
D. Ezetimibe
E. Increase fat intake

C. Dietary restriction of fats

Treatment of abetalipoproteinemia involves dietary restriction of fats, and high-dose vitamin E therapy

Abetalipoproteinemia is a rare genetic condition caused by a mutation in the microsomal triglyceride transfer protein resulting in deficiencies in apolipoproteins. Apolipoproteins are essential in the synthesis and exportation of chylomicrons and VLDL. The end results is malabsorption of dietary fats, cholesterol, and fat soluble vitamin (e.g. vitamins K, A, D and E).

Clinical features include steatorrhoea, poor growth, neurological dysfunction, and impaired vision.

Treatment is with dietary restriction of fats, and high-dose vitamin E therapy.

Question 34 of 348

A 28-year-old man presents to the clinic for review of his familial hypercholesterolaemia. He is currently on 80mg once daily atorvastatin.

His blood results are as follows:

Hb	135 g/l	Na +	138 mmol/l
Platelets	322 * 10 /l 9	K +	4.6 mmol/l
WBC	10.5 * 10 /l 9	Urea	6.6 mmol/l
Neuts	6.2 * 10 /l 9	Creatinine	72 µmol/l
Total cholesterol	7.5 mmol/L (normal range < 5)		
LDL cholesterol	5.5 mmol/L (normal range < 3)		

What treatment would you begin?

A. Fenofibrate
B. Nicotinic acid
C. Ezetimibe
D. Evolocumab
E. Cholestyramine

C. Ezetimibe

First line management for familial hypercholesterolaemia is high dose statins. Ezetimibe is used as a second line agent

High dose statin therapy is first line for familial hypercholesterolaemia. If serum total or low-density lipoprotein (LDL) cholesterol concentration is not appropriately controlled on statin therapy, then the second line agent ezetimibe can be co-administered with the statin. Ezetimibe acts by decreasing cholesterol absorption in the small intestine.

Question 35 of 348

A 47-year-old woman with a history of paroxysmal atrial fibrillation undergoes a thyroidectomy for Graves' disease. Initially, her symptoms settle. However, on postoperative scans, residual thyroid tissue is noted and after several months, her symptoms recur, including palpitations from atrial fibrillation. She is referred for radioiodine therapy.

Three days before the radioiodine administration is due, she is admitted with acute-onset abdominal pain and, given the history of atrial fibrillation, ischaemic colitis is suspected. An urgent CT scan of the abdomen is performed with contrast, which rules out any acute pathology and so she is discharged.

With regards to the radioiodine therapy, when is the earliest that this can take place?

A. 3 days
B. 2 weeks
C. 8 weeks
D. 16 weeks
E. 26 weeks

C. 8 weeks

Radioiodine therapy should be avoided until 8 weeks following CT contrast administration

Iodine in intravenous contrast interferes with the administration of radioiodine. As the levels of iodine in contrast are several hundred-fold higher than the recommended daily allowance, this may be retained by the body for months. As such, it can interfere with therapeutic uptake of radioiodine by the thyroid gland. Various studies have demonstrated a 6- 8 week time period is the minimum to wait before administering radioiodine, following CT contrast. **8 weeks** is therefore the correct answer.

Going ahead with the procedure as planned, **3 days** after this CT scan with contrast is therefore too soon. The contrast would interfere with the radioiodine.

Similarly, **2 weeks** is still too soon to go ahead with the administration.

16 weeks is an unnecessarily long amount of time to wait - 8 weeks is sufficient according to current studies.

Similarly, **26 weeks** is longer than needed - the question asks what the earliest time of administration should be.

Question 36 of 348

A 42-year-old woman presents with a three-week history of neck pain and swelling that worsens upon swallowing. She also reports fatigue, tremors, nervousness, occasional palpitations, and unintentional weight loss. Examination reveals blood pressure of 125/80 mmHg, heart rate of 104 beats per minute, and temperature of 37.8 ºC. The thyroid gland is tender on palpation and enlarged without any palpable nodules. Laboratory results are as follows:

TSH	0.1 mIU/L ng/mL	(0.4 - 4.0)
Free T4	8 ng/dL	(0.7 - 1.9)
ESR	101 mm/hr	

Anti-thyroglobulin and anti-thyroperoxidase antibodies are mildly elevated, while radioactive iodine uptake is low.

What is the most likely diagnosis?

A. Acute infectious thyroiditis
B. Graves disease
C. Hashimoto thyroiditis
D. Solitary toxic nodule
E. Subacute granulomatous thyroiditis

E. Subacute granulomatous thyroiditis

Thyrotoxicosis with tender goitre = subacute (De Quervain's) thyroiditis

Subacute granulomatous thyroiditis, also known as de Quervain's thyroiditis, is the correct diagnosis. The patient presents with clinical and biochemical signs of thyrotoxicosis, as indicated by tachycardia, tremors, nervousness, low-grade fever, fatigue, weight loss, palpitations, suppressed TSH, and elevated free T4 levels. These symptoms, in conjunction with an enlarged tender thyroid gland and high ESR level, are indicative of de Quervain's thyroiditis. Typically following a viral infection (not specified in this scenario), this condition is characterised by an initial phase of thyrotoxicosis followed by hypothyroidism. Management often involves symptomatic relief with non-steroidal antiinflammatory drugs (NSAIDs) and beta-blockers.

Acute infectious thyroiditis, or suppurative thyroiditis, is a rare and potentially serious condition usually seen in the elderly and immunocompromised individuals. Gram-positive bacterial infections mainly cause it. Symptoms include sudden-onset fever, chills, malaise, a tender, painful thyroid gland, and signs of hyperthyroidism. In severe cases, the infection causes compression on adjacent structures, leading to dysphagia and hoarseness of voice. It is important to note that de Quervain's thyroiditis has a gradual onset of symptoms and does not cause compression symptoms, making acute infectious thyroiditis less likely.

Graves' disease is not the appropriate diagnosis here. Despite the biochemical and clinical features of thyrotoxicosis, the absence of Graves' ophthalmopathy and dermopathy, specific anti-thyroid stimulating hormone receptor antibodies, and a low radioactive iodine uptake are inconsistent with this condition.

Hashimoto's thyroiditis is a relevant differential diagnosis; however, it typically presents with a painless goitre. In de Quervain's thyroiditis, mild elevations in anti-thyroglobulin and anti-thyroperoxidase antibodies may be observed in fewer than 25% of cases. By contrast, these antibody levels are significantly higher in Hashimoto's thyroiditis.

Solitary toxic nodule is not consistent with this case presentation. Such nodules are generally single, palpable and non-tender associated with thyrotoxicosis. In contrast to this condition, the thyroid gland is diffusely enlarged and tender with no palpable; the findings align with de Quervain's thyroiditis rather than a solitary toxic nodule.

Question 37 of 348

A 35-year-old makes an urgent appointment in the diabetes clinic. She has had type 1 diabetes since the age of 20 but has controlled this well with insulin. She has no other medical problems and since diagnosed during a hospital admission with diabetic ketoacidosis has had no further hospital admissions or problems. She is concerned because she has fallen pregnant and wants to makes sure that she is not doing anything to harm the foetus. She is not looking to have a termination. She found out after she missed her period one week ago and took two pregnancy tests which were both positive. She had been taking a basal-bolus regime of insulin as well as atorvastatin. She had retinal screening and urine testing nine months ago. Her HbA1c was last measured three months ago at 39mmol/mol. She has been advised to continue with her insulin. She has also stopped the atorvastatin due to concerns about its effects in pregnancy.

What is the most appropriate action to avoid complications of diabetes?

A. Restart atorvastatin
B. Increase short-acting insulin dose
C. Advise termination of pregnancy
D. Refer for retinal screening
E. Increase basal insulin dose

D. Refer for retinal screening

Patients with diabetes should have increased frequency of retinal screening during pregnancy due to increased risk of retinopathy

The most appropriate plan is to repeat the retinal screening and test the urine. In diabetes, patients should achieve good diabetic control prior to planning for pregnancy. If this has not been achieved, then NICE advises contraception and to offer termination if pregnancy does occur due to increased risks in pregnancy. This patient has good diabetic control as evidenced by the lack of complications and HbA1c.

When anticipating pregnancy, contraindicated drugs such as atorvastatin and ACE inhibitors should be stopped. Labetalol is an appropriate option for blood pressure control. The dose of insulin does not automatically need to be adjusted up or down but blood glucose would need close monitoring during pregnancy. It is advised, however, if the patient has not had retinal screening within the last six months to offer this urgently as there can be rapid development of diabetic retinopathy in pregnancy.

Question 38 of 348

A 45-year-old man presents to review to the endocrinology clinic. He has been suffering from erectile dysfunction for six months and seen his GP for this. He has had serum testosterone tested and this was found to be low. This is confirmed on repeat testing. LH and FSH are found to be within normal range. The patient describes erectile dysfunction as well as loss of nocturnal erections. He describes increased stress at work due to a recent promotion. He also feels that the erectile dysfunction has put a strain on his marriage.

He also has been suffering from worsening headaches, occurring at night, loss of energy and loss of sexual desire.

What is the most likely aetiology for his erectile dysfunction?

A. Psychological
B. Acquired primary hypogonadism
C. Genetic primary hypogonadism
D. Secondary hypogonadism
E. Androgen insensitivity syndrome

D. Secondary hypogonadism

In male patients with low libido have been found to have a low testosterone first line investigation should include prolactin and LH to assess for a central cause

The patient describes symptoms of hypogonadism with loss of sexual desire, erectile dysfunction and low energy. This is supported by low morning testosterone. This combination makes a psychological cause unlikely. As the LH and FSH are not elevated a primary hypogonadism is excluded. In either acquired or genetic primary hypogonadism there would be increased LH and FSH due to loss of negative feedback. In secondary hypogonadism, LH and FSH are low or normal with low testosterone. Potential cause include increased prolactin, which can be by tumour or drug-induced. Prolactin reduces LH and FSH and can therefore be a cause. Other potential secondary causes of hypogonadism include hypopituitarism, Kallmann's syndrome and isolated hypogonadotrophic hypogonadism. Androgen insensitivity syndrome is associated with elevated testosterone.

Question 39 of 348

A 18-year-old man with type 1 diabetes and poor compliance attends hospital with shortness of breath, vomiting and feeling unwell. He is found to be in diabetic ketoacidosis and transferred to the high dependency unit. He has recovered well and is asymptomatic for a period but on day 3 of his treatment starts to feel unwell. He is tired and lethargic. His arms and legs ache and there is cramping at times. He walks to the bathroom and his legs collapse beneath him.

On examination, he is alert and has moist mucosa. There are no fasciculations or myoclonus. He has 4/5 power in all muscle groups with retained sensation. His abdomen is soft and his chest clear. A set of observations show that he is tachypnoeic at 24 breaths/min.

Bloods	admission	Now
Na^+	128 mmol/l	133 mmol/l
K^+	6.1 mmol/l	4.5 mmol/l
Urea	9.2 mmol/l	5.6 mmol/l
Creatinine	134 µmol/l	87 µmol/l
Glucose	27.1mmol/l	12mmol/l
Ketones	3.1 mmol/l	0.2 mmol/l
pH	7.01	7.35

What is the likely course of his deterioration?

A. Anxiety
B. Hypophosphataemia
C. Cerebral pontine myelinolysis
D. Sepsis
E. Disequilibration syndrome

B. Hypophosphataemia

Recovering DKA are at risk of hypophosphataemia

This gentleman has developed weakness following treatment for DKA. This is unlikely cerebral pontine myelinolysis as the sodium has been corrected by only 5mmol/l in 2-3 days. There are no focal signs suggesting sepsis though it is likely on the differential. Anxiety should only be made once other diagnoses are excluded. Disequilibration syndrome occurs in dialysis patients and is not relevant here. Therefore the answer is hypophosphataemia that often arises as a side effect of insulin with cells forming ATP and taking up free phosphate to achieve this.

Question 40 of 348

A 40-year-old woman attends the endocrinology clinic with a 4-week history of intermittent headaches and milky discharge from her nipples. Over the last 5 months, she also reports absent menstrual periods despite usually having a regular 28-day cycle. There is no past medical history of note and she has no allergies.

Laboratory tests:

Thyroid-stimulating hormone (TSH)	0.5 mU/L	(0.5-5.5)
Free T4	7.5 pmol/L	(9.0 - 18)
Adrenocorticotrophic hormone	6 ng/L	(0 - 47)
IGF-1	10 nmol/L	(12.4 - 30.3)
Prolactin	6000 IU/mL	(59 - 619)
β-HCG	< 1 u/L	(< 1)

MRI head: there is a visible pituitary adenoma of approximately 1.5cm in size causing compression of the optic chiasm.

Given the likely diagnosis, what is the most appropriate next step in the management of this patient?

A. Bromocriptine
B. Dexamethasone
C. Domperidone
D. Stereotactic radiotherapy
E. Trans-sphenoidal surgery

A. Bromocriptine

Dopamine agonists (e.g. cabergoline, bromocriptine) are first-line treatment for prolactinomas, even if there are significant neurological complications

Given the history of amenorrhoea and galactorrhoea and the blood results demonstrating an elevated prolactin level with low-normal thyroid, IGF-1 and ACTH levels, this patient has a diagnosis of prolactinoma. This is confirmed by the presence of a pituitary mass of 1.5cm in size that classifies this growth as a macroadenoma. The first-line treatment for all prolactinomas (regardless of size) is the use of dopamine agonists such as bromocriptine. Dopamine suppresses the production of prolactin from the anterior pituitary gland and thus helps improve this patient's symptoms.

Dexamethasone is commonly indicated in patients with evidence of increased intracranial pressure or space-occupying lesions. Although this patient has a significant pituitary adenoma, evidence of compression of the optic chiasm and neurological symptoms including headaches, dexamethasone is not first-line in the treatment of prolactinomas. Rather, dopamine agonists are preferred.

Domperidone is a dopamine antagonist and would be contraindicated in this patient given that dopamine suppresses prolactin production and further inhibition could drive the growth of the prolactinoma.

Stereotactic radiotherapy is less popular in the treatment of such small lesions, given the risk of damage to adjacent structures and would therefore not be a first-line treatment for this patient.

Trans-sphenoidal surgery is an alternative treatment option for prolactinomas. However, this is reserved if medical treatment with dopamine agonists fails. As dopamine agonists have not yet been initiated for this patient, bromocriptine would be the correct answer.

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A 40-year-old school teacher presented to the medical assessment unit with a 2-month history of polyuria and elevated blood glucose levels. There was no family history of diabetes and she was not currently on any medication. On examination, her body mass index was 22 kg/m with normal general physical and systemic examination. Urine was negative for ketones.

Blood glucose	16.5 mmol/l
pH	7.40
HCO_3	25 mmol/l
Na^+	140 mmol/l
K^+	3.7 mmol/l

Which one of the following test may be useful in establishing the underlying diagnosis considering her clinical profile?

A. Oral glucose tolerance test
B. Serum ferritin and total iron binding capacity
C. Mitochondrial gene mutation (A3243G)
D. Anti-GAD antibody
E. Toxicology screen

D. Anti-GAD antibody

Latent autoimmune diabetes of adulthood is a subtype of diabetes in which patients may present with phenotypic features of T2DM, while displaying the presence of markers of autoimmunity (antiGAD antibodies)

Latent autoimmune diabetes of adulthood is a subtype of diabetes in which patients may present with phenotypic features of type 2 DM, while displaying the presence of markers of autoimmunity (anti-GAD antibodies). Most of these patients are in the age group of 30-50 years, may or may not have a normal BMI and may present with elevated blood glucose levels. These patients can be managed initially with oral hypoglycaemic agents, although the β-cell function may decline over months to a few years, necessitating a relatively early requirement for insulin use.

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A 30-year-old female, who was diagnosed two months earlier with Graves disease and was started on carbimazole 40 mg per day , presented complaining of sore throat.

Investigations reveal:

Haemoglobin	11.5 g/dl
MCV	80 fl
White cell count	4.2 x 10 /l9
Neutrophils	2.0 x 10 /l9
Lymphocytes	2.3 x 10 /l9
Basophils	0.08 x 10 /l9
Eosinophils	0.1 x 10 /l9
Platelets	170 x 10 /l9

What is the most appropriate treatment for this patient?

A. Discontinue carbimazole and give propylthiouracil
B. Discontinue carbimazole and give radioactive iodine
C. Discontinue carbimazole and give antibiotics
D. Reduce the dose of carbimazole
E. Continue carbimazole

E. Continue carbimazole

Patients taking carbimazole may develop neutropaenia (although the incidence is very low) so they are warned about a sore throat while taking the medication.

This patient develpped a sore throat but her total white cell count and differential are normal, so she should be reassured and continue carbimazole.

If there is neutropaenia, carbimazole should be discontinued and substituted by propylthiouracil till the neutrophil count has recovered and then the patient is planned for radioactive iodine.

Question 43 of 348

A 28-year-old lady is diagnosed with gestational diabetes in her first pregnancy. Her fasting blood glucose is 5.9 mmol/l and blood glucose after oral glucose tolerance test (OGTT) is 8.2mmol/l. Blood glucose control during pregnancy is achieved with diet, exercise and metformin. She gives birth to a healthy child at 39 weeks. A fasting blood glucose at day 1 post-partum is 5.2mmol/l.

Which of the following statements is correct with respect to follow-up monitoring for diabetes?

A. OGTT 6-13 weeks postpartum
B. Fasting blood glucose test 6-13 weeks postpartum
C. No routine follow up unless further pregnancy
D. HbA1c 6-13 weeks postpartum
E. Annual fasting blood glucose checks only

B. Fasting blood glucose test 6-13 weeks postpartum

Women with gestational diabetes whose glucose returns to normal after birth need a postnatal glucose check 6-13 weeks postpartum to stratify their risk of developing diabetes in the future. NICE recommends that this is a fasting blood glucose. Further follow up will depend on the result of this postnatal check. Even if postnatal glucose is less than 6mmol/l, annual fasting glucose checks are still recommended thereafter.

Question 44 of 348

A 70-year-old woman is reviewed in the chronic kidney disease clinic. She also has a history of hypertension for which she takes amlodipine 5mg od and ramipril 10mg od. Her most recent results are as follows:

Blood pressure today is 128/74 mmHg.

	Recent	12 months ago
Na +	140 mmol/l	141 mmol/l
K +	4.5 mmol/l	4.3 mmol/l
Urea	11.2 mmol/l	10.5 mmol/l
Creatinine	124 µmol/l	114 µmol/l
eGFR	39 ml/min	43 ml/min

What is the most appropriate next step in management?

A. Start atorvastatin 20mg on
B. Reduce ramipril to 5mg od and recheck U&Es in 4 weeks
C. Start simvastatin 40mg on
D. Increase amlodipine to 10mg od
E. Check her QRISK2 score

A. Start atorvastatin 20mg on

QRISK2 should not be used in patients with chronic kidney diseas (CKD). NICE now recommends that all patients with CKD should take a statin.

- *Offer atorvastatin 20 mg for the primary or secondary prevention of CVD to people with CKD*
- *Increase the dose if a greater than 40% reduction in nonHDL cholesterol is not achieved and eGFR is 30 ml/min*
- *Agree the use of higher doses with a renal specialist if eGFR is less than 30 ml/min*

Question 45 of 348

You review a 28-year-old woman who is 26 weeks pregnant. She has just had a routine oral glucose tolerance test as her BMI is 34 kg/m². The following results were obtained:

Time (hours)	Blood glucose (mmol/l)
0	7.4
2	11.2

There have been no other antenatal problems and her anomaly scan was normal. What is the most appropriate action?

A. Repeat oral glucose tolerance test in 4 weeks
B. Start metformin + advice about diet / exercise + self-monitorglucose levels
C. Advice about diet / exercise + self-monitor glucose levels
D. Start insulin + advice about diet / exercise + self-monitorglucose levels
E. Reassure results within normal limits

D. Start insulin + advice about diet / exercise + self-monitor glucose levels

NICE have recently changed their gestational diabetes guidelines. Insulin should be started in the fasting glucose is >= 7 mmol/l. Aspirin should also be considered given the increased risk of pre-eclampsia.

Question 46 of 348

A 55-year-old male presents to his general practitioner with a 2-month history of sweating, fatigue and daytime tiredness. He attributed his tight wedding ring to 'fluid retention' and in the last 2 weeks, he has been experiencing worsening headaches and deterioration in his vision. He has otherwise no past medical history and does not take any regular medications.

Which of the following findings would be in keeping with the above?

A. Homonymous hemianopia
B. Overbite
C. Positive Tinel's sign
D. Reduced FVC on spirometry
E. Dysdiadochokinesia

C. Positive Tinel's sign

This is a case of acromegaly which is caused by excessive growth hormone secretion. In adults, the excess growth hormone secretion is usually caused by a benign growth hormone-secreting pituitary adenoma.

It is often associated with hypertension, diabetes, obstructive sleep apnoea, carpal tunnel syndrome, colonic polyps/malignancies, cardiomyopathies, and arrhythmias.

1. Homonymous hemianopia: this is false because the visual field disturbance associated with a pituitary tumour is bitemporal hemianopia
2. Overbite: patients with present with underbite, or prognathism
3. Positive Tinel's sign: this is true in carpal tunnel syndrome
4. 4 and 5: these features are not characteristic of acromegaly

Question 47 of 348

A 37-year-old man presents to the endocrinology clinic for annual review of type 1 diabetes mellitus. He was diagnosed with diabetes following an admission with diabetic ketoacidosis (DKA) at the age of 21. Since then he has been managed with a basal-bolus regime of insulin. He has since then had one admission with DKA in the year following his diagnosis due to excessive alcohol intake and has since then significantly reduced his alcohol intake. He had retinal photography 11 months ago which he reports as normal. His blood pressure is 135/81mmHg. He had blood tests showing a normal cholesterol as well as HbA1c at 42mmol/mol. He has given urine today to measure the albumin to creatinine ratio, but there was no protein in the sample on dipstick testing.

What is the most appropriate change to make his medications?

A. Increase long-acting insulin dose
B. Start ACE inhibitor
C. Start calcium-channel blocker
D. Start statin
E. Decrease long-acting insulin dose

D. Start statin

Patients with T1DM for over 10 years should be considered for statin therapy atorvastatin 20mg OD

This is a patient having a diabetic review. He has had diabetes for 16 years and this increases his cardiovascular risk and NICE guidelines advise that all patients with type 1 diabetes mellitus who have had their diagnosis for over 10 years should be offered a statin, even if cholesterol is not measured to be high. His HbA1c demonstrates that he has good control of his diabetes; if it had been higher then increasing long-acting dose of insulin could be considered, but even then the capillary glucose levels should be reviewed through the day to see if it would be more appropriate to increase the bolus doses instead. As there is not evidence of episodes of hypoglycaemia the dose of insulin should not be decreased. Patient should aim to maintain a blood pressure <140/80mmHg if no end-organ damage, and <130/80mmHg of there is end organ damage, such as retinopathy, neuropathy or nephropathy. There is no evidence that the patient has any of these complications. ACE inhibitors should be started if there is any urinary protein, but so far this is negative.

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A 53-year-old obese HGV driver, normally taking BD Novomix 30 insulin presents to your outpatient clinic to clarify some driving regulations he had overheard while eating with colleagues. He is extremely tearful and anxious. He is worried about losing his livelihood as a result of his diabetes.

He was first diagnosed with type 2 diabetes 9 years ago and became insulin dependent 2 years ago. He reports good compliance with insulin every day. However, 18 months ago, he took the same units of insulin after exercising and felt giddy. A spot blood glucose check demonstrated 2.8 mmol/l, which improved immediately after drinking Lucozade that he carried with him. No hospitalisation was required. He has no other past medical history. He has no visual field or peripheral nerve impairments.

What is the advice you give him regarding driving?

A. Can continue driving, review in 1 year
B. Can continue driving, no further reviews required
C. Must stop driving and give up license permanently
D. Must stop driving temporarily and review in 6 months
E. Patient can drive type 1 vehicles (cars, motorcycles) but not type2 vehicles (lorries, HGV) and should reconsider his profession

A. Can continue driving, review in 1 year

The salient points in this case history are that the patient, although insulin dependent for treating his type 2 diabetes, retains hypoglycaemia awareness. A recent change in the DVLA guidelines of May 2012 allows HGV drivers to retain their license even if taking insulin, provided they have not suffered from hypoglycaemia requiring the assistance of others within the past 12 months, and the patient has no visual field impairments. The patient must then be reviewed annually by a diabetes consultant, with 3 months of blood glucose monitoring data available. A 1-year license is then issued annually.

Question 49 of 348

A 38-year-old woman is referred to the outpatient department by her GP with pain in her calves when walking 50 meters. She reports no other symptoms and has no other past medical history other than migraine. She is on no regular medication and her family history includes her mother having diabetes and her father dying of a heart attack aged 46. She currently smokes 35 cigarettes per day and drinks a glass of wine every evening. Her occupation is as a financial advisor.

Examination reveals tendon xanthomas affecting the extensor tendons of his fingers. On examining her face, it is noticed she has xanthelasma around both eyes and corneal arcs.

Which of the following is the most likely diagnosis?

A. Tangier disease
B. Homozygous familial hypercholesterolaemia
C. Heterozygous familial hypercholesterolaemia
D. Familial hypertriglyceridaemia
E. Apo CII deficiency

C. Heterozygous familial hypercholesterolaemia

This patient's symptoms suggest intermittent claudication and combined with the signs of hypercholesterolaemia especially the tendon xanthomas would suggest one of the familial hypercholesterolaemias. Patients with homozygous familial hypercholesterolaemia, present with early cardiovascular disease, sometimes as early as the second decade of life, whereas, patients with heterozygous familial hypercholesterolaemia rarely present before the age 30. Thus, this case is more suggestive of heterozygous familial hypercholesterolaemia.

In heterozygous familial hypercholesterolaemia, total cholesterol would typically be above 7.9 mmol/l, with normal triglyceride levels. In homozygotes it is typically above 15 mmol/l.

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A 35-year-old female presented to the emergency department with severe right flank pain radiating to her groin. She had recently been referred to the rheumatology department by her GP for investigation of joint pains, dry eyes and dry mouth. She was not taking any regular medication.

On examination her blood pressure was 132/68 mmHg. Abdominal examination revealed right flank tenderness. Blood tests are shown below:

Na +	136 mmol/L	(135 - 145)
K +	2.8 mmol/L	(3.5 - 5.0)
Urea	3.6 mmol/L	(2.0 - 7.0)
Creatinine	70 µmol/L	(55 - 120)
Bicarbonate	9 mmol/L	(22 - 28)
Chloride	116 mmol/L	(95 - 105)
Calcium	2.3 mmol/L	(2.1-2.6)
Phosphate	1.1 mmol/L	(0.8-1.4)

What is the most likely diagnosis?

A. Bartter syndrome
B. Fanconi syndrome
C. Gitelman syndrome
D. Lactic acidosis
E. Distal renal tubular acidosis

E. Distal renal tubular acidosis

Distal renal tubular acidosis can cause calcium phosphate renal stones and is linked to Sjogrens syndrome

This patient presents with renal colic. Blood tests show severe metabolic acidosis and hypokalaemia which fits with a distal renal tubular acidosis (RTA). The history of dry eyes and mouth is suggestive of Sjogrens syndrome, which is frequently associated with distal RTA. Patients with distal RTA are at risk of developing calcium phosphate stones.

Fanconi syndrome causes a proximal RTA which tends to cause milder acidosis and hypokalaemia, along with other electrolyte disturbances such as hypophosphataemia.

Gitelman and Bartter syndromes cause a hypokalaemic alkalosis and are therefore incorrect.

Lactic acidosis would cause a raised anion gap acidosis, whereas the anion gap in this case is normal (12).

Question 51 of 348

A 48-year-old man attends the clinic for review. He complains of tiredness and weakness. He has a past medical history of alcoholism and currently drinks 88 units of beer per week. He takes regular thiamine. He is euvolaemic on examination.

Blood results are as follows:

Hb	122 g/l	Na +	128 mmol/l
Platelets	385 * 10 /l 9	K +	3.6 mmol/l
WBC	8.5 * 10 /l 9	Urea	3.2 mmol/l
Neuts	4.6 * 10 /l 9	Creatinine	38 µmol/l
Lymphs	2.2 * 10 /l 9	CRP	8 mg/l

A paired serum and urine test is as follows:

Serum osmolarity	271mOsm/kg (normal range 285-295)
Urine osmolarity	50 mOsm/kg (low)
Urinary sodium	8 mmol/l (low)

What is the most likely diagnosis?

A. Hypothyroidism
B. Potomania
C. SIADH
D. Addison's disease
E. Diabetes insipidus

B. Potomania

Beer potomania is a cause of hyponatremia which occurs due to a low dietary intake of solutes. Urine osmolarity will be low (<100 mosmol/kg) indicating that ADH is appropriately suppressed

Identifying the cause of hyponatraemia can be challenging. The first step is to confirm that it is a true hypotonic hyponatraemia. This is done by measuring the plasma osmolarity.

- If the measured plasma osmolarity is low this confirms true hypotonic hyponatraemia.
- If the plasma osmolarity is normal then this is suggestive of pseudohyponatraemia and should prompt you to measure proteins and lipids, which if present in high levels, can cause a pseudohyponatraemia due to the measuring technique.
- If the serum osmolarity is high then this confirms hypertonic hyponatraemia and should prompt you to check for high levels of solutes in the plasma, hyperglycaemia being the most common.

To identify the cause of the hyponatraemia there are several crucial considerations

- Volume status of the patient: hypervolaemic, euvolaemic, or hypovolaemic
- Paired serum and urine osmolarities
- Urinary sodium
- Thyroid and adrenal function
- History of patient e.g. diarrhoea, burns, vomiting, and kidney dysfunction. This is extremely important as may give clues to the cause (e.g. we could expect hypovolaemia and hyponatraemia with prolonged diarrhoea due to excessive loss of fluid and sodium from the GI tract)
- Drug history: several drugs are implicated in hyponatraemia through several mechanisms including SIADH

In this case, the low measured serum osmolarity confirms a true hypotonic hyponatraemia. The decreased plasma sodium can occur due to too much fluid (e.g. decreased urinary output or increased oral intake) or too little sodium (e.g. decreased oral intake or increased output through kidneys, GI tract, or skin). It can also be due to a combination of disturbed water and sodium balance e.g. combined sodium and water loss with a greater amount of sodium loss.

In this case, the patient is euvolaemic which narrows down the differential. Euvolamic hyponatraemia can occur due to:

- Hypothyroidism
- Psychogenic polydipsia
- Beer potomania

It is now very important to consider the urine osmolarity and sodium.

Urinary sodium is arguably the most useful biochemical measurement. Some key points include:

- In hyponatremia we would expect a low urinary sodium as the body attempts to conserve sodium
- Therefore a high urinary sodium >20 in the presence of hyponatraemia suggests renal sodium loss (e.g. CKD, diuretics, and salt losing nephropathy). Other causes of high urinary sodium include endocrine disease (e.g. Addison's), primary polydipsia, SIADH and reset osmostat
- A low urinary sodium (<20) in the presence of hyponatraemia suggest that the kidneys are conserving sodium. This should prompt you to consider extra-renal sodium losses (e.g. via GI tract or skin). Urinary sodium must be interpreted with caution. For example, if a patient is on diuretics the urinary sodium may be high after taking them but may go very low after their effects wear off, particularly if the patient is dehydrated
- The RAAS system is the dominant factor in sodium balance so consider this carefully
- In secondary hyperaldosteronism (e.g. heart failure, and cirrhosis) we get increased sodium retention and therefore a low urinary sodium In hypoaldosteronism (e.g. Addison's, true hypervolaemic states (e.g. polydipsia, SIADH)) we get high urinary sodium

Some key points with regards to urine osmolarity:

- In the presence of hyponatremia the bodies natural response to raising sodium would be to increase free water output resulting in a high urinary output with low urine osmolarity
- Urine osmolarity helps to differentiate between conditions associated with impaired free water excretion (high urine osmolarity >100), as seen in the majority of cases, and a few conditions associated with low urine osmolarity <100
- Urine osmolarity < 100 suggests that the kidneys are not concentrating urine e.g. ADH is suppressed
- Urine osmolarity > 100 suggests that ADH is not suppressed and is concentrating urine and impairing free water excretion
- Urine osmolarity will be raised in the majority of cases of hyponatraemia either due to inappropriate ADH release (e.g. SIADH), appropriate ADH release (hypovolaemia) or pseudo 'appropriate' ADH release (perceived intravascular depletion but actual hypervolaemic states e.g. heart failure and cirrhosis)
- With primary polydipsia, malnutrition (e.g. tea and toast syndrome, beer potomania) and a reset osmostat, the urine osmolarity is maximally dilute and generally less than 100

In this example, the history of excessive beer consumption, euvolaemic fluid status, low urine osmolarity, and a low urinary sodium, makes beer drinkers potomania the most likely cause of the hyponatraemia.

Potomania, also known as beer drinker's potomania is a specific hypoosmolality syndrome related to massive consumption of beer, which is poor in solutes and electrolytes. With little food or other sources of electrolytes, consumption of large amounts of beer or other dilute alcoholic drinks leads a low level of urinary solute excretion (approximately 200-300 mOSM). This limits the amount of free water excretion to approximately 4 litres. Any intake above 4 litres would lead to a dilution of the serum sodium concentration and thus hyponatraemia.

Question 52 of 348

A 50-year-old female is admitted with epigastric pain radiating to her back. She has no past medical history and takes no regular medications. Her alcohol history includes 4 glasses of wine per week.

Blood results are as follows:

Hb	115 g/l	Na^+	138 mmol/l
Platelets	288 * 10 /l 9	K^+	3.8 mmol/l
WBC	10.8 * 10 /l 9	Urea	6.8 mmol/l
Neuts	8.8 * 10 /l 9	Creatinine	72 µmol/l
Lymphs	1.8 * 10 /l 9	CRP	84 mg/l
Triglyceride	12.3 mmol/l (normal range < 2.3 mmol/L)	Bilirubin	16 µmol/l
ALT	24 u/l	Amylase	924 U/L
ALP	76 u/l	Albumin	38 g/l

An abdominal ultrasound is performed:

Abdominal ultrasound report	Np gall stones identified. No intra or extra hepatic duct dilation evident

What treatment will reduce the risk of pancreatitis reoccurrence?

A. Ursodeoxycholic acid
B. Fenofibrate
C. Simvastatin
D. Nicotinic acid
E. Laparoscopic cholecystectomy

B. Fenofibrate

Fibrates are the most effective drug for treating hypertriglyceridaemia

The patient has clinical features of pancreatitis. The raised amylase confirms the diagnosis. The two most common causes of pancreatitis in the UK are excess alcohol and gallstones.

The normal liver function tests and ultrasound rule out gallstone disease as the cause. Therefore, a laparoscopic cholecystectomy or treatment with ursodeoxycholic acid is not indicated.

The patient drinks 4 glasses of wine per week. A 250ml glass of wine at 12% alcohol contains 250 * 0.12 = 30 ml of pure alcohol. 10 ml of pure alcohol = 1 unit. Therefore, each glass of wine contains 3 unit. Her alcohol intake per week is therefore 12 units which is within the recommended intake. It is unlikely that alcohol is the cause.

A patient has a significantly high triglyceride level. While gallstones and alcohol account for roughly 80% of cases of pancreatitis, hypertriglyceridemia is the next most common aetiology, accounting for roughly 1-4% of cases. The exact mechanism is unclear but it is thought to involve increased concentrations of chylomicrons in the blood. These low-density particles are very large and may obstruct capillaries leading to local ischemia and acidaemia. This local damage can expose triglycerides to pancreatic lipases.

Fibrates, statins, and nicotinic acid will all lower triglyceride levels, however, fibrates are the most effective drug in doing so.

Question 53 of 348

A 55-year-old male presents with excessive sweating and headache. You order an oral glucose tolerance test which is as follows:

Time (mins)	0	30	60	90	120
Growth hormone (ng/ml)	2.2	2.4	3.1	3.6	3.7
Glucose	9.1	10.8	11.5	12.8	15.2

Given the likely diagnosis, what other investigation will be required in the future?

A. Transesophageal echocardiography
B. Colonoscopy
C. Flexible sigmoidoscopy
D. CT colonography
E. No further investigations required

B. Colonoscopy

Patients with acromegaly should be offered regular colonoscopic screening, starting at the age of 40 years. The frequency of repeat colonoscopy should depend on the findings at the original screening and the activity of the underlying acromegaly

The clinical features and results of the oral glucose tolerance test confirm a diagnosis of acromegaly. In normal a rise in glucose should cause suppression of growth hormone (GH) to undetectable values.

Cardiovascular disease is highly prevalent in Acromegaly due to a combination of risk factors associated with this disease, in particular, secondary diabetes mellitus, hypertension and hyperlipidaemia. Patients should therefore be screened for cardiovascular risk factors and these should be managed aggressively. A transthoracic echo would be prudent to perform, however transesophageal echocardiography is unnecessary.

Patients with acromegaly have high levels of insulin like growth factor which predisposes to malignancy, in particularly colorectal carcinoma. Colonoscopy, flexible sigmoidoscopy and CT colonography can all be used to screen for colorectal carcinoma, however colonoscopy is the most sensitive test making this the best answer.

Patients with acromegaly should be offered regular colonoscopic screening, starting at the age of 40 years. The frequency of repeat colonoscopy should depend on the findings at the original screening and the activity of the underlying acromegaly.

Question 54 of 348

A 52-year-old female presents with galactorrhoea. On systematic enquiry she reveals that she has been feeling very fatigued recently. She has no past medical history and takes no regular medicines.

Blood results are as follows:

Hb	125 g/l	Na +	132 mmol/l
Platelets	422 * 10 /l 9	K +	3.8 mmol/l
WBC	9.2 * 10 /l 9	Urea	6.4 mmol/l
Neuts	6.2 * 10 /l 9	Creatinine	41 µmol/l
Lymphs	2.4 * 10 /l 9	CRP	4 mg/l
Prolactin	440 ng/dL (normal 5 - 40)		

What investigation will you perform?

A. MRI pituitary
B. Synacthen test
C. Thyroid function tests
D. Toxicology screen
E. No further investigations required

C. Thyroid function tests

Primary hypothyroidism can cause hyperprolactinaemia. High leveles of thyrotrophin releasing hormone (TRH) stimulate prolactin release

The patient has galactorrhoea secondary to hyperprolactinaemia. From the history there is not much information to narrow down our differential diagnosis which is currently wide. Hypothyroidism is a very common condition and can cause hyperprolactinaemia.

High levels of thyrotrophin releasing hormone (TRH) seen in primary hypothyroidism stimulate prolactin release. Hypothyroidism can also cause hyponatraemia as seen in this case. Thyroid function tests are therefore the best answer. A pregnancy test should also be considered.

The Synacthen test is used to assess for hypoadrenalism. Hypoadrenalism is not linked to hyperprolactinaemia.

Certain drugs can cause hyperprolactinaemia (e.g. dopamine antagonists). However a toxicology screen would not detect these agents.

Once the common causes of hyperprolactinaemia have been excluded (e.g. hypothyroidism, pregnancy, and drugs) then you should consider investigating for a pituitary adenoma with an MRI scan.

Question 55 of 348

A 22-year-old man presents with excessive thirst and urination. He has a strong family history of diabetes. His BMI is 20.5 kg/m².

Blood results are as follows:

Hb	145 g/L	Male: (135-180)Female: (115 - 160)
Platelets	210 * 10 /L 9	(150 - 400)
WBC	10.8 * 10 /L 9	(4.0 - 11.0)
Na +	135 mmol/L	(135 - 145)
K +	4.2 mmol/L	(3.5 - 5.0)
Urea	6.8 mmol/L	(2.0 - 7.0)
Creatinine	74 µmol/L	(55 - 120)
Random glucose	14.1 mmol/l	(<11.1)
CRP	2 mg/L	(< 5)

Which mutation is associated with the best response to low-dose sulphonylureas?

A. Glucokinase
B. HNF1A mutation
C. HNF1B mutation
D. HNF4A mutation
E. PDX1 mutation

B. HNF1A mutation

MODY associated with HNF1A often respond well to treatment with low-dose sulfonylureas

The patient most likely has a diagnosis of maturity-onset diabetes of the young (MODY) given his raised blood glucose, young age, strong family history of diabetes, and normal BMI.

HNF1A mutation is correct. MODY associated with HNF1A often responds well to treatment with low-dose sulfonylureas.

Glucokinase is incorrect. This mutation is associated with MODY 2. This subtype has a low rate of complications and often does not need treatment.

HNF1B mutation is incorrect. This subtype does not respond favourably to sulphonylureas.

HNF4A mutation is incorrect. Although patients with this subtype may also respond favourably to sulphonylureas, the response is usually less marked compared to HNF1A mutations.

PDX1 mutation is incorrect. This subtype does not respond favourably to sulphonylureas.

Question 56 of 348

A 52-year-old man presents to the endocrine clinic with a 1-month history of polyuria and polydipsia. He has no significant past medical history and takes no regular medications.

Blood results:

Na +	156 mmol/L	(135 - 145)
K +	3.6 mmol/L	(3.5 - 5.0)
Urea	14.2 mmol/L	(2.0 - 7.0)
Creatinine	162 µmol/L	(55 - 120)

Osmolarity studies after water deprivation are as follows:

Urine osmolarity	105 mmol/L	(300-900)
Plasma osmolarity	332.5 mOsm/kg	(275 - 295)

Results following administration of desmopressin:

Urine osmolarity	322 mmol/L	(300-900)
Plasma osmolarity	324.5 mOsm/kg	(275 - 295)

What is the most likely diagnosis?

A. Complete cranial diabetes insipidus
B. Nephrogenic diabetes insipidus
C. Partial cranial diabetes insipidus
D. Primary polydipsia
E. Syndrome of inappropriate antidiuretic hormone release

A. Complete cranial diabetes insipidus

Water deprivation test: cranial DI

urine osmolality after fluid deprivation: low

urine osmolality after desmopressin: high

Complete cranial diabetes insipidus is correct. The water deprivation test confirms a diagnosis of diabetes insipidus (DI) due to a failure of the urine to concentrate with dehydration (urine osmolarity <300 mmol/L). Following administration of desmopressin there is a >50% rise in urine osmolarity confirming the diagnosis of cranial DI.

Partial cranial diabetes insipidus is incorrect. This subtype of cranial DI is confirmed when the patient's symptoms resolve with desmopressin however the urine osmolarity fails to meet the diagnostic criteria (e.g. >50% increase).

Nephrogenic diabetes insipidus is incorrect. This condition is characterised by the failure of the urine to concentrate with desmopressin.

Primary polydipsia is incorrect. This condition is characterised by a normal physiological response to water deprivation e.g. the urine will concentrate (>600 mmol/L).

Syndrome of inappropriate antidiuretic hormone release is incorrect. Syndrome of inappropriate antidiuretic hormone release (SIADH) is associated with hyponatraemia, a decreased plasma osmolarity (<275 mOsm/kg), a relatively low urine osmolarity (< 100 mOsm/kg), and a high urinary sodium (> 40 mmol/L).

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A 64-year-old woman attends the emergency department with heart palpitations. She has a past medical history of type 2 diabetes mellitus. Observations are as follows: heart rate 92 beats per minute, and blood pressure 145/85 mmHg,

Blood results are as follows:

Hb	116 g/L	Male: (135-180)Female: (115 - 160)
Platelets	156 * 10 /L 9	(150 - 400)
WBC	8.2 * 10 /L 9	(4.0 - 11.0)
Na +	136 mmol/L	(135 - 145)
K +	6.5 mmol/L	(3.5 - 5.0)
Cl -	125 mmol/L	(96 - 106)
Urea	9.2 mmol/L	(2.0 - 7.0)
Creatinine	115 µmol/L	(55 - 120)
Bicarbonate	12 mmol/L	(23 - 29)
Glucose	34.5 mmol/L	(4 - 7)
CRP	2 mg/L	(< 5)

What is the most likely diagnosis?

A. Addison's disease
B. Diabetic ketoacidosis (DKA)
C. Type 1 renal tubular acidosis
D. Type 2 renal tubular acidosis
E. Type 4 renal tubular acidosis

E. Type 4 renal tubular acidosis

Renal tubular acidosis (type 4) causes hyperkalaemia

The patient has a metabolic acidosis as indicated by the low serum bicarbonate. The anion gap (AG) is a derived variable primarily used for the evaluation of metabolic acidosis to determine the presence of unmeasured anions. The normal anion gap varies with different assays but is typically 4 to 12 mmol/L.

The anion gap = (Na + K) - (Cl + HCO3) = (136 + 6.5) - (125 + 12) = 5.5. Thus the patient has a normal anion gap metabolic acidosis (NAGMA).

Causes of NAGMA include ('ABCD'):

- Addison's
- Bicarbonate loss: GI (e.g. diarrhoea) or renal (e.g. renal tubular acidosis)
- Chloride excess
- Diuretics (e.g. acetazolamide)

Causes of high anion gap metabolic acidosis (HAGMA) include:

- Lactate
- Toxins (e.g. methanol, paracetamol, propylene glycol)
- Ketones
- Renal failure

Type 4 renal tubular acidosis is correct. The presence of NAGMA, hyperkalemia, and a history of diabetes favours a diagnosis of type 4 renal tubular acidosis.

Type 1 renal tubular acidosis is incorrect. Although this condition also presents with NAGMA, it is associated with hypokalemia.

Addison's disease is incorrect. Addison's can result in NAGMA, with hyperkalemia as a consequence of hypoaldosteronism. However, the presence of hyperglycaemia and hypertension makes this a less likely diagnosis. One would expect hypoglycaemia and hypotension in Addison's.

Diabetic ketoacidosis (DKA) is incorrect. Although the patient is significantly hyperglycaemia, the presence of ketones would result in a HAGMA.

Type 2 renal tubular acidosis is incorrect. This condition is associated with NAGMA and an hypokalemia.

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A 64-year-old man presents with confusion. A systematic enquiry was otherwise unremarkable. He has a history of bipolar disorder for which he takes lithium. He does not take any other regular medications. He drinks approximately 6 pints of beer per day. He is euvolemic on examination.

Blood results are as follows:

Na +	125 mmol/L	(135 - 145)
K +	3.6 mmol/L	(3.5 - 5.0)
Urea	6.2 mmol/L	(2.0 - 7.0)
Creatinine	72 µmol/L	(55 - 120)
TSH	4.8 mU/L	(0.5-5.5)
Free T4	14.2 pmol/L	(9.0 - 18)
Early morning cortisol	440 nmol/l	(>350)

Osmolarity studies are requested:

What is the most likely explanation?

A. Beer potomania
B. Cerebral salt wasting
C. Diabetes insipidus
D. Primary polydipsia
E. Syndrome of inappropriate antidiuretic hormone ADH release (SIADH)

E. Syndrome of inappropriate antidiuretic hormone ADH release (SIADH)

SIADH criteria includes Na < 135, serum osmolality <271 and urinary osmolality >100 in a euvolaemic patient

The patient has significant hyponatraemia. Since the serum osmolarity is < 275 we have confirmed a true hypotonic state.

In contrast, if the serum osmolarity was normal (275-295) then this would imply isotonic hyponatremia (i.e. pseudohyponatraemia). This is a measuring defect which can occur due to high levels of lipids (e.g. triglycerides) or proteins (e.g. macroglobulinaemia). These macromolecules take up a proportion of plasma volume thereby resulting in a spurious hyponatremia (as the lab measures Na / plasma volume rather than Na / plasma fluid).

If the serum osmolarity is > 295 then this implies a hypertonic hyponatremia. This occurs due to high levels of osmotically active solutes (e.g. hyperglycaemia or mannitol). This results in an osmotic shift of fluid into the extracellular space resulting in dilutional hyponatremia.

In this clinical case, we are dealing with a true hypotonic hyponatremia. The causes of this are wide but can broadly be divided into:

Too much fluid (e.g. decreased output or increased intake) Too little sodium (e.g. decreased intake or increased output) Can also be due to a combination e.g. sodium and water loss (with sodium loss > water loss).

It is therefore absolutely essential to assess the fluid status of the patient to help identify the cause.

Urinary sodium is an exceptionally useful tool, and indeed after clinical examination, this is the most useful investigation. In hyponatremia, we would expect low urinary sodium as a physiological response to conserve sodium. Therefore urinary sodium >10 is suggestive of renal sodium loss (e.g. CKD, diuretics, hypoaldosteronism, and salt-losing nephropathy). Other causes of high urinary sodium include endocrine disease (e.g. Addison's), primary polydipsia, and SIADH. In SIADH, an initial hypervolaemic state results in aldosterone suppression resulting in natriuresis and high urinary sodium. In secondary hyperaldosteronism (e.g. heart failure, nephrotic syndrome) we get increased sodium retention and therefore low urinary sodium.

It is extremely important to note that urine osmolarities and electrolytes must be interpreted with caution. Urinary sodium will be high after taking diuretics, but may go very low after their effects wear off, particularly if the patient is dehydrated.

The urine osmolarity is often not particularly helpful. In the presence of hyponatremia, the body's natural response to raise sodium would be to increase free water output resulting in a high urinary output with low urine osmolarity. Therefore urine osmolarity helps to differentiate between conditions associated with impaired free water excretion (high urine osmolarity >100), as seen in the majority of cases, and a few conditions associated with low urine osmolarity <100.

Urine osmolarity	320 mmol/L	(300-900)
Urine sodium	54 mmol/L	(>40)
Plasma osmolarity	261.5 mOsm/kg	(275 - 295)

The urine osmolarity will be raised (>100) in the majority of cases either due to inappropriate ADH release (e.g. SIADH), appropriate ADH release (hypovolaemia) or pseudo 'appropriate' ADH release (perceived intravascular depletion but actual hypervolaemic states e.g. nephrotic syndrome, heart failure and cirrhosis). However, if it is low (<100) then this suggests psychogenic polydipsia or beer drinker's protomania.

Syndrome of inappropriate antidiuretic hormone ADH release (SIADH) is correct. The patient fulfils the diagnostic criteria of SIADH making this the most likely diagnosis. Importantly he fulfils the biochemical criteria, has normal thyroid and adrenal function, is euvolemic, and is not on diuretics which may confound the biochemical analysis.

Beer potomania is incorrect. Potomania is a specific hypo-osmolality syndrome related to the massive consumption of beer, which is poor in solutes and electrolytes. In this condition, one would expect the urine osmolarity to be <100 since free water excretion is not impaired in this condition.

Cerebral salt wasting is incorrect. Both cerebral salt wasting and SIADH are characterised by hyponatremia with elevated urinary sodium and concentrated urine. The key distinguishing factor is that in cerebral salt wasting the patient is hypovolemic whereas SIADH patients are euvolaemic.

Diabetes insipidus is incorrect. The fact that the patient is on lithium is a red herring. Lithium is a well-known cause of diabetes insipidus (DI), however, DI is essentially the opposite condition to SIADH. In DI we have a failure of ADH to act on the kidneys resulting in polyuria, dehydration,

raised serum osmolarity and hypernatraemia,

Primary polydipsia is incorrect. In this condition, one would expect the urine osmolarity to be <100 since free water excretion is not impaired.

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An 89-year-old woman is reviewed in the frailty clinic.

She was recently admitted under the care of the elderly team with an episode of delirium, which was triggered by a variety of factors, including a change in housing and constipation.

A course of trimethoprim was prescribed during this admission for a suspected UTI on the basis of urinary urgency but a subsequent urine specimen showed no growth.

You take a detailed history now that she has recovered from her delirium and you strongly suspect she is suffering from urge incontinence, which significantly impacts her quality of life.

She has previously undergone a period of bladder training for this, but given her symptoms persist, you are keen to start the patient on medication to improve her urge incontinence.

What is the most appropriate medication to use in this case?

A. Darifenacin
B. Duloxetine
C. Mirabegron
D. Oxybutynin
E. Tolterodine

C. Mirabegron

Anticholinergics for urge incontinence are associated with confusion in elderly people - mirabegron is a preferable alternative

Urge incontinence can generally be managed with bladder training, but if this is unsuccessful, a trial of medication is recommended in the NICE guidelines.

The correct answer, in this case, is **mirabegron**. Although anticholinergic drugs are first-line, this is recommended as an acceptable alternative when they are contra-indicated.

Anticholinergic drugs (such as **darifenacin, oxybutynin** and **tolterodine**) are first line, but there are some safety concerns, particularly in elderly patients at risk of delirium. This makes these options incorrect choices for this patient, who has been admitted recently with delirium.

Duloxetine is incorrect. It is not used in the management of urge incontinence but is a second-line treatment for stress incontinence when other measures have failed.

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A 46-year-old man presents to the Endocrinology clinic after a referral from his GP for asymptomatic hypercalcaemia, which was recently discovered on some routine blood tests. His past medical history is significant only for hypertension, for which he is taking amlodipine, ramipril and chlorthalidone.

Selected investigation results are shown below:

Na +	141 mmol/L	(135 - 145)
K +	4.4 mmol/L	(3.5 - 5.0)
Calcium	2.85 mmol/L	(2.2 - 2.6)
Urea	6.6 mmol/L	(2.0 - 7.0)
Creatinine	98 µmol/L	(55 - 120)
Parathyroid hormone	5.5 pmol/L	(1.6 - 6.9)
Urinary calcium	30mg/24h	(100 - 300)

What is the most likely explanation for this patient's hypercalcaemia?

A. Chlorthalidone use
B. Familial hypocalciuric hypercalcaemia
C. Hypercalcaemia of malignancy with ectopic parathyroidhormone-related protein secretion
D. Primary hyperparathyroidism
E. Secondary hyperparathyroidism

B. Familial hypocalciuric hypercalcaemia

Low urinary calcium in the presence of hypercalcaemia is suggestive of either familial hypocalciuric hypercalcaemia or thiazide diuretic use

The correct answer is **familial hypocalciuric hypercalcaemia**. This patient has hypercalcemia with low urinary calcium, which narrows the diagnosis down to familial hypocalciuric hypercalcemia (FHH) or thiazide diuretic usage (other causes of hypercalcemia tend to cause elevated urinary calcium). The diagnosis of FHH is clinched by the inappropriately normal parathyroid hormone. This is because, in FHH, loss of function mutations in the CASR calcium-sensing receptor decrease sensitivity to calcium, meaning PTH remains unsuppressed at higher-than-normal serum calcium levels. Hypocalciuria results from a loss of CASR-mediated negative feedback of tubular reabsorption/ excretion of calcium.

Chlorthalidone use is incorrect, as this would give hypocalciuric hypercalcaemia with a suppressed PTH.

Hypercalcaemia of malignancy with ectopic parathyroid hormonerelated protein secretion is incorrect. This would classically lead to hypercalciuria. In addition, serum PTH may be suppressed rather than inappropriately normal in this scenario, as the PTHrp peptide is detected by a different assay from the conventional PTH assay.

Primary hyperparathyroidism is incorrect. Although this could explain the hypercalcaemia and inappropriately normal PTH, this would lead to hypercalciuria rather than hypocalciuria.

Secondary hyperparathyroidism is incorrect. Here, PTH is released in response to hypocalcaemia, often in the context of renal disease. The elevated serum calcium and normal renal function seen in this patient do not support this diagnosis.

The parathyroid hormone level is often not suppressed, as would be expected in all non-hyperparathyrodism related cases of hypercalcaemia. This is due to the decreased sensitivity to increases in extracellular calcium.

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A 39-year-old man is assessed in the Neurology clinic. His past medical history is significant for migraine with aura, for which he is taking topiramate. He is not taking any other medications.

Selected investigation results are shown below:

Na +	138 mmol/L	(135 - 145)
K +	3.1 mmol/L	(3.5 - 5.0)
Urea	5.7 mmol/L	(2.0 - 7.0)
Creatinine	78 µmol/L	(55 - 120)

Venous blood gas:

pH	7.29	(7.35 - 7.45)
Bicarbonate	16 mmol/L	(22 - 29)

Urinalysis:

Glucose	3+
Protein	2+
Blood	negative

What is the most likely diagnosis?

A. Membranous glomerulonephritis
B. Type 1 renal tubular acidosis
C. Type 2 renal tubular acidosis
D. Type 3 renal tubular acidosis
E. Type 4 renal tubular acidosis

C. Type 2 renal tubular acidosis

Fanconi syndrome causes proximal renal tubular acidosis and can be drug induced

The correct answer is **type 2 renal tubular acidosis**. This patient has metabolic acidosis with hypokalaemia, proteinuria and glycosuria. The presence of proteinuria and glycosuria suggest a problem at the proximal convoluted tubule, as protein and glucose should be fully reabsorbed at this part of the nephron. Type 2 renal tubular acidosis is a consequence of a defect of the proximal tubular cells, leading to incomplete protein and glucose reabsorption, as well as defective bicarbonate reabsorption leading to acidosis. This is the most likely diagnosis in this patient and has likely been precipitated by the drug topiramate.

Membranous glomerulonephritis is incorrect. To diagnose membranous glomerulonephritis, one would expect to see nephrotic range proteinuria (>3g per 24h), and ideally, histological confirmation on renal biopsy. Membranous glomerulonephritis is more associated with NSAIDs and penicillamine than topiramate and is unlikely to cause metabolic acidosis in the context of the normal renal function seen here.

Type 1 renal tubular acidosis is incorrect. Although this can cause hypokalaemic metabolic acidosis, the distal tubular cells are affected rather than the cells of the proximal tubule. This means there is less of an association with proteinuria and glycosuria. Finally, topiramate is not known to precipitate type 1 renal tubular acidosis.

Type 3 renal tubular acidosis is incorrect. This tends to be seen in children or as a result of congenital carbonic anhydrase deficiency. It is epidemiologically far rarer than type 2 renal tubular acidosis and is not specifically associated with topiramate use.

Type 4 renal tubular acidosis is incorrect. This is associated with hyperkalemia rather than hypokalaemia and tends to be associated with mineralocorticoid receptor antagonists and NSAIDs rather than topiramate.

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A 42-year-old woman presents with feeling warm and more anxious than usual. She had an upper respiratory tract infection three weeks ago. On examination, you find a new tender goitre.

You send blood tests including thyroid function tests which are outlined in the table below.

TSH	0.2 mU/L	(0.5-5.5)
Free T4	25 pmol/L	(9.0 - 18)

Given the likely underlying diagnosis, what results do you expect from thyroid scintigraphy?

A. Globally normal uptake
B. Globally increased uptake
C. Globally reduced uptake
D. Well defined area of decreased uptake
E. Well defined area of increased uptake

C. Globally reduced uptake

De Quervain's thyroiditis: initial hyperthyroidism, painful goitre and globally reduced uptake of iodine-131

Subacute thyroiditis is thought to occur following viral infection and typically presents with hyperthyroidism and a new painful goitre. The investigation of choice is thyroid scintigraphy which reveals globally reduced uptake of iodine-131. Most cases are self-limiting and do not require treatment. The other answers provided are not the most common expected findings for De Quervain's thyroiditis.

Global normal uptake with uniform uptake of iodine-131 is most associated with a normal thyroid gland. Given the abnormal thyroid function and painful goitre, this would be an unexpected result in this case.

Globally increased uptake is most commonly associated with Grave's disease.

A well-defined area of decreased uptake would be most consistent with a cold (hypofunctioning) thyroid nodule. This finding would also not be consistent with the painful goitre or hyperthyroidism noted on blood tests in this case.

A well-defined area of increased uptake would be most consistent with a hot (hyperfunctioning) thyroid nodule.

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A 32-year-old woman presents with recurrent headaches and blurred vision. She is fatigued and has recurrent constipation requiring laxatives. There is a past medical history of hypertension and she is on amlodipine, ramipril and bendroflumethiazide.

On examination, her blood pressure is 178/102 mmHg. Upon fluid balance review, she seems hypervolaemic with bilateral peripheral oedema. She is of slim build and her cranial nerves and neurological examination show no abnormalities. Fundoscopy reveals grade IV hypertensive retinopathy with papilloedema seen.

Her electrocardiogram reveals features of left ventricular hypertrophy. Her urine dip is negative for protein and blood. A CT head reveals no acute bleed.

Blood results are as follows:

Na +	154 mmol/L	(135 - 145)
K +	2.9 mmol/L	(3.5 - 5.0)
Bicarbonate	23 mmol/L	(22 - 29)
Aldosterone	70 pmol/L	(100 - 500)
Renin	2 mU/L	(5-50)

What is the most likely diagnosis?

A. Bartter's syndrome
B. Excess salt in diet
C. Gitelman's syndrome
D. Liddle syndrome
E. Renal artery stenosis

D. Liddle syndrome

Hypernatraemia associated with hypervolaemia can occur due to hypertonic saline, hypertonic sodium bicarbonate, excess salt in diet, or hyperaldosteronism

Liddle syndrome is the correct option. This rare autosomal dominant syndrome is due to dysregulation of the epithelial sodium channel (ENaC) due to a genetic mutation at the 16p13-p12 locus, leading to the Na channel not being degraded, thus leading to chronic reabsorption of sodium and water in the nephron. This mimics a state of hyperaldosteronism as aldosterone is usually the hormone implicated in inserting the ENaCs into the nephron. Therefore, this patient has a state of hypervolaemic hypernatraemia with chronically high blood pressure creating the end-organ damage to the optic nerve and heart described above.

The feedback loop in the kidneys has meant that both renin and aldosterone are low for her body to attempt to lower the sodium and water retention. This patient is fatigued and constipated due to hypokalaemia. Liddle's is treated with a combination of a low sodium diet and potassium-sparing diuretics (e.g. amiloride).

Liddle's has a classic triad in the textbooks of hypertension, hypokalemia and metabolic alkalosis. However, hypernatraemia is also a feature due to the above processes and this should not be forgotten in the clinical work-up.

Bartter's syndrome is an incorrect option. This is an autosomal recessive syndrome which causes salt-wasting from the kidney and subsequently a state of hyperaldosteronism and usually low blood pressure, not high blood pressure as seen in this case.

Gitelman's syndrome is an incorrect option as it is another autosomal recessive syndrome similar to Bartter's, which causes low blood pressure usually.

Excess salt in the diet is incorrect. Even though it would cause hypernatraemia and chronic excess would cause states of hypertension; you would not find hypokalaemia. Furthermore, it would be a more unlikely cause at the young age of this patient with the anti-hypertensive therapy resistance.

Renal artery stenosis is an incorrect answer because this condition causes difficulty to control hypertension with a high plasma renin activity found, instead of low, as more renin is produced in response to the low pressure sensed by the constricted renal artery. The kidneys think that the blood pressure is systemically low when it is a locally mediated effect from the stenosis.

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A 38-year-old woman is 10 weeks pregnant and presents with increasing fatigue, weight gain and feeling very cold. Moreover, her main issue is constipation only responsive to large doses of laxatives. In recent years she has had well-controlled hypothyroidism on 50 micrograms of levothyroxine.

On examination her hair is very thin, her skin feels slightly waxy, she has mild peripheral oedema and has a larger body habitus than previously.

Blood tests are as follows:

Calcium	2.4 mmol/L	(2.1-2.6)
Phosphate	1.1 mmol/L	(0.8-1.4)
Magnesium	0.8 mmol/L	(0.7-1.0)
TSH	5.0 mU/L	(0.5-5.5)
Free T4	9.8 pmol/L	(9.0 - 18)

What is the best next management option?

A. 100 micrograms levothyroxine
B. 25 micrograms levothyroxine
C. 75 micrograms levothyroxine
D. Continue 50 micrograms levothyroxine
E. Stop the levothyroxine

C. 75 micrograms levothyroxine

Women with hypothyroidism may need to increase their thyroid hormone replacement dose by up to 50% as early as 4-6 weeks of pregnancy

75 micrograms levothyroxine is the correct answer. This is because during pregnancy (as early as 4-6 weeks) the amount of thyroglobulin binding protein increases, thus there is less free thyroid hormone in the bloodstream exacerbating this woman's state of hypothyroidism.

Increasing the dose of 50 by 50% to 75 is usually sufficient as a measure to induce a state of euthyroid.

25 micrograms levothyroxine is incorrect as this would worsen the state of hypothyroidism.

Continue 50 micrograms levothyroxine is incorrect as this would not address the issue of the state of hypothyroidism on the current dose.

Stop the levothyroxine is incorrect as this would almost certainly exacerbate the state of hypothyroidism yet further.

100 micrograms levothyroxine is incorrect as this would be an increase on the dose by 100%; at this stage most likely too much and may cause a state of hyperthyroidism.

Question 65 of 348

A 69-year-old woman presents to the general practitioner. She reports that when she gets dressed she has noticed that she has been involuntarily leaking urine throughout the day however she denies any episodes of sudden urgency. Otherwise, she is fit and well and performs her activities of daily living independently.

What is the most appropriate first-line management?

A. Bladder retraining
B. Mirabegron
C. Oxybutynin
D. Pelvic floor muscle training
E. Tamsulosin

D. Pelvic floor muscle training

Urinary incontinence - first-line treatment:

urge incontinence: bladder retraining

stress incontinence: pelvic floor muscle training

This woman is presenting with stress incontinence, a leaking of urine normally triggered by coughing, laughing, or sneezing. Although often associated with pelvic floor damage for example during childbirth, it can also present as a result of age-related changes. The first-line management for stress incontinence is **pelvic floor muscle training** which is the correct answer here.

Bladder retraining is useful in cases of urge incontinence however this patient denies any history of urinary urgency. Urge incontinence occurs due to an overactive bladder. Bladder retraining aims to regain control over the function of the bladder. As stress incontinence is related to weakened pelvic floor muscles, bladder retraining would not be as effective as pelvic floor muscle training in this case.

Mirabegron is a beta-3 agonist that relaxes the detrusor muscle of the bladder, increasing bladder capacity. As the primary problem with stress incontinence is related to the pelvic floor muscles, mirabegron would not be the first-line treatment in this case.

Oxybutynin is an antimuscarinic that is also prescribed for an overactive bladder. Again, this relaxes the detrusor muscles but as the primary pathology in stress incontinence is related to the pelvic floor muscles, this would not be the first-line management step here.

Tamsulosin is an alpha-1 antagonist which relaxes the smooth muscle in the bladder neck and urethra. This is useful in the treatment of overflow incontinence as a result of urethral obstruction for example from benign prostatic hyperplasia. It has no effect on the pelvic floor muscles which are weakened in stress incontinence and so has no role here.

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A 76-year-old man with advanced prostate cancer and bone metastases is admitted to hospital, complaining of generalised abdominal pain and presenting with some confusion. A number of blood tests are performed, including the following:

Calcium	3.2 mmol/L	(2.1-2.6)

Aggressive intravenous fluid rehydration is administered (4-6 litres per day), which is unsuccessful. Pamidronic acid is also tried, which is unsuccessful, as a repeat blood test shows a similar result:

Calcium	3.18 mmol/L	(2.1-2.6)

What is most reasonable to try next?

A. Calcitonin
B. Furosemide
C. Ibandronic acid
D. Increase intravenous fluids
E. Prednisolone

A. Calcitonin

Refractory hypercalcaemia of malignancy may be treated with subcutaneous calcitonin if therapy with fluids and pamidronate fails

The case above is an example of refractory hypercalcaemia on a background of malignancy. As intravenous fluid treatment is unsuccessful, bisphosphonates are tried next. Given that these are also unsuccessful, the most suitable step to try next, given the patient's history, is **calcitonin**. This is administered subcutaneously.

Furosemide is a loop diuretic. These are sometimes used in hypercalcaemia, particularly those who cannot tolerate aggressive fluid rehydration. However, in this patient, calcitonin is a more suitable choice.

Ibandronic acid is another example of a bisphosphonate. Given that pamidronic acid has already been tried, and was unsuccessful, calcitonin would be a more reasonable step at this stage, rather than trying another bisphosphonate.

Increasing fluids beyond the current 4-6 litres per 24 hour period would not be appropriate. The patient is already taking a large volume of fluid and further increase may lead to overload.

Prednisolone is a steroid and may be used if the patient had a background of sarcoidosis or other granulomatous disease, rather than malignancy.

Question 67 of 348

A 36-year-old woman is reviewed 3 months post-partum. During the pregnancy she was diagnosed with gestational diabetes. Following the delivery her glycaemic control has failed to improve and she has been diagnosed as having type 2 diabetes mellitus. She is only slightly overweight (body mass index 27.1 kg/m²) and you are worried about missing maturity onset diabetes of the young (MODY) or type 1 diabetes.

Which one of the following is most suggestive of MODY?

A. Ketosis during periods of hyperglycaemia
B. Family history of early onset diabetes mellitus
C. A history of polycystic ovarian syndrome
D. Lack of response to sulphonylureas
E. A history of autoimmune disease

B. Family history of early onset diabetes mellitus

sMODY is inherited in an autosomal dominant fashion so a family history is often present

The correct answer is a **family history of early onset diabetes mellitus**. Maturity onset diabetes of the young (MODY) is a group of monogenic disorders characterized by autosomal dominant inheritance, early onset of hyperglycemia (usually before 25 years of age), and a primary defect in pancreatic beta-cell function. A family history of early onset diabetes mellitus is most suggestive of MODY because it indicates that there may be a genetic predisposition to the condition, which is consistent with the autosomal dominant inheritance pattern seen in MODY.

Ketosis during periods of hyperglycaemia is more suggestive of type 1 diabetes rather than MODY. Type 1 diabetes results from autoimmune destruction of pancreatic beta cells, leading to an absolute insulin

deficiency. In the absence of sufficient insulin, the body relies on breaking down fats for energy, resulting in ketone production and ketosis. In contrast, patients with MODY typically have a defect in pancreatic beta-cell function but still produce some insulin, so they are less likely to develop ketosis.

A **history of polycystic ovarian syndrome** (PCOS) is not particularly suggestive of MODY. PCOS is associated with insulin resistance and an increased risk for developing type 2 diabetes mellitus. While some forms of MODY can be associated with insulin resistance, it would not be considered the most characteristic feature when trying to differentiate between types of diabetes.

Lack of response to sulphonylureas can be seen in some subtypes of MODY (e.g., MODY 1 and MODY 3). However, it does not necessarily rule out other types or subtypes since there are several different genetic defects that can cause MODY. Furthermore, lack of response to sulphonylureas could also occur in type 2 diabetics who have progressed to needing insulin therapy.

A **history of autoimmune disease** is more suggestive of type 1 diabetes, as it results from an autoimmune process that destroys pancreatic beta cells. While there is no strong association between MODY and autoimmune diseases, a history of autoimmune disease could increase the likelihood of developing other types of diabetes, such as type 1 or Latent Autoimmune Diabetes in Adults (LADA).

Question 68 of 348

A 27-year-old woman who is 11 weeks pregnant comes for review. This is her second pregnancy. During her first pregnancy she was diagnosed with gestational diabetes which resolved following the birth of her son.

What is the most appropriate management at this stage?

A. Perform an oral glucose tolerance test
B. Advise on a diabetic diet and start metformin at 20 weeks
C. Arrange a fasting glucose
D. Arrange a HbA1c test
E. Advise on a diabetic diet and start insulin at 20 weeks

A. Perform an oral glucose tolerance test

The oral glucose tolerance test remains the investigation of choice for gestational diabetes

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A 22-year-old woman attended with symptoms of dysuria and increased frequency. A urine dip was positive for leucocytes, nitrites, and glucose. Capillary blood glucose was 11mmol/L. Ketones were normal. She was treated for a urinary tract infection and returned for repeat urine and capillary blood glucose testing 2 weeks later. Her urine sample was again positive for glucose, but negative for leucocytes and nitrites. Her capillary blood glucose was 13. Ketones were normal.

She has a family history of hypertension and her father has a HNF1A genetic mutation.

Given the likely diagnosis, what is the most appropriate initial treatment for the likely diagnosis?

A. Dietary and lifestyle change
B. Insulin
C. Low-dose gliclazide
D. Metformin
E. No treatment required

C. Low-dose gliclazide

MODY associated with HNF1A often respond well to treatment with low-dose sulfonylureas

Genetic testing would be required to confirm, but the likely diagnosis here is MODY3 which is associated with HNF1A mutations. These mutations are usually inherited in an autosomal dominant pattern. The presentation is often incidental, with recurrent hyperglycaemia without ketosis.

Low dose gliclazide is the correct answer. HNF1A mutations lead to a reduction in the amount of insulin secreted by the pancreas.

Metformin is incorrect. The main mechanism of action of metformin is increased sensitivity to insulin. Gliclazide is the preferred treatment as the HNF1A mutation reduces insulin production by the pancreas. It responds better to insulin secretagogue treatment than an insulinsensitising agent.

Insulin is incorrect. Most patients with MODY do not require insulin as there is generally some insulin secretion from the pancreas particularly in early diagnosis. Treatment with a secretagogue such as gliclazide is therefore preferable.

No treatment required is incorrect. All forms of MODY apart from glucokinase MODY increase the risk of long-term complications of diabetes. Treatment to stabilise and control blood glucose levels is therefore advisable.

Dietary advice and lifestyle changes is an incorrect answer. Although this advice would form part of the management, the main treatment would be pharmacological with a low dose of gliclazide. Unlike in early type-2 diabetes or pre-diabetes where there is a possibility of improving insulin sensitivity by decreasing glucose spikes, MODY is not reversible.

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A 28-year-old woman with well-controlled hypothyroidism on a stable dose of 110 micrograms of thyroxine daily presents to the clinic for a routine check-up. She reports good health overall and has no additional medical issues. She has recently found out she is pregnant, currently in the fourth week of gestation.

What is the most appropriate management advice for this patient?

A. Check TSH before making dose adjustments
B. Continue on the current dose
C. Increase dose to 165 micrograms per day
D. Increase dose to 180 micrograms per day
E. Increase dose to 200 micrograms per day

C. Increase dose to 165 micrograms per day

Increase dose to 165 micrograms per day is the correct answer.

Women with hypothyroidism may need to increase their thyroid hormone replacement dose by up to 50% as early as 4-6 weeks of pregnancy

During pregnancy, there is a physiological increase in thyroid hormone requirements to support maternal health and normal fetal development. Consequently, patients with hypothyroidism often require an adjustment of their levothyroxine dosage by up to 50% to satisfy the increased demands of both mother and fetus. This patient is currently on a daily dose of 110 mcg of levothyroxine.

The recommended increment can be calculated as follows:

- 110 mcg x 50% = 55 mcg.

Therefore, the dose should be increased to a maximum of (110 + 55) = 165 mcg per day, which is the correct answer for this case.

Check TSH before making dose adjustments is incorrect. Rather than waiting for TSH test results, it is necessary to adjust thyroxine dosages during pregnancy to meet the increased requirements. Subsequent monitoring with TSH assays post-adjustment is recommended throughout the pregnancy to ensure a euthyroid state.

Continue on the current dose would not suffice as it fails to address the increased hormonal needs during pregnancy.

Increase dose to 180 micrograms per day is incorrect because this suggests a rise of 70 mcg from her baseline dosage, amounting to a disproportionate increase of approximately 63%, which overshoots standard recommendations.

Increase dose to 200 micrograms per day is also incorrect as it means an excessive enhancement of her baseline dosage by 90 mcg (an 81% increment), far exceeding the advised guidelines for dose escalation during pregnancy.

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A 56-year-old woman presents to endocrinology clinic. She has been referred after CT scanning revealed a 3mm mass in the thyroid. She originally had a CT scan of her neck as she has rheumatoid arthritis and neck pain, and was due to undergo general anesthesia and intubation, and the anesthetist wanted to ensure that her neck was stable. She was due to have a coronary arterial bypass graft. She has a past medical history of type 2 diabetes mellitus, high cholesterol and hypertension. She has no symptoms of overactive or underactive thyroid function.

What is the most appropriate action?

A. Reassure and discharge back to her GP
B. Arrange for US guided FNAC
C. Arrange for nuclear scan
D. Repeat CT in 6 months
E. Refer for urgent hemithyroidectomy

A. Reassure and discharge back to her GP

A non-palpable thyroid mass less than 1cm in size found incidentally without worrying features does not need biopsy

This thyroid mass is not concerning. A patient with a non-palpable nodule which has been found on imaging and is less than 1cm in size does not need biopsy and can be safely managed in primary care. However, if the patient did have a worrying feature such as age outside of 20-60 years old, had rapid growth of mass, vocal cord paralysis or had a history of neck irradiation or thyroid cancer then urgent investigations should be done, primarily a US guided FNAC. Repeating the CT would be inappropriate in terms of the amount of radiation exposure. A diagnostic hemithyroidectomy would be appropriate following a biopsy showing a Thy3a, Thy3f or a Thy4 lesion.

Question 72 of 348

A 42-year-old man presents for review in the endocrinology clinic. He has returned following investigations for hyperthyroidism as requested by the consultant. He originally presented to the emergency department with palpitations and was diagnosed with atrial fibrillations, and further investigations revealed an undetectable TSH with a free T4 of 52ng/dl. He was started on bisoprolol and apixaban since his discharge and has found his palpitations have improved significantly, but he still has them. On examination, he has no obvious neck swelling or tenderness. He returns after having had thyroid-stimulating hormone receptor antibodies tested. These are reported as undetectable.

What is the next appropriate investigation?

A. Trial of carbimazole
B. Scan of radionuclide thyroid uptake
C. US-guided fine needle aspiration cytology
D. CT scan of the neck

E. Holter monitor for 48 hours

B. Scan of radionuclide thyroid uptake

In hyperthyroidism, if TSH antibodies are negative then a radionuclide uptake scan can distinguish between types of hyperthyroidism

This patient has a typical presentation of hyperthyroidism, There is also biochemical evidence of hyperthyroidism, but autoantibodies for Grave's disease are negative. Thyroid-stimulating hormone receptor antibodies (TSH-RAb, commonly shortened to TRAb) has an up to 98% sensitivity and 99% specificity for Graves' disease. If this test is negative, then a radionuclide thyroid uptake scan can help determine the cause. In Graves' disease there is diffuse high uptake, in thyroiditis there is low uptake, and if there are nodules then there is uneven uptake. US-guided fine needle aspiration cytology is recommended in the presence of features suggestive of malignancy. CT scan of the neck would only be appropriate in the case of a large goitre of which the limits would not be noted on examination or ultrasound.

Carbimazole treatment would only be appropriate as a short-term treatment awaiting radio-iodine treatment, in the medium term to induce remission in confirmed Graves' disease, and in the long-term where surgery and radio-iodine treatments are not an option. Whilst awaiting results, it would be more appropriate to up-titrate betablockers to gain symptomatic relief, rather than give carbimazole without a confirmed diagnosis.

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A 33-year-old woman presents to the endocrinology clinic for review. She has a past medical history of Graves' disease which was treated with radioiodine treatment. This finished one year ago. Following this, she has retained a clinically and biochemically euthyroid state. She originally presented with weight loss and insomnia, and these symptoms have not re-occurred. Her GP has requested blood tests prior to the appointment which has shown a TSH of 2.6 and a free T4 of 8.2mg/dl. She wanted to have a review as she has recently stopped taking oral contraceptive tablets and is planning to become pregnant.

What is the most appropriate plan?

A. Advise to avoid pregnancy
B. Measure serum thyroid-stimulating hormone receptor antibodies
C. Arrange for US scan of neck
D. Repeat TSH and free T4 in one year's time
E. Measure thyrotrophin receptor stimulating antibodies

B. Measure serum thyroid-stimulating hormone receptor antibodies

Pregnant woman with a history of Grave's disease should have thyroid stimulating hormone binding antibody titres measured even if euthyroid as the antibodies can cross the placental barrier

This is a patient with a past medical history of Graves' disease who is clinically and biochemically euthyroid who is planning pregnancy. It is important, even with no biochemical evidence of hyperthyroidism, to exclude the serum presence of thyroid-stimulating hormone receptor antibodies as these can cross the placenta and cause foetal problems. If they were positive, then treatment should be initiated to control the antibody levels, despite the normal TSH and T4. Thyrotrophin receptor stimulating antibodies should be checked in hyperthyroidism at 30-36 weeks gestation to help assess the risk of neonatal thyroid problems. There is no reason she should not become pregnant.

Question 74 of 348

A 45-year-old man with headaches returns to the endocrinology clinic following investigations. He originally complained of headaches waking him up at night, low energy, weight loss, low libido and postural dizziness. Investigations have revealed a normal full blood count, low free T4, low testosterone, low morning cortisol, low LH and FSH and low TSH. He also had an MRI which is suspicious for pituitary adenoma. He is keen to start whichever is necessary to feel better. He has no other medical problems and takes no regular tablets.

Which hormonal replacement would be contra-indicated in the immediate setting?

A. Hydrocortisone
B. Prednisolone
C. Dexamethasone
D. Testosterone
E. Levothyroxine

A. Levothyroxine

In hypothyroidism with adrenal failure do not start thyroid hormone replacement before glucocorticoid replacement as otherwise an adrenal crisis can be precipitated

This patient has panhypopituitarism secondary to a pituitary adenoma. It is critical for patients with adrenal and thyroid insufficiency to replace steroid function prior to replacing thyroid function as otherwise an adrenal crisis can be precipitated. Therefore levothyroxine is contraindicated in the immediate setting, even though the patient will need replacement after he has started steroid replacement. Typically he would need hydrocortisone split into three doses to replace his steroid needs, and this is preferred over dexamethasone and prednisolone.

Ultimately the treatment may be trans-sphenoidal surgery.

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A 72-year-old Japanese female presents to the emergency department with sudden onset shortness of breath associated with palpitations. She has previously experienced similar palpitations 6 months ago but did not seek medical attention. She was last completely well and described by her daughter to be at baseline 24 hours ago when they had dinner together. The patient denies any chest pain, nausea, vomiting or sweating. On examination, the patient is pyrexic at 38.2 degrees and tachycardic, with a regular pulse at 130-140 beats per minute. Heart sounds demonstrate a gallop rhythm; auscultation of her chest reveals bibasal inspiratory coarse crackles and no wheeze. She has bilateral mild lower limb pitting oedema to low ankles. Examination of the abdominal and neurological systems is unremarkable. A chest radiograph demonstrates bibasal alveolar shadowing with mild bilateral pleural effusions. An ECG demonstrates sinus tachycardia at 130 beats per minute. Blood tests are as follows:

Hb	123 g/l
Platelets	299 * 10 /l9
WBC	9.5 * 10 /l9
Na +	139 mmol/l
K +	4.2 mmol/l
Urea	7.2 mmol/l
Creatinine	98 µmol/l
TSH	< 0.01 mU/l
Free T4	140 pmol/l
Free T3	40 pmol/l

Nursing staff have kindly taken blood cultures and taken measures to cool the patient.

What is the next most appropriate immediate treatment?

- A. Intravenous propranolol
- B. Lugol's iodine
- C. Oral carbimazole
- D. Oral propylthiouracil
- E. Oral prednisolone

A. Intravenous propranolol

In thyroid storm with IV beta-blockers are a important first-line treatment

The patient has presented with sudden onset heart failure associated with sinus tachycardia, pyrexia and thyrotoxicosis: this represents a thyroid storm and is an endocrinological emergency. The treatment comprises of four aims: resuscitation, treat the sympathetic consequences of thyrotoxicosis, block underlying hyperthyroidism and treat any heart failure present. The first step involved intravenous followed by oral beta-blockade. Diltiazem is an appropriate alternative if the patient cannot tolerate beta blockers. In cases of simultaneous poor ventricular function and thyroid storm, intravenous infusions of short-acting beta blockers such as esmolol are also appropriate, which can be switched off immediately at the earliest sign of worsening cardiac function secondary to beta blockade. Thyroid blockers can be instituted after this immediate therapy. Oral corticosteroids are also important to reduce peripheral T4 to T3 conversion. However, both treatments can be instituted after achieving haemodynamic stability.

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A 28-year-old woman is referred to the endocrinology department with abnormal thyroid function tests and neck pain. She has a past medical history of bipolar affecting disorder and takes lithium. She smokes five cigarettes daily. She is unemployed. She gave birth to her first child six weeks ago and has recently recovered from a coryzal illness. She is not breastfeeding.

On examination, there is a tender swelling in her neck. She is sweaty, mildly tremulous and tachycardia (heart rate 104 beats per minute). Examination of her eyes is unremarkable.

Blood tests:

Hb	136 g/L	Male: (135-180)Female: (115 - 160)
Platelets	189 * 10 /L 9	(150 - 400)
WBC	8.2 * 10 /L 9	(4.0 - 11.0)
Na +	137 mmol/L	(135 - 145)
K +	4.2 mmol/L	(3.5 - 5.0)
Urea	5.2 mmol/L	(2.0 - 7.0)
Creatinine	66 µmol/L	(55 - 120)
CRP	12 mg/L	(< 5)
TSH	0.1 miU/L	(0.2 - 5.5)
Free T4	34 pmol/L	(0 - 24.5)

Additional tests:

Thyroid peroxidase antibodies	negative
Anti TSH receptor antibodies	negative

A radioactive iodine uptake scan reveals globally reduced uptake of iodine-131.

What is the likely diagnosis?

- A. De Quervain's thyroiditis
- B. Factitious hyperthyroidism
- C. Grave's disease
- D. Lithium associated thyroiditis
- E. Post-partum thyroiditis

A. De Quervain's thyroiditis

De Quervain's thyroiditis: initial hyperthyroidism, painful goitre and globally reduced uptake of iodine-131

De Quervain's thyroiditis is the correct answer. This woman presents with painful swelling in her neck (a likely goitre in the context of hyperthyroidism) post-coryzal illness with raised inflammatory markers (CRP), in association with globally reduced uptake of iodine-131. These findings are consistent with viral sub-acute thyroiditis.

Factitious hyperthyroidism is incorrect. Although she has a history of depression (and this is a risk factor for thyroid hormone misuse), this would not explain the raised CRP or neck pain.

Grave's disease is incorrect. This is associated with increased uptake of iodine-131 on a radioactive iodine scan.

Lithium associated thyroiditis is incorrect. This is classically 'painless' thyroiditis.

Post-partum thyroiditis is incorrect. While she is post-partum and this is a plausible differential, this condition is not associated with neck pain or raised inflammatory markers, making this diagnosis unlikely.

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A 24-year-old woman presents to the emergency department with dyspnoea. She has a past medical history of type 1 diabetes. She is poorly compliant with insulin treatment. She does not smoke or drink alcohol and lives with her family.

Her observations are heart rate 90 beats per minute, respiratory rate 24/minute, blood pressure 120/77 mmHg and temperature 36.7ºC.

Arterial blood gas:

pH	7.11	(7.35-7.45)
pO2	14.1 kPa	(11-13)
pCO2	3.4 kPa	(4.5-6)
HCO3	10 mmol/L	(22-26)
Glucose	24 mmol/L	(< 7.8)
Lactate	2.6 mmol/L	(0-2)
Potassium	4.8 mmol/L	(3.5-5.5)
Sodium	136 mmol/L	(135-145)
Blood ketones	3.4 mmol/L	(<0.6)

She is treated with intravenous normal saline with appropriate potassium replacement and a fixed rate insulin infusion (FRII) with normalization of her academia, ketonemia and hyperglycaemia.

What electrolyte abnormality is she at risk of developing during the resolution of this condition?

A. Hypercalcaemia
B. Hyperkalaemia
C. Hypernatraemia
D. Hypermagnesemia
E. Hypophosphataemia

E. Hypophosphataemia

Recovering DKA are at risk of hypophosphataemia

Hypophosphataemia is the correct answer. This patient has diabetic ketoacidosis (elevated blood glucose level, acidaemia and ketonemia). A rise in insulin due to exogenous insulin administration causes an intracellular shift of phosphate. There is an element of phosphaturia in addition.

Hypercalcaemia is incorrect. This has been noted to occur in the development of DKA, likely secondary to severe metabolic acidosis and insulin deficiency but would therefore be not expected to occur as DKA resolves.

Hyperkalaemia is incorrect. Most patients with DKA are potassium deficient and the risk is of hypokalaemia, not hyperkalaemia. Furthermore drives potassium into cells, resulting in hypokalaemia.

Hypermagnesemia is incorrect. Insulin administration causes uptake of magnesium into cells and may cause hypomagnesemia rather than hypermagnesemia.

Hyperphosphataemia is incorrect. It is low rather than high phosphate that is the risk in recovering DKA patients.

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A 9-year-old boy is referred to the department of paediatrics by his general practitioner. He has developed secondary sexual characteristics at the age of 7. He has no significant past medical history and does not take any regular medications. His father commenced puberty at 9 years of age.

On examination, he has a coarse voice and facial hair. His testicles have enlarged. There is acne and adult body odour. The neurological examination is unremarkable. His blood pressure was 155/88 mmHg. There is no rash.

Blood tests:

Hb	136 g/L	Male: (135-180)Female: (115 - 160)
Platelets	388 * 10 /L 9	(150 - 400)
WBC	4.2 * 10 /L 9	(4.0 - 11.0)
Na^+	138 mmol/L	(135 - 145)
K^+	2.9 mmol/L	(3.5 - 5.0)
Urea	4.2 mmol/L	(2.0 - 7.0)
Creatinine	66 µmol/L	(55 - 120)
CRP	4 mg/L	(< 5)
Testosterone	42 ng/dl	(7-20)
FSH	1.2 IU/L	(<3)
LH	1.1 IU/L	(0.02-4.8)
TSH	1.2 mIU/L	(0.5-5.5)

From the listed options, which is the most likely diagnosis?

A. Brain tumour
B. Hypothyroidism
C. Liddle's syndrome
D. McCune-Albright syndrome
E. 11-beta hydroxylase deficiency

E. 11-beta hydroxylase deficiency

CAH due to 11-beta hydroxylase deficiency can cause apparent mineralocorticoid excess syndrome (AMES) resulting in hypertension and hypokalemia

11-beta hydroxylase deficiency is correct. This is the second most common cause of congenital adrenal hyperplasia. While CAH typically presents in the neonatal period, non-classical cases may present later with evidence of precocious puberty, such as in this case. Precocious puberty is when puberty occurs before 9 in males. This patient also has evidence of hypokalemia and hypertension, which can occur in this condition. The elevated serum testosterone but normal LH and FSH suggest a peripheral cause of precocious puberty (in this case adrenal) rather than a central cause.

A brain tumour is incorrect. The LH and FSH are normal. We would expect them to be raised with a central cause of precocious puberty.

Hypothyroidism is incorrect. While this can cause precocious puberty, the TSH is within normal limits.

McCune-Albright syndrome is incorrect. This is a cause of precocious puberty but typically it is associated with cafe-au-lait spots, which are absent here.

Liddle's syndrome is incorrect. This is an inherited renal tubular disorder that may cause hypertension and hypokalemia but it does not cause precocious puberty.

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A 78-year-old man with metastatic lung carcinoma presents with increasing lethargy and a number of falls. He describes feeling very unsteady on standing from his bed. His appetite has been poor and he has vomited two times each day for the past three days. There is no diarrhoea or abdominal pain and he denies any shortness of breath. He takes regular paracetamol for pain and omeprazole for dyspepsia.

On examination, he is a tanned gentleman with cachexia. He appears pale with dry mucosa and his abdomen is soft and non-tender. There is reduced air entry at the right base with bronchial breathing overlying. His heart sounds are normal and his capillary refill time is prolonged to 4 seconds. Bloods pressure is 85/65mmHg, heart rate 86/min, respiratory rate 23/min

Hb	102 g/l	Na +	129 mmol/l
Platelets	189 * 10 /l 9	K +	5.0 mmol/l
WBC	5.6* 10 /l 9	Urea	7.2 mmol/l
Neuts	4.2 * 10 /l 9	Creatinine	87 µmol/l
Lymphs	0.7 * 10 /l 9	CRP	32 mg/l
Eosin	0.1 * 10 /l9		
CT-chestabdomenpelvis	There is a 4cm mass in the right lower lobe with extension to adjacent pleura. Bilateral pleural effusions are present. There is mediastinal lymphadenopathy and enlargement of para-aortic nodes with masses seen in both adrenals. Findings are in keeping with a primary lung malignancy with metastatic spread		

What is the likeliest explanation of this presentation?

A. SIADH
B. Terminal decline
C. Addisonian crisis
D. Proton pump inhibitor side effects
E. Pneumonia

C. Addisonian crisis

Metastatic malignancy can cause Addison's disease

This gentleman presents in a shock like state with poor peripheral perfusion. This could be caused by pneumonia but there are no signs of sepsis and no consolidation on CT. SIADH and PPIs can cause hyponatraemia but not cardiovascular collapse. Terminal decline is the diagnosis of exclusion. This gentleman does have metastatic deposits in both adrenal glands which can impair function and cause Addison's. Furthermore, he has hyponatraemia, hyperkalaemia and hyperpigmentation all which are features seen in Addison's.

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A 66-year-old man presents to his general practitioner with increasing lethargy and confusion. Past medical history includes depression, Barrett's oesophagus, and benign prostatic hyperplasia. Current medications are sertraline 100mg, lansoprazole 30mg BD, and tamsulosin 400mg. Blood tests are as follows:

Hb	104 g/l	Na +	118 mmol/l
Platelets	168* 10 /l 9	K +	4.1 mmol/l
WBC	8.7* 10 /l 9	Urea	7.9mmol/l
Neuts	2.5* 10 /l 9	Creatinine	173 µmol/l
Lymphs	3.0* 10 /l 9	Eosin	0.6 * 10 /l9
Corrected calcium	3.01mmol/l		
Total protein	95g/l	Albumin	30g/l

Urinary sodium 7mmol/l Urinary osmolality 100mOsm/kg Plasma osmolality 280mOsm/kg

What is the most likely cause of this patient's hyponatraemia?

A. Syndrome of inappropriate ADH secretion due to underlying malignancy
B. Multiple myeloma
C. Psychogenic polydipsia
D. Adrenal insufficiency
E. Drug-induced syndrome of inappropriate ADH secretion

B. Multiple myeloma

Hyperlipidaemia can cause pseudohyponatraemia

This patient has pseudohyponatraemia secondary to raised protein levels from an underlying myeloma. Raised protein levels and hyperlipidaemia are both important causes to consider when investigating hyponatraemia. These patients have a normal total sodium but concentrations are skewed due to the high volume of lipids or protein.

Syndrome of inappropriate ADH secretion causes a euvolemic hyponatraemia. Patients have a urinary sodium >20mmol/l with raised urinary osmolality and normal/low plasma osmolality. Psychogenic polydipsia is rare and both urinary and plasma osmolalities are low in these patients. Adrenal insufficiency can cause hyponatraemia but the presence of hypercalcaemia, renal impairment, and raised total in this patient make myeloma the most likely underlying cause.

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A 42-year-old woman is admitted to the emergency department with reduced GCS following a suspected drug overdose. She is a type 2 diabetic and only takes gliclazide. On examination, she is pale and sweaty. As part of her initial assessment, the following result is obtained:

Blood glucose	2.0 mmol/L	(>3.9 mmol/L)

She is given an infusion of 50% dextrose and her glucose is checked again:

Blood glucose	2.5 mmol/L	(>3.9 mmol/L)

What additional treatment is useful in halting the underlying process?

A. Adrenaline
B. Cabergoline
C. Glucagon
D. Noradrenaline
E. Octreotide

A. Octreotide

In sulphonylurea overdoses, if the patient remains hypoglycaemic despite infusion of sufficient glucose, consider administration of octeotride

Sulfonylurea overdose can cause prolonged hypoglycaemia. The drug binds to receptors on the islet β-cells leading to insulin release. This leads to the suppression of endogenous glucose production (gluconeogenesis) and hypoglycaemia. Initial treatment involves infusion of highly concentrated glucose (50% via a large vein); this will correct the hypoglycaemia but acts to further stimulate insulin secretion from the sensitised β-cells. For this reason, patients will be monitored and require glucose infusions for several days. Blood glucose should be maintained around 5-7 mmol/L.

Octreotide is a long-acting synthetic somatostatin analogue that binds to somatostatin-2 receptors on the pancreatic beta cells. It blocks insulin secretion by preventing calcium influx. It can be used when the patient remains hypoglycaemic despite glucose infusion.

Glucagon and may be used to treat severe hypoglycaemia when there is no available IV access. It can also be used in beta-blocker overdose. It is not recommended in the management of sulfonylurea overdose, as it stimulates insulin secretion.

Cabergoline is an ergot derivative and a potent dopamine D,, receptor agonist which can be used to treat hyperprolactinaemia and Parkinson's disease.

Adrenaline and noradrenaline are catecholamines & vasopressors which are used in a variety of emergency situations. They are not useful in this scenario.

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A 45-year-old woman was admitted on the medical take with cardiac sounding chest pain. She had a body mass index of 34 kg/m . She had type 2 diabetes mellitus and hypertension. She was treated for stable angina. On discharge, she was keen to know more about obesity and her potential treatment options.

Which of the following is the best treatment option for her obesity at this stage?

A. Gastric banding
B. Surgical gastric bypass
C. Sibutramine
D. Lifestyle measures (dietary, exercise, and behaviouralinterventions)
E. Orlistat

D. Lifestyle measures (dietary, exercise, and behavioural interventions)

According to NICE guidelines, the first-line treatment for obesity is a combination of dietary, exercise, and lifestyle interventions

Obesity and being overweight are defined as abnormal or excessive fat accumulation that may impair health. Health risks include cardiovascular disease, hypertension, hypercholesterolaemia, diabetes, musculoskeletal problems, respiratory problems (obstructive sleep apnoea and asthma), fatty liver, gallstones, and increased risk of some cancers (colon, breast, and endometrial).

According to NICE guidelines, the first-line treatment is a combination of dietary, exercise, and behavioural interventions; pharmacological therapy is recommended if there is failure of first-line interventions. Orlistat should only be used when more than 3 months' use of dietary and lifestyle measures has failed.

Surgery is recommended if:

- There is a failure of non-surgical measures (no clinically beneficial weight loss over 6 months).
- The patient has a BMI of 40 kg/m or more, or a BMI of 35-40 kg/m and other significant disease that could be improved if they lost weight.

The patient has a BMI greater than 50 kg/m .

The patient commits to long-term follow-up which needs to be done under the supervision of a specialist obesity team.

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A 45-year-old woman is brought into the resuscitation room with a Glasgow coma scale of 11 (E2 V5 M4). A concerned neighbour called the emergency services, who found them in a moribund state. The neighbour states that she had seemed low over the past couple of months and that she had been wearing more layers of clothes than seemed appropriate.

On initial examination, they feel cool to touch. Pulse is regular and bradycardic with a heart rate of 38 beats per minute. Heart sounds 1+2 are present. Respiratory rate is 8 with oxygen saturations of 91% on 15 L. Auscultation of the chest is clear. Temperature is 33ºC. BM is 2.7.

Blood tests return as:

Hb	130 g/L	Male: (135-180)Female: (115 - 160)
Platelets	220 * 10 /L 9	(150 - 400)
WBC	9 * 10 /L 9	(4.0 - 11.0)
Calcium	2.5 mmol/L	(2.1-2.6)
TSH	25 mU/L	(0.5-5.5)
Free T4	0.4 pmol/L	(9.0 - 18)
Creatine kinase	6000 U/L	(35 - 250)
Na +	130 mmol/L	(135 - 145)
K +	4 mmol/L	(3.5 - 5.0)
Bicarbonate	22 mmol/L	(22 - 29)
Urea	8 mmol/L	(2.0 - 7.0)
Creatinine	130 µmol/L	(55 - 120)

What is the most appropriate management of this patient?

A. Intubation and urgent CT head
B. IV hydrocortisone, IV dextrose and rewarming
C. IV T4, IV fluids, IV dextrose and intubation
D. IV fluid resuscitation, IV dextrose, intubation and IV T4
E. IV hydrocortisone, IV T4, IV dextrose and IV fluids

E. IV hydrocortisone, IV T4, IV dextrose and IV fluids

If myxedema coma is suspected, IV corticosteroids should be given alongside IV thyroid replacement until coexisting adrenal insufficiency has been excluded

The most appropriate answer is 5 as they need treatment of the underlying cause as well as correction of life-threatening abnormalities. This patient is suffering from severe hypothyroidism and is in a myxedema coma. This is best treated with IV hydrocortisone and thyroxine in case there is co-existing adrenal insufficiency. Their blood sugars are low necessitating correction and there is a raised creatine kinase requiring management with IV fluids.

CT head and intubation is appropriate but is not the best option of those above. The other answers do no include thyroxine and hydrocortisone as a combined answer so, although an aspect of the overall management of the patient, are not the most pressing intervention.

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A 52-year-old woman presents with a two-day history of nausea and fever. On admission she is confused and her husband states that she was recovering from a recent upper respiratory tract infection and sore throat. He also mentions she has previously been experiencing episodes of diarrhoea and palpitations over the last three months.

Examination reveals a temperature of 40.6ºC, pulse rate of 160 beats per minute and blood pressure of 110/70 mmHg. Her pulse is irregularly irregular. Heart sounds 1 and 2 are present with no added sounds, lung fields are clear and her abdomen is soft and none tender, with bowel sounds being present.

Blood tests are taken and reveal:

Hb	13.2 g/dL
Platelets	180 * 10 /l9
WBC	10.2 * 10 /l9
Na +	135 mmol/l
K +	4.2 mmol/l
Urea	7.2 mmol/l
Creatinine	132 µmol/l
TSH	0.03 mu/l
Free T4	31 pmol/l
Total thyroxine (T4)	220 nmol/l

What is the most appropriate immediate treatment?

A. Propylthiouracil, corticosteroids and propranolol
B. Propylthiouracil and propranolol
C. Radio-iodine, corticosteroids and propranolol
D. Propylthiouracil and corticosteroids
E. Radio-iodine, propranolol and carbimazole

A. Propylthiouracil, corticosteroids and propranolol

Thyrotoxic storm is treated with beta blockers, propylthiouracil and hydrocortisone

This patient is having a thyrotoxic storm (hyperthyroid crisis) a rare medical emergency that is caused by an exacerbation of hyperthyroidism and characterised by decompensation of one or more organ systems in people with untreated or poorly treated hyperthyroidism. The precipitating cause is most commonly infection, as with this case, although it is important to check for other causes. The patient above is in atrial fibrillation and shows signs of renal impairment due to dehydration. First line treatment for this medical emergency is propylthiouracil, corticosteroids and propranolol, although chlorpromazine can be added for severe anxiety.

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A 41-year-old woman is admitted to hospital with acute epigastric abdominal pain that radiates to her back. She has nausea but has not vomited. A diagnosis of acute pancreatitis is suspected and she is commenced on intravenous fluids. Her observations include a blood pressure of 129/72 mmHg, pulse of 88 bpm, and oxygen sats of 97%.

Blood tests are performed and reveal:

Hb	13.9 g/l
Platelets	194 * 10 /l9
WBC	8.6 * 10 /l9
Na +	139 mmol/l
K +	4.2 mmol/l
Urea	4.1 mmol/l
Creatinine	92 µmol/l
Bilirubin	10 µmol/l
ALP	39 u/l
ALT	34 u/l
γGT	44 u/l
Albumin	48 g/l
Triglycerides	12.1 mmol/l
HDL cholesterol	1.1 mmol/l
LDL cholesterol	3.5 mmol/l

What is the most appropriate treatment for this patients condition?

A. Atorvastatin
B. Fenofibrate
C. Lovastatin
D. Ezetimibe
E. Alirocumab

B. Fenofibrate

Fibrates are the most effective drug for treating hypertriglyceridaemia

The diagnosis is hypertriglyceridaemia, which has caused this patients acute pancreatitis. Fibrates are the treatment for hypertriglyceridaemia at high enough levels to cause acute pancreatitis.

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A 64-year-old man has been admitted with hypercalcaemia on a background of metastatic prostate cancer. A recent CT of the chest, abdomen, and pelvis confirmed multiple bony, sclerotic metastases in the lumbar spine and pelvis.

IV fluids have been given since admission. IV pamidronate 60mg was given 3 days ago and IV denosumab 120mg was given yesterday.

A trend of adjusted calcium is shown below.

Day 1	Day 2	Day 3	Today	Range	
Adjusted calcium	3.32 mmol/L	3.26 mmol/L	3.24 mmol/L	3.16 mmol/L	(2.1-2.6)

The renal function today is shown below.

Na +	126 mmol/L	(135 - 145)
K +	3.7 mmol/L	(3.5 - 5.0)
Bicarbonate	23 mmol/L	(22 - 29)
Urea	8.9 mmol/L	(2.0 - 7.0)
Creatinine	132 µmol/L	(55 - 120)

What is the most appropriate next step in management?

A. Calcitonin
B. Further dose of denosumab
C. Further dose of pamidronate
D. Indapamide
E. Prednisolone

A. Calcitonin

Refractory hypercalcaemia of malignancy may be treated with subcutaneous calcitonin if therapy with fluids and pamidronate fails

Calcitonin is the correct answer. Calcitonin has been found to be most useful when combined with hydration, bisphosphonates and/or denosumab, all of which the above patient has received. Calcitonin acts by increasing the excretion of renal calcium and decreasing bone resorption.

Denosumab is commonly used in malignancy-associated hypercalcaemia and can be used in the emergency management of refractory hypercalcaemia. Denosumab can be repeated weekly, therefore given that it was given only the day previously, it is not appropriate to give a **further dose of denosumab**.

A **further dose of pamidronate** is inappropriate as the last dose was given 3 days ago. Bisphosphonates take 2-3 days to see an effect and maximal effect may not show until the 7th day.

Loops diuretics can be used in hypercalcaemia, however, thiazide-like diuretics, such as **indapamide**, are contraindicated as they can worsen hypercalcaemia.

Steroids, such as **prednisolone**, are not beneficial for hypercalcaemia unless associated with sarcoidosis.

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A 29-year-old nulligravida woman attends her GP as she has not menstruated for 6 months. Menarche was at age 14. She had irregular periods from age 14-16, and then took oral hormonal contraception until last year when she got married. The patient does not know her family history as she is adopted. She is otherwise fit and well, enjoying running recreationally and eating healthy. She does not drink, smoke or use illicit drugs. Her vital signs are normal and body mass index is 22 kg/m².

Physical examination shows no abnormalities. Urine pregnancy test is negative.

Which of the following is the most appropriate* screening *test for this patient?

A. Karyotype
B. MRI of the pituitary
C. Serum 17-hydroxyprogesterone
D. Serum prolactin
E. Ultrasound of the pelvis

D. Serum prolactin

The patient's presentation is consistent with secondary amenorrhoea. The first investigative step is normally to do a pregnancy test, which in this case is negative. There are no obvious explanations for secondary amenorrhoea from her presentation. Hence the next step is to obtain serum prolactin, TSH and FSH blood results (as this will help differentiate between the most common causes of secondary amenorrhoea following pregnancy: hyperprolactinaemia, thyroid dysfunction and premature ovarian failure respectively).

Choice 1: Premature ovarian insufficiency is characterised by elevated FSH and low oestradiol. A karyotype may be considered if Turner syndrome or fragile X syndrome is suspected, as these patients usually have ovarian dysfunction. However, this patient has no dysmorphic features or other co-morbidities to warrant karyotype evaluation.

Choice 2: An MRI of the pituitary should be performed in patients with persistently elevated prolactin (with or without galactorrhoea), unexplained headaches, bitemporal hemianopia or other concerning neurologic findings. It is not an appropriate screening test in this patient.

Choice 3: Serum 17-hydroxyprogesterone is elevated in non-classic congenital adrenal hyperplasia due to 21-hydroxylase deficiency, a condition that presents with hyperandrogenism in late childhood. This patient has no findings of androgen excess (e.g. male pattern hair distribution, severe acne).

Choice 5: An ultrasound may be indicated in patients with abnormal pelvic pain, pelvic organ enlargement or masses on examination, or signs of hyperandrogenism (e.g. polycystic ovarian syndrome, androgen tumour), none of which are seen in this patient.

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An 85-year-old woman is brought into A+E with hypothermia, sinus bradycardia and unresponsive. A CT head reveals no acute intra-cranial pathology. Passive warming and intravenous fluids are commenced. Subsequent blood tests reveal the panel below. A collateral history from family members reveals symptoms of lethargy, cold intolerance and weight gain over the last few months.

TSH	>30.0 mU/L
T3	<0.05 mU/L

What is the most appropriate initial treatment?

A. Levothyroxine and Liothyronine
B. Levothyroxine
C. Lugol's iodine
D. Liothyronine
E. Carbimazole

A. Levothyroxine and Liothyronine

This is myxedema coma, which is rare but has a high mortality rate. Administration of both liothyronine (T3) and levothyroxine (T4) is the most appropriate treatment. Liothyronine (T3) has a greater biologic activity and faster onset than levothyroxine (T4) and should be continued until there is clinical improvement. The administration of hydrocortisone is also important as patients there may be coexisting adrenal insufficiency (primary or secondary).

Supportive measures are important such as re-warming, fluid resuscitation, electrolyte correction and vasopressors if required.

Carbimazole is used for hyperthyroidism.

Lugols iodine is used for rapid control of hyperthyroidism such as a thyroid storm or prior to a total thyroidectomy for poor medical compliance.

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A 62-year-old woman attends her GP complaining of weight gain, lethargy and hair loss. She denies any intercurrent illness. Thyroid function tests are performed and the results are as follows:

TSH	0.3 mu/l
Free T4	8 pmol/l

Which investigation is most likely to be diagnostic?

A. Thyroid ultrasound
B. Radio-iodine uptake scan
C. Anti-thyroid peroxidase (TPO) antibodies
D. Fine-needle aspiration of thyroid
E. MRI pituitary gland

E. MRI pituitary gland

This patient has hypothyroidism. The vast majority of cases are primary hypothyroidism with a high TSH and low T4. The common causes are:

- Autoimmune (Hashimoto's disease, atrophic)
- Iodine deficiency
- Thyroiditis (post-viral, post-partum)
- Iatrogenic (thyroidectomy, radioiodine, drugs)

Secondary hypothyroidism is very rare and results in a low TSH and low T4. In these cases, pituitary insufficiency is most likely and therefore an MRI of the gland should be performed.

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A 30-year-old male with background of type one diabetes mellitus presents with abdominal pain and shortness of breath. Investigations confirm he has diabetic ketoacidosis.

Which one of the following investigations would suggest a discussion for possible intensive care admission?

A. Lactate 3 mmol/L
B. Bicarbonate level 19 mmol/L
C. pH 7.27
D. White cell count 30 x 10^9/L
E. Potassium 3.4 mmol/L

E. Potassium 3.4 mmol/L

Parameters indicate severe diabetic ketoacidosis:

- pH < 7
- Blood ketone > 6 mmol/L
- Bicarbonate < 5 mmol/L
- Anion gap >16 mmol/l
- Potassium < 3.5 mmol/L on admission
- Tachycardia or bradycardia
- Systolic blood pressure <90 mmHg
- Oxygen saturation <92% on air
- GCS < 12

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A 62-year-old man comes to the Emergency department with nausea and vomiting which has steadily worsened over the past 2-3 weeks. He had Type 2 diabetes for the past 7 years and is currently treated with metformin, sitagliptin and empagliflozin. He tells you he has lost some 5kg in weight over the past month. On examination his blood pressure is 110/65 mmHg, his pulse is 85 beats per minute and regular. Emergency blood testing reveal elevated ketones and a glucose of 12.2 mmol/l.

Which of the following is the most appropriate way to manage his glucose control?

A. Add liraglutide
B. Add long-acting insulin
C. Change the empagliflozin for liraglutide
D. Change the empagliflozin for long-acting insulin
E. Stop the metformin

D. Change the empagliflozin for long-acting insulin

Given the duration of Type 2 diabetes and the fact that patient has lost weight in the past month, the possibility that he is insulinopenic is raised. In this situation, calorie loss and metabolic disturbance can be exacerbated by the use of SGLT-2 inhibitors and patients may present as here, with euglycaemic ketoacidosis. The SGLT-2 inhibitor should be withdrawn, and given he is insulinopaenic, long-acting insulin added.

In this situation the empagliflozin must be withdrawn, therefore options including adding liraglutide and long-acting insulin are incorrect. GLP-1 agonists such as liraglutide work less well in patients who are relatively insulinopenic, so liraglutide is incorrect. Stopping the metformin won't remove the cause of ketosis, the empagliflozin.

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A 28-year-old pregnant woman at eight weeks gestation with a history of type 1 diabetes mellitus attends a routine check-up. She takes a basal/bolus insulin regimen and reports no symptoms of hypoglycaemia. On examination, her blood pressure is 110/80 mmHg, pulse rate is 78 beats per minute, and body mass index (BMI) is 25 kg/m². Her latest glycated haemoglobin (HbA1c) level is 45 mmol/mol. The results of her investigations are as follows:

Hb	120 g/L	Female: (115 - 160)
Platelets	290 * 10 /L 9	(150 - 400)
WBC	8.5 * 10 /L 9	(4.0 - 11.0)
Urea	5.1 mmol/L	(2.0 - 7.0)
Creatinine	98 µmol/L	(55 - 120)
ALT	38 u/L	(3 - 40)
Albumin	41 g/L	(35 - 50)

What is the most appropriate advice for this patient?

A. Add vildagliptin
B. Folic acid supplementation 0.5 mg/day
C. Increased frequency of retinal screening
D. Refer for detailed anomaly scan
E. Weight loss

C. Increased frequency of retinal screening

Patients with diabetes should have increased frequency of retinal screening during pregnancy due to increased risk of retinopathy

Increased frequency of retinal screening is the correct answer. Women who are pregnant with pre-existing diabetes are at an elevated risk of worsening retinopathy; therefore, they require more frequent retinal screenings. These screenings must be conducted alongside meticulous glycaemic control to mitigate the risk of complications.

Add vildagliptin is incorrect. The patient's HbA1C level is below 48 mmol/mol, which falls within the target range during pregnancy, thus negating the necessity for additional pharmacotherapy. Furthermore, metformin is the only oral anti-diabetic medication recommended during pregnancy alongside insulin; other oral hypoglycaemic agents should be discontinued.

Folic acid supplementation 0.5 mg/day is not advised as per current guidelines; a higher dosage of 5mg/day until the 12th week of gestation is typically prescribed to ensure adequate folate levels. This higher dose is particularly important in pregnant women with pre-existing diabetes to reduce the risk of neural tube defects.

Refer for detailed anomaly scan is customarily performed at around 20 weeks of gestation. At eight weeks of gestation, conducting this type of scan would be premature.

Weight loss advice is generally reserved for women with a BMI exceeding 27 kg/m^2. Given that this patient's BMI is 25 kg/m^2, recommending weight loss would be inappropriate.

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A 19-year-old gentleman with a background history of asthma presents to the Emergency Department complaining of leg weakness and the inability to walk. He had run a marathon the day before. On examination, there is 3/5 weakness of the leg extensors bilaterally. Tone, reflexes and coordination are unimpaired and plantars are downgoing bilaterally. Straight leg raise and sensation to light touch and pain stimulus are unimpaired.

Blood tests show the following:

Hb	13.4g/dl
WBC	6.2 x 10 /l9
Na +	136mmol/l
K +	2.9mmol/l
Urea	6.8mmol/l
Creatinine	104µmol/l

What is the most appropriate treatment in this case?

A. Oral potassium and encourage bed rest
B. Hourly forced vital capacity measurements and plasma exchange
C. Oral potassium and encourage gentle exercise
D. Hourly forced vital capacity measurements and IV immunoglobulin
E. Plasma exchange and oral potassium supplementation

C. Oral potassium and encourage gentle exercise

Hypokalaemic periodic paralysis is a rare autosomal dominant periodic paralysis but can also occur as a result of a spontaneous mutation (a third of cases have no family history). Attacks often occur in the morning with a history of strenuous exercise or a high carbohydrate meal the day before or may be provoked by stress eg. infections, lack of sleep.

Weakness can range from an isolated muscle group to generalised weakness and tends to affect proximal muscles first.

Serum potassium decreases during attacks but may not necessarily fall below the normal range (3.5 -5mmol/l).

The mainstay of treatment for an acute attack is oral potassium supplementation and encouragement of gentle exercise. Intravenous potassium is reserved for those unable to swallow or with cardiac arrhythmias. Acetazolamide or dichlorphenamide are used as first-line prophylactic agents.

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A 58-year-old man presents with a two-month history of weight loss and a one-week history of increasing confusion. His partner reports that his clothes are now loose on him and that he has started to forget things and that he has been unable to reach for objects off the top shelf at the supermarket over the last two months due to increasing weakness. Six weeks ago he had been treated for an islet cell carcinoma of the pancreas with chemotherapy and has no other past medical history.

Examination reveals an abbreviated mental test score of 5/10 and weakness in the shoulders and getting out of the chair. Heart sounds 1 and 2 are present with no added sounds, his chest is clear and the abdomen is soft and non-tender.

Observations reveal a blood pressure of 158/95 mmHg, a pulse rate of 90 beats per minute, a temperature of 37.5ºC and a respiratory rate of 14 breaths per minute. Random blood glucose is 16.2 mmol/L.

Blood tests are performed and reveal:

Hb	14.2 g/l
Platelets	180 * 10 /l9
WBC	4.9 * 10 /l9
Na +	150 mmol/l
K +	2.6 mmol/l
Urea	5.2 mmol/l
Creatinine	100 µmol/l
Bilirubin	15 µmol/l
ALP	70 u/l
ALT	28 u/l
γGT	47 u/l
Albumin	48 g/l

What is the most likely diagnosis?

A. Paraneoplastic encephalitis
B. Cerebral metastases
C. Post chemotherapy Cushing's syndrome
D. Post chemotherapy hypothyroidism
E. Ectopic ACTH secretion

E. Ectopic ACTH secretion

The confusion, hypertension and proximal myopathy, along with the hypernatraemia, hypokalaemia and hyperglycaemia all point towards a diagnosis of Cushing's syndrome. The subtype is most likely ectopic secretion of ACTH by the islet cell carcinoma, a neuroendocrine tumour and can release ectopic hormones. The post-chemotherapy Cushing's syndrome is unlikely, as the chemotherapy started after the proximal myopathy had begun to take effect. Further, in ectopic ACTH secretion, the hypokalaemia tends to be more pronounced, as in this case.

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A 65-year-old man is reviewed in clinic. He has a past medical history of an NSTEMI, hypercholesterolaemia, hypertension and depression. He is euvolaemic on examination. His drug history includes sertraline, bisoprolol, ramipril and furosemide.

Blood results are follows:

Hb	138 g/l	Na^+	126 mmol/l
Platelets	440 * 10 /l 9	K^+	3.8 mmol/l
WBC	10.8 * 10 /l 9	Urea	7.2 mmol/l
Glucose	6.5 mmol/l	Creatinine	86 µmol/l
Total cholesterol	6.5 * 10 /l 9	Triglycerides (fasting)	12.5 mmol/L (normal < 1.7)

You perform a paired serum and urine osmolarity:

Serum osmolarity	290 mOsmol/kg (normal 275-295)
Urine osmolarity	600 mOsmol/kg
Urine sodium	40 mmol/l

What is the most likely cause of the hyponatraemia?

A. SIADH
B. Dilutional hyponatraemia secondary to heart failure
C. Pseudohyponatraemia
D. Furosemide
E. Hypothyroidism

C. Pseudohyponatraemia

Pseudohyponatraemia is characterised by a normal measured serum osmolarity, however the calculated osmolarity (based on an erroneously low plasma sodium result) is reduced. This results in a raised osmolar gap

Identifying the cause of hyponatraemia can be challenging. The first step is to confirm that it is a true hypotonic hyponatraemia. This is done by measuring the plasma osmolarity.

- If the measured plasma osmolarity is low this confirms true hypotonic hyponatraemia.
- If the plasma osmolarity is normal then this is suggestive of pseudohyponatraemia and should prompt you to measure proteins and lipids, which if present in high levels, can cause a pseudohyponatraemia due to the measuring technique.
- If the serum osmolarity is high then this confirms hypertonic hyponatraemia and should prompt you to check for high levels of solutes in the plasma, hyperglycaemia being the most common.

In this case, the normal measured serum osmolarity suggests pseudohyponatraemia. Pseudohyponatraemia occurs due to a measuring technique defect. Using standard techniques, serum sodium is measured as a ratio of sodium to plasma volume. If the patients plasma has high amounts of proteins or lipids, the plasma volume will be increased resulting in a measured hyponatraemia. This is not a true hyponatraemia as the actual ratio of sodium to plasma fluid will be normal. Common causes include hyperproteinaemia (e.g. TPN, IVIG) and hyperlipidaemia (in particular hypertriglyceridemia).

In this case, the measured serum osmolarity can be compared with the calculated serum osmolarity. The calculated serum osmolarity = 2Na + 2K + glucose + urea = 273.3 mOsmol/kg. The osmolar gap = measured osmolarity - calculated osmolarity = 290 - 273.3 = 16.7 (normal <10). A raised osmolar gap is suggestive of the presence of other osmotically active particles. In this case it is due to the presence of a significantly raised triglyceride level.

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A 75-year-old man is admitted onto the ward with confusion. He has a past medical history of an ischaemic stroke 3 months ago. His drug history includes atorvastatin, ramipril and clopidogrel. He lives alone and has no package of care. On examination he has very poor mobility. His heart rate is 105 bpm and blood pressure 105/75 mmHg. His JVP is not visible.

Blood results are as follows:

Hb	138 g/l	Na^+	155 mmol/l
Platelets	340 * 10 /l 9	K^+	4.9 mmol/l
WBC	12.2 * 10 /l 9	Urea	12.6 mmol/l (6.2 the previous month)
Neuts	8.4 * 10 /l 9	Creatinine	115 µmol/l (58 the previous month)

What is the most likely cause of the hypernatraemia?

A. Osmotic diuresis
B. Diabetes insipidus
C. Reduced water intake
D. Hyperaldosteronism
E. SIADH

C. Reduced water intake

Hypernatraemia associated with hypovolaemia occurs due to a free water deficit. Common causes include reduced water intake (e.g. elderly), GI losses (e.g. vomiting and diarrhoea), skin losses (e.g. burns), and renal losses (e.g. osmotic diuresis)

The patient is clinically and biochemically dehydrated. We are therefore dealing with a case of hypovolaemic hypernatraemia.

Hypernatraemia occurs most commonly due to

- Insufficient water (primarily in patients who either do not experience thirst normally, or cannot act on it).
- Hypotonic fluid loss (e.g. water loss in excess of salt loss) via either the skin, gut or kidneys
- Salt overload e.g. acute salt poisoning (hypertonic saline, hypertonic sodium bicarbonate), hyperaldosteronism

In an elderly person, with decreased mobility, the most likely explanation would be due to reduced water intake.

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A 75-year-old lady presents to the acute medical unit. She is referred by her GP who is concerned that she has been exposed to the chicken pox virus. Her grandson currently has a generalised vesicular rash and she had been in contact with him earlier that day. Her past medical history includes heart failure and giant cell arteritis. Her current medications include prednisolone, ramipril and bisoprolol. She has been taking prednisolone 40mg for the past two weeks.

How will you manage this patient?

A. Intravenous ganciclovir
B. Intravenous aciclovir
C. Oral aciclovir
D. Check Varicella Zoster antibodies
E. Varicella Zoster immunoglobulin

D. Check Varicella Zoster antibodies

Any patient who is taking 40 mg or more of prednisiolone for more than 7 days or who is taking 20 mg or more of prednisolone for more than 14 days is classed as immunocompromised

Varicella Zoster immunoglobulin (VZIG) prophylaxis is recommended for individuals who fulfil all of the following three criteria:

1. Significant exposure to chickenpox or herpes zoster
2. A clinical condition that increases the risk of severe varicella; this includes immunosuppressed patients, neonates and pregnant women
3. No antibodies to Varicella Zoster (VZ) virus

Any patient who is taking 40 mg or more of prednisolone for more than 7 days or who is taking 20 mg or more of prednisolone for more than 14 days is classed as immunocompromised.

This patient is therefore immunosuppressed and has been exposed to the VZ virus. Antibodies to VZ virus should, therefore, be checked so that VZ immunoglobulin can be administered if the patient is seronegative.

Primary varicella zoster virus (VZV) infection or chickenpox is typically a benign illness in immunocompetent individuals. However, the risk of life-threatening complications, such as disseminated VZV and pneumonitis, is greater in immunosuppressed individuals, with significant rates of mortality.

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A 22-year-old female presents with nausea. She states that it has been going on for approximately 10 weeks. On systemic enquiry she also complains of constipation and fatigue. She has no past medical history and takes no regular medicines.

Blood results are as follows:

Hb	110 g/l	Na +	142 mmol/l
Platelets	422 * 10 /l 9	K +	3.8 mmol/l
WBC	8.2 * 10 /l 9	Urea	6.2 mmol/l
Neuts	5.8 * 10 /l 9	Creatinine	52 µmol/l
Lymphs	2.8 * 10 /l 9	CRP	4 mg/l

Thyroid function tests are also performed:

TSH	2.2 mIU/L (normal 0.4 - 4.0)
Total T3	3.9 nmol/l (normal 1.1-2.7)
Total T4	190 nmol/l (normal 60-160)
Free T3	3.6 pmol/l (normal 1.2 - 8.2)
Free T4	16 nmol/l (normal 11.7-31.2)

What investigation will you perform next?

A. Thyroid ultrasound
B. Thyroid peroxidase antibody levels
C. Thyroglobulin levels
D. Pituitary function tests
E. Pregnancy test

E. Pregnancy test

Raised total T3 and T4 but normal fT3 and fT4 suggest high concentrations of thyroid binding globulin, which can be seen during pregnancy

The patient has raised total T3 and T4 but normal fT3 and fT4 suggest high concentrations of thyroid binding globulin. This pattern of results is commonly seen during pregnancy. Her symptoms could be attributed to hypothyroidism, however equally they are common symptoms in pregnancy. She is a female of child rearing age and therefore a pregnancy test should be performed.

Thyroid peroxidase antibody and thyroglobulin levels should be measures in suspected autoimmune hypothyroidism. The normal TSH, fT3 and fT4 makes autoimmune hypothyroidism very unlikely.

There is nothing biochemically or clinically to suggest pituitary disease.

A thyroid ultrasound would be indicated in the presence of a thyroid nodule.

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You are asked to review a patient with hyperglycaemia on the ward. The patient is a 58-year-old female with type 1 diabetes mellitus. The nurse states that her capillary blood glucose is 12.8 mmol/l. Her drug history includes Humulin M3 (28 units before breakfast, and 38 units before evening meal). She had her normal insulin with breakfast at 07:30.

You review her at 11:30. The patient denies polyuria or polydipsia. She is clinically euvolaemic. She is medically stable and awaiting discharge from physiotherapy. You ask the nurse to check plasma ketones which return at 0.4mmol/l. On review of her insulin chart, you note that her blood glucose levels are usually well controlled.

How will you manage this patient?

A. 6 units of actrapid
B. Reassurance to the patient and nurse and continue monitoring
C. Intravenous insulin sliding scale
D. Diabetic ketoacidosis protocol
E. Increase Humulin M3 to 32 units before breakfast starting thefollowing day

B. Reassurance to the patient and nurse and continue monitoring

When managing hyperglycaemia in an insulin dependent diabetic, avoid the use of stat insulin, unless the patient is symptomatic or has high ketones (>0.6 mmol/l). It is safer to increase the 'usual' insulin if persistently hyperglycaemic

The patient has had a once off 'blip' in blood glucose control. When managing hyperglycaemia in an insulin dependent diabetic, you should avoid the use of stat insulin, unless the patient is symptomatic or has high ketones (>0.6 mmol/l). It is far safer to increase the 'usual' insulin if persistently hyperglycaemic.

The patient is medically stable, asymptomatic, and the ketones are at a satisfactory level. Therefore the patient and nurse should be reassured, and the blood glucose level monitored.

If the patient had frequent high blood sugars then it would be appropriate to titrate the normal insulin up.

An intravenous insulin sliding scale is indicated when the patient is undergoing fasting and will be missing more than one meal.

The diabetic ketoacidosis (DKA) protocol should be implemented in patients with DKA which is diagnosed when ketones are significantly elevated (> 3 mmol/l) and the patient is acidotic (HCO3 < 15).

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A 25-year-old female presents with galactorrhoea. She reports no other symptoms on systematic enquiry. She has no past medical history and takes no regular medicines.

Blood results are as follows:

FSH	0.2 mIU/ml (normal 1 - 8)	LH	0.5 mIU/ml (normal 1- 18)
Oestradiol	482 pg/ml (normal 27 - 123)	Progesterone	46 ng/mL (normal 5 -20)
Prolactin	82 ng/dL (normal 5 - 40)		

What investigation will you perform?

A. MRI pituitary
B. Thyroid function tests
C. Pregnancy test
D. Pelvic ultrasound
E. No further investigations

C. Pregnancy test

Pregnancy is an important differential to consider when assessing a female patient with hyperprolactinaemia

The hormone results are entirely consistent with pregnancy. Oestradiol and progesterone are secreted from the corpus luteum in early pregnancy. At approximately the third month of gestation, the placenta takes over as the source of oestrogen and progesterone. High levels of these hormones results in suppression of LH and FSH. During pregnancy, prolactin levels also increase. Prolactin causes enlargement of the mammary glands and prepares for milk production.

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A 17-year-old woman presents to the clinic with amenorrhea. She advises you that she has never had a menstrual cycle. On examination, you note little axillary and pubic hair. There are also bilateral groin swellings. Her vagina and labia have a normal appearance.

Blood results are as follows:

Testosterone	11.2 nmol/L	(< 1.8)
FSH	16 IU/L	(1.8 - 22.5)
LH	120 IU/L	(1.2-103)

What is the most likely diagnosis?

A. Complete androgen insensitivity syndrome (CAIS)
B. Kallmann's syndrome
C. Mullerian agenesis
D. Partial androgen insensitivity syndrome (PAIS)
E. Turner's syndrome

A. Complete androgen insensitivity syndrome (CAIS)

Primary amenorrhoea, little or no axillary and pubic hair, elevated testosterone → androgen insensitivity syndrome

Complete androgen insensitivity syndrome (CAIS) is correct. The clinical features of primary amenorrhea, lack of secondary sexual characteristics, and groin swellings (suggestive of undescended testes) are suggestive of androgen insensitivity syndrome. The hormonal profile findings of very high testosterone, elevated LH, and a relatively normal FSH are classical findings of partial androgen insensitivity syndrome. The presence of a normal vagina and labia favour complete rather than partial androgen insensitivity syndrome.

Kallmann's syndrome is incorrect. Kallmann's syndrome is a recognised cause of delayed puberty secondary to hypogonadotropic hypogonadism. Cryptorchidism can also occur. However, the biochemical profile, in this case, is not in keeping with Kallmann's syndrome - which would present with low sex hormone levels, and inappropriately low/normal LH, and FSH levels.

Mullerian agenesis is incorrect. Mullerian agenesis is a congenital malformation characterised by a failure of the Mullerian ducts to develop, resulting in a missing uterus and variable degrees of vaginal hypoplasia of its upper portion. Both Mullerian agenesis and androgen insensitivity syndrome can present with primary amenorrhea, shortened vagina, and absent cervix. However, in patients with androgen insensitivity, the gonads are testes, which produce normal androgens. The presence of bilateral groin swellings (indicative of undescended testes) makes Mullerian agenesis a less likely diagnosis.

Partial androgen insensitivity syndrome (PAIS) is incorrect. The clinical and biochemical features are suggestive of androgen insensitivity syndrome. However the presence of a normal vagina and labia favour complete rather than partial androgen insensitivity syndrome. PAIS results in a spectrum of disorders that ranges from a female phenotype with clitoromegaly and/or minimal posterior labial fusion (Lub syndrome) to genital ambiguity to an unequivocally male phenotype with a minor defect such as isolated hypospadias or azoospermia.

Turner's syndrome is incorrect. Turner's syndrome can cause primary ovarian failure due to gonadal dysgenesis. However, the biochemical profile, in this case, is not in keeping with Turner's syndrome - which would present with low sex hormone levels, and markedly raised LH and FSH levels.

Question 102 of 348

A 22-year-old woman attends the clinic with a 12-month history of infertility and irregular periods. She also complains of excess hair growth over her face and chest. Observations are as follows: heart rate 82 beats per minute, blood pressure 115/75 mmHg, respiratory rate 16 breaths per minute, SpO2 98% (on air), temperature 37.2ºC.

Blood results are as follows:

Hb	125 g/L	Male: (135-180)Female: (115 - 160)
Platelets	228 * 10 /L $_9$	(150 - 400)
WBC	8.2 * 10 /L $_9$	(4.0 - 11.0)
Na $^+$	132 mmol/L	(135 - 145)
K $^+$	5.1 mmol/L	(3.5 - 5.0)
Urea	6.2 mmol/L	(2.0 - 7.0)
Creatinine	74 µmol/L	(55 - 120)
CRP	2 mg/L	(< 5)
Glucose	5.2 mmol/L	(4-7)
Early morning cortisol	140 nmol/l	(>350)

What investigation is required?

A. ACTH stimulation testing
B. Dexamethasone suppression test
C. Karyotyping
D. Serum prolactin
E. Transvaginal ultrasound

A. ACTH stimulation testing

ACTH stimulation testing may be used to diagnose congenital adrenal hyperplasia

ACTH stimulation testing is correct. The clinical and biochemical features suggest non-classic congenital adrenal hyperplasia (CAH). This form is milder, more common, and often diagnosed in later childhood or early adulthood. Usually, women with non-classic CAH present with signs of excess androgens (i.e., excess body hair, early puberty, irregular periods). Hypoaldosteronism (e.g. hyponatraemia, hyperkalaemia, metabolic acidosis) can also occur. The biochemical features and low early morning cortisol in this case are highly suggestive of hypoaldosteronism.

Dexamethasone suppression test is incorrect. Although Cushing's disease remains within the differential diagnosis, there are no other clinical (e.g. hypertension, proximal myopathy, weight gain, easy bruising) or biochemical features (e.g. hyperglycaemia, hypernatraemia, hypokalaemia) to support this diagnosis. Furthermore, the low early morning cortisol makes this an unlikely diagnosis.

Karyotyping is incorrect. Although Turner's syndrome can be associated with hirsutism and menstrual irregularities, the biochemical features of hypoaldosteronism favour a diagnosis of CAH.

Serum prolactin is incorrect. Although hyperprolactinaemia can be associated with hirsutism and menstrual irregularities, the biochemical features of hypoaldosteronism favour a diagnosis of CAH.

Transvaginal ultrasound is incorrect. Although ovarian tumours can result in androgen excess, this usually results in virilization which is not present in this case. Furthermore, hypoaldosteronism would not be expected to occur with ovarian pathology.

Question 1 of 249

A 45-year-old woman with chronic alcohol abuse admitted 3 days ago for nausea and severe diarrhoea now complains of peri-oral and finger tingling. She was admitted for hydration after 1 week of severe watery diarrhoea. She has been receiving intravenous hydration and dextrose but has not been able to take oral nutrition secondary to continued nausea. Her blood pressure is 130/74 mmHg, pulse is 68/min, and respiratory rate is 16/min. She is afebrile.

Physical examination is significant for facial twitching on percussion of her facial nerve just anterior to the ear, as well as the induction of carpal spasm after the inflation of a blood pressure cuff on her arm.

Which of the following is most likely to have caused these findings?

A. Hyperuricaemia
B. Hypernatraemia
C. Hypomagnesaemia
D. Hypophosphataemia
E. Hypouricaemia

C. Hypomagnesaemia

Magnesium deficiency causes hypocalcaemia

This patient is displaying classic signs of hypocalcaemia, including hyperexcitability of her facial nerve (Chvostek's sign), induced carpal spasm (Trousseau's sign), and tingling of the extremities and lips. Calcium homeostasis is a complicated process involving PTH, vitamin D, albumin and numerous electrolytes. Acquired hypoparathyroidism is the most common form of true hypocalcaemia, most often occurring transiently after thyroid surgery or after the removal of a parathyroid adenoma. Occasionally, hypomagnesaemia can produce hypocalcaemia by decreasing both the body's production of PTH and its sensitivity to the hormone. In this case, it is likely that the patient became magnesium depleted from her course of watery diarrhoea, likely baseline poor nutritional status and alcohol abuse.

Choice 1: Hyperuricaemia is not a cause of hypocalcaemia. Chronic kidney disease, however can lead to hypocalcaemia in the setting of secondary hyperparathyroidism, but there is no evidence of renal failure in this patient.

Choice 2: Fluid balance (hyper- or hyponatraemia) does not play a role in calcium homeostasis.

Choice 4: Hypophosphataemia is not a cause of hypocalcaemia. Actually, hypocalcaemia often leads to hyperphosphataemia secondary to increased PTH-mediated bone resorption. Elevations in phosphate may also contribute to hypocalcaemia by complexing with circulating calcium and suppressing conversion of 25-OH to 1, 25-OH vitamin D.

Choice 5: Urate levels do not affect calcium homeostasis.

Question 2 of 249

A 70-year-old woman is brought to the emergency department due to confusion, dizziness and weakness. She has had several similar episodes in the past 3 months. Her past medical history includes type 2 diabetes mellitus of 20 years duration, hypertension, and hyperlipidaemia. Her diabetes is managed with metformin and glimepiride.

Her observations are taken as follows: temperature 37ºC, blood pressure 110/70 mmHg, heart rate 90/min, and respiratory rate 16/min. Physical examination shows no abnormalities.

Blood tests are taken, and her finger-stick glucose is 2.7 mmol/L. Glucagon IM and 50 ml of 50% dextrose IV are given. She remains confused, and a repeat finger-stick glucose is 2.9 mmol/L. Serum insulin and C-peptide levels, drawn before dextrose administration, are elevated.

Which of the following is the next best step in the management of this patient?

A. Recheck insulin and C-peptide levels
B. Spiral computed tomography (CT) of the abdomen
C. Repeat glucagon IM
D. Observe for 30 minutes and recheck finger-stick glucose
E. Administer octreotide IM

E. Administer octreotide IM

In sulphonylurea overdoses, if the patient remains hypoglycaemic despite infusion of sufficient glucose, consider administration of octeotride

This patient has symptomatic hypoglycaemia and elevated insulin and Cpeptide levels, suggesting that the cause of her hypoglycaemia is excessive insulin secretion by her pancreas. Injectable insulins do not have C-peptide. There are two possible causes of excessive endogenous insulin secretion: insulinoma and sulphonylurea overdose. In this patient taking glimepiride, the latter is the more likely cause. In sulphonylurea overdoses, if the patient remains hypoglycaemic despite the infusion of sufficient glucose, consider administration of octreotide.

Sulphonylurea overdose is treated with bolus dextrose solution. Most patients recover after this intervention. However, since sulphonylureas cause the pancreas to continue releasing insulin, patients are at an increased risk of persistent or recurrent hypoglycaemia. In such patients, 50 to 100 mcg octreotide is administered intramuscularly or subcutaneously. Octreotide is a somatostatin analogue that inhibits insulin release from pancreatic beta-islet cells.

Rechecking insulin and C-peptide levels is inappropriate in this case and would lead to unnecessary delays in management. This patient with symptomatic hypoglycaemia needs urgent treatment.

While insulinoma is a possible cause of raised insulin and C-peptide levels, it is rare and quite unlikely in this patient. Moreover, getting a CT scan would delay the management of her hypoglycaemia.

Repeating glucagon administration may control the hypoglycaemia, but it will not address the underlying cause of persistent hypoglycaemia. The best next step is to administer octreotide.

This patient has symptomatic hypoglycaemia in the setting of sulphonylurea overdose. Observing for 30 minutes would worsen her hypoglycaemia and may lead to coma or death.

Question 3 of 249

A 35-year-old woman is reviewed in endocrinology outpatient clinic. She reports a three-month history of headaches, palpitations, mild constipation, and urinary frequency. She is persistently hypertensive despite treatment with the maximum dose of lisinopril, amlodipine, and indapamide.

Her observations are:

- Respiratory rate: 18/min
- Oxygen saturation: 99% on room air
- Heart rate: 115bpm
- Blood pressure: 187/98mmHg
- Temperature: 36.2 C
- GCS: 15/15

An ECG is performed which shows sinus tachycardia.

A set of blood tests has been performed. The results are as follows:

Hb	140 g/L	Female: (115 - 160)
Platelets	387 * 10 /L 9	(150 - 400)
WBC	9.0 * 10 /L 9	(4.0 - 11.0)
Calcium	2.80 mmol/L	(2.1-2.6)
Phosphate	1.2 mmol/L	(0.8-1.4)
Magnesium	0.8 mmol/L	(0.7-1.0)
Thyroid-stimulating hormone (TSH)	5.0 mU/L	(0.5-5.5)
Free T4	12 pmol/L	(9.0 - 18)
Parathyroid hormone	52 pg/mL	(10 - 55)

Given the likely diagnosis, what is the most appropriate blood pressure treatment?

A. Atenolol
B. Bisoprolol
C. Nicorandil
D. Phenoxybenzamine
E. Spironolactone

D. Phenoxybenzamine

This patient has symptoms of hypercalcaemia (constipation, urinary frequency), coupled with a mildly raised serum calcium level. The serum parathyroid hormone (PTH) level is at the upper end of normal - this is suggestive of primary hyperparathyroidism. This patient has several cardinal symptoms of a phaeochromocytoma (headache, palpitations, hypertension). This patient is also likely to have a phaeochromocytoma, as the combination of primary hyperparathyroidism (secondary to a parathyroid adenoma or parathyroid hyperplasia) and a phaeochromocytoma is in keeping with multiple endocrine neoplasia type 2 (MEN-2A). The patient will require cross-sectional imaging to localise the phaeochromocytoma. Treatment should be initiated in the interim to control symptoms.

Phenoxybenzamine is the correct answer. Alpha-blockers should be initiated first-line. Initiation of beta-blockers before alpha-blockade will cause unopposed stimulation of alpha-receptors, causing vasoconstriction and hypertensive crisis.

Atenolol is the incorrect answer. Atenolol is a beta-blocker and will cause unopposed stimulation of alpha receptors.

Bisoprolol is the incorrect answer. Bisoprolol is another beta-blocker - like atenolol, it can risk causing a hypertensive crisis.

Nicorandil is the incorrect answer. Nicorandil is a treatment option for lowering blood pressure but is not specifically used to treat hypertension secondary to a phaeochromocytoma.

Spironolactone is the incorrect answer. Spironolactone is used to treat hypertension but as above, it is not a targeted treatment for hypertension due to a phaeochromocytoma.

Question 4 of 249

A 53-year-old woman is referred to an endocrinology clinic. She has a history of Grave's disease which had previously been controlled on carbimazole however recently presented to her general practitioner due to a two-month history of diarrhoea, palpitations, and heat intolerance. She is post-menopausal and has not had any recent illness or relevant past medical history. The general practitioner performed some thyroid function tests, results of which are as follows:

TSH	0.2	(0.5-5.5 mu/L)
Free T4	25.2	(9-18 pmol/L)

On examination in the clinic, she is warm, tachycardic but otherwise well. A smooth, non-tender goitre is noted however there are no eye signs.

What is the most appropriate treatment?

A. Advice on medication compliance
B. Prednisolone
C. Propylthiouracil
D. Radioiodine therapy
E. Thyroidectomy

D. Radioiodine therapy

Radioiodine therapy is the treatment of choice for patients with a relapse of Graves disease in the absence of contraindications, such as pregnancy and active severe Graves ophthalmopathy

Given this woman's medical history, findings of a smooth non-tender goitre, and laboratory results, the most likely diagnosis here is a relapse of her Grave's disease. In these cases and the absence of any contraindications, radioiodine therapy is the preferred treatment.

Prednisolone can be used in subacute thyroiditis however this is not the diagnosis here as evidenced by the lack of recent illness and non-tender goitre.

Propylthiouracil is an alternative antithyroid drug to carbimazole and is preferred in early pregnancy. As this woman is presenting with a relapse of Grave's disease, radioiodine therapy is preferred. Also, note she is post-menopausal, and therefore pregnancy is not a contraindication here to either radioiodine therapy or carbimazole.

Thyroidectomy is not the first-line treatment in a relapse of previously well-controlled Grave's disease.

Question 5 of 249

A 16-year-old boy presents with several months of generalised weakness and muscle cramps. He also describes polydipsia and cravings for salty foods. He has no significant past medical history.

On examination, he appears generally well in himself.

Observations are as follows:

- Respiratory rate of 18/min
- Blood pressure of 116/78mmHg
- Heart rate of 78/min

Blood tests are done and the results are as follows:

Na +	129 mmol/L	(135 - 145)
K +	2.8 mmol/L	(3.5 - 5.0)
Magnesium	0.54 mmol/L	(0.7 - 1.0)

A urine dipstick shows no glucose or protein present. A urine sample is sent to the lab for further analysis:

Urine calcium/creatinine ratio	0.05	(<0.14)

What is the most likely diagnosis?

A. Bartter syndrome
B. Dent disease
C. Fanconi syndrome
D. Gitelman syndrome
E. Liddle syndrome

D. Gitelman syndrome

Gitelman's syndrome: normotension, hypokalaemia + hypocalciuria

The syndrome presented here is that of **Gitelman syndrome**. This is a rare genetic disorder caused by mutations in the thiazide-sensitive sodium-chloride symporter in the distal convoluted tubule. Symptomatic patients present with symptoms identical to those taking excessive thiazide diuretics - hypokalaemia, hypomagnesaemia, and hyponatraemia. Patients are normotensive. Importantly, patients also display hypocalciuria, which differentiates the condition from Bartter syndrome. Treatment involves salt replacement.

Bartter syndrome is a similar condition, except that it involves a mutation in the Na/K/2Cl cotransporter in the loop of Henle. As such, the condition mimics loop diuretic abuse. Whilst also presenting with hypokalaemia, hypomagnesaemia, and normotension, the condition crucially presents with hypercalciuria, rather than hypocalciuria as seen in Gitelman syndrome.

Dent disease is a rare X-linked recessive condition that affects the proximal tubules. It is characterised by tubular proteinuria, hypercalciuria, and glycosuria. It is one of the causes of Fanconi syndrome. The scenario above is more closely aligned with Gitelman syndrome.

Fanconi syndrome is a syndrome of proximal tubule dysfunction. It presents with polyuria and polydipsia, like in this scenario, but patients would also have proteinuria and glycosuria, unlike the patient here. Hypokalaemia would also be noted, as well as hypophosphataemia. It may be genetic, such as being caused by Dent disease or acquired.

Liddle syndrome is an autosomal dominant condition - a mutation in the epithelial sodium channels (ENaC) of the kidneys, lungs, and sweat glands. The condition causes hypertension, which does not respond to medications, and hypokalaemia. Typically, children are asymptomatic at presentation. The patient in this scenario is normotensive.

Question 6 of 249

A 23-year-old woman attends a fertility clinic with her partner. She complains of oligomenorrhoea and galactorrhoea and has failed to get pregnant after 18 months of regular unprotected intercourse. Blood tests reveal a serum prolactin level of 6000 mIU/l (normal <500 mIU/l). A pituitary MRI is arranged which shows a microprolactinoma.

Which of the following is the best initial treatment?

A. Octreotide
B. Bromocriptine
C. Trans-sphenoidal hypophysectomy
D. Pituitary radiotherapy
E. Transfrontal hypophysectomy

B. Bromocriptine

Dopamine agonists (e.g. cabergoline, bromocriptine) are first-line treatment for prolactinomas, even if there are significant neurological complications

This patient has a prolactinoma. In the majority of cases, symptomatic patients are treated medically with dopamine agonists (e.g. bromocriptine) which inhibit the release of prolactin from the pituitary gland. Surgery is performed for patients who cannot tolerate or fail to respond to medical therapy. A trans-sphenoidal approach is generally preferred unless there is a significant extra-pituitary extension. Radiotherapy is rarely performed and octreotide is a somatostatin analogue used in the treatment of acromegaly.

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A 58-year-old man attends the clinic for a routine review. He was diagnosed with type 2 diabetes mellitus (T2DM) two years ago, myocardial infarction one year ago, and hypertension. His current medications include metformin 1 g twice daily, ramipril 5 mg once daily, aspirin 75 mg once daily and atorvastatin 80 mg once daily.

On examination, his BMI is 27 kg/m², his blood pressure is 138/84 mmHg, and his heart rate is 77 bpm. On auscultation, his chest is clear, and heart sounds are dual with no murmurs. He is clinically euvolaemic.

Hb	137 g/L	Male: (135-180)
Platelets	165 * 10 /L 9	(150 - 400)
WBC	6.7 * 10 /L 9	(4.0 - 11.0)
Na +	137 mmol/L	(135 - 145)
K +	4.5 mmol/L	(3.5 - 5.0)
Urea	9.7 mmol/L	(2.0 - 7.0)
Creatinine	144 µmol/L	(55 - 120)
CRP	3 mg/L	(< 5)
HbA1c	62 mmol/mol	

What is the most appropriate next step in the management of his diabetes?

A. Dapagliflozin
B. Gliclazide
C. Increase the dose of metformin
D. Semaglutide
E. Sitagliptin

A. Dapagliflozin

SGLT-2 inhibitors should be used in addition to metformin as initial therapy for T2DM if CVD, high-risk of CVD or chronic heart failure

Adding an SGLT-2 inhibitor such as **dapagliflozin** is the correct answer. NICE guidelines recommend using SGLT-2 inhibitors in addition to metformin as initial therapy in patients with T2DM who have established cardiovascular disease (CVD), high cardiovascular risk, or chronic heart failure. This patient's history of myocardial infarction places him in the category of established CVD, making SGLT-2 inhibitors the most appropriate next step.

Also, NICE recommends offering an SGLT-2 inhibitor to adults with type 2 diabetes and chronic kidney disease (CKD) who are already taking either an angiotensin-receptor blocker (ARB) or an angiotensinconverting enzyme (ACE) inhibitor (such as this case) at the highest tolerated dose, provided they have an albumin-to-creatinine ratio (ACR) over 30 mg/mmol and meet the criteria specified in the marketing authorisation, including relevant estimated glomerular filtration rate (eGFR) thresholds. SGLT-2 inhibitors have shown evidence of reducing the risk of end-stage renal disease, mortality, and hospitalisation in adults with type 2 diabetes and CKD.

Adding a sulphonylurea such as **gliclazide** is another option for secondline therapy, but it does not provide the same cardiovascular benefits as SGLT-2 inhibitors. In patients with T2DM and established CVD or high risk of CVD, SGLT-2 inhibitors are preferred due to their ability to reduce the risk of cardiovascular events. Sulphonylureas also cause weight gain and are best avoided in this patient, who is already overweight.

Increase the dose of metformin is not necessary, as the patient is already on the maximum tolerated dose (1 g twice daily). Increasing the dose further will not provide additional benefits in terms of glycaemic control.

Adding a GLP-1 receptor agonist such as **semaglutide** is another option in patients with T2DM when triple therapy is not effective and specific criteria are met. It is a particularly useful adjunct for weight loss in obese patients, which is not the case here. This patient is only on metformin, and SGLT-2 inhibitors would be preferred given his established cardiovascular disease (previous myocardial infarction).

Adding a DPP-4 inhibitor such as **sitagliptin** is an option for second-line therapy in patients with T2DM when HbA1c targets are not met with metformin alone. However, in patients with established cardiovascular disease (CVD), as seen in this patient with a history of myocardial infarction, NICE guidelines recommend adding an SGLT-2 inhibitor as it has additional cardiovascular benefits.

Question 8 of 249

A 59-year-old man who has had type 1 diabetes mellitus for 40 years comes to the clinic for review. He has peripheral diabetic sensory neuropathy, impotence, and has received laser therapy for bilateral diabetic retinopathy. His main complaint that he is now suffering from unpredictable vomiting of undigested food. Despite modifying his diet he still suffers from the problem. On one occasion when he was admitted to hospital the condition responded well to erythromycin. Barium swallow has demonstrated significantly prolonged gastric emptying.

Which of the following is the most appropriate long-term intervention?

A. Ondansetron
B. Gastric pacemaker
C. Chlorpheniramine
D. Domperidone
E. Erythromycin

D. Domperidone

Ondansetron, a centrally acting anti-emetic is largely ineffective for gastroparesis, erythromycin is used for acute episodes of diabetic

Diabetic gastroparesis is best managed long term with domperidone

Pro-kinetic anti-emetics are the intervention of choice for the treatment of diabetic gastroparesis, the diagnosis here. Vomiting is often unpredictable and doesn't respond to dietary modification, making the condition extremely debilitating for sufferers. Domperidone, a dopamine D2 receptor antagonist is the main prospect for long-term intervention, as it doesn't have the neurological liabilities which are associated with metoclopramide use, it may be associated with galactorrhoea due to a rise in prolactin levels in some patients who take it.

gastroparesis, and gastric pacemaker therapy is designated a research therapy of last resort by NICE. Chlorpheniramine is an anti-histamine that has some anti-emetic properties, although it is predominantly a centrally acting agent.

Question 9 of 249

A 38-year-old woman is referred by her GP for management of Graves' disease, diagnosed by the presence of a goitre, suppressed thyroid stimulating hormone, and presence of thyroid antibodies on screening. She has no past medical history of note, drinks 10 units of alcohol per week and smokes 20 cigarettes per day. On examination her blood pressure is 112/88 mmHg, pulse is 89 beats per minute and regular, she has a fine tremor. There is a smooth goitre and marked proptosis.

Which of the following has the greatest negative impact on prognosis of her thyroid eye disease?

A. Alcohol consumption
B. Cigarette smoking
C. DR4 HLA type
D. LATS titre
E. Use of block replace therapy

B. Cigarette smoking

Smoking is the most important modifiable risk factor for the development of thyroid eye disease

A systematic review published in 2006 has confirmed the strong link between cigarette smoking and thyroid eye disease. Across 15 studies a strong association between thyroid eye disease in patients with Graves' disease and smoking was established, with an odds ratio of up to 20 for thyroid eye disease in current smokers vs non-smokers who have Graves'.

Block replace therapy establishes stable control of thyroid function, and is actually associated with reduced incidence of thyroid eye disease because thyroxine is consistently in the normal range. Alcohol consumption within the recommended safe limits may actually reduce the severity of thyroid eye disease in Graves'. Thyroid eye disease is primarily driven by pathogenic T cells, as such it isn't closely related to LATS titre. HLA DR4 is more strongly associated with Type 1 diabetes, rheumatoid arthritis and autoimmune hepatitis than with thyroid disease.

Question 10 of 249

A 28-year-old woman presents with flu-like symptoms, palpitations and pain over the anterior neck over the past 2-3 weeks. She has also suffered rapid weight loss and feels increasingly anxious that there may be something seriously wrong with her. Her thyroid-stimulating hormone has been measured at <0.05 IU by her GP. On examination her blood pressure is 128/82 mmHg, her pulse is 95 beats per minute and regular, and she has a fine tremor. There is mild tenderness over the anterior neck. Body mass index is 22 kg/m²

Which of the following would you also expect to find?

A. Erythema nodosum
B. Exophthalmos
C. Multiple small thyroid nodules on ultrasound scan
D. Positive anti-thyroid antibodies
E. Reduced uptake on thyroid scintigraphy

E. Reduced uptake on thyroid scintigraphy

In De Quervain's thyroiditis there is globally reduced uptake of iodine-131 during thyroid scintigraphy

The most likely diagnosis, given the history of 2-3 weeks of flu-like symptoms and suppressed TSH, is subacute thyroiditis where thyroid inflammation drives increased release of stored thyroid hormone, rather than the clinical picture being due to overproduction of T3 and T4. Symptoms of hyperthyroidism should be managed

with beta blockade as required, and there is no role for thioamides. Pain over the thyroid can be managed with non-steroidal anti-inflammatory drugs. After a period of hyperthyroidism, rebound hypothyroidism may be seen, followed by a recovery to euthyroidism.

Erythema nodosum is not associated with subacute thyroiditis.

Exophthalmos and positive anti-thyroid antibodies are associated with autoimmune thyroid disease. Multiple small thyroid nodules are a feature of multinodular goitre.

Question 11 of 249

A 55-year-old woman is admitted following a fall whilst ice-skating, landing on her wrist and hitting her head on the ice. She felt slightly dizzy before the fall and has felt 'not quite right' for many months and was admitted to the hospital 1 month prior due to nephrolithiasis.

An x-ray confirms a scaphoid fracture. A CT head was unremarkable.

Hb	105 g/L	Male: (135-180)Female: (115 - 160)
Platelets	380 * 10 /L $_9$	(150 - 400)
WBC	10.0 * 10 /L $_9$	(4.0 - 11.0)
Na $_+$	140 mmol/L	(135 - 145)
K $_+$	2.6 mmol/L	(3.5 - 5.0)
Urea	4.5 mmol/L	(2.0 - 7.0)
Creatinine	87 µmol/L	(55 - 120)
CRP	10 mg/L	(< 5)
Chloride	122 mmol/L	(101-110)
Bicarbonate	10 mmol/L	(22-28)

A venous blood gas is drawn:

pH	7.2	(7.35-7.45)
lactate	0.8 mmol/L	(0.0-2.0)
pC02	39 mmHg	(35 - 45)
p02	70 mmHg	(75-105)

Urine dip:

pH	6.5
Protein	Trace
Blood	Trace
Leucocytes	Negative

From the following options, what would be the most likely cause of her biochemical abnormalities?

A. Carbonic anhydrase II deficiency
B. Fanconi syndrome
C. Hypoaldosteronism
D. Sjogren's syndrome
E. Topiramate use

D. Sjogren's syndrome

Type 1 (distal) renal tubular acidosis may be caused by Sjogren's syndrome

This patient has an unexplained metabolic acidosis with hypokalemia. If we calculate the anion gap we get a value of 10.6, indicating that this is a normal anion gap metabolic acidosis (NAGMA).

Causes of NAGMA include:

- Acetazolamide use
- Topiramate use
- Renal tubular acidosis type 1 and type 2
- Diarrhoea
- Ureterosigmoidostomy
- Post hypocapnic state

Sjogren's syndrome is correct. This is the only option that can cause type 1 renal tubular acidosis (RTA). Type 1 renal tubular acidosis is characterised by NAGMA, hypokalemia (due to renal potassium loss), high urinary pH (due to inability to acidify urine), and can also be a cause of renal stones. Type 1 RTA can be the first presentation of Sjogren's, so these patients should have autoimmune screening - anti-Ro-, anti-La, rheumatoid factor, and ANA.

Fanconi syndrome is incorrect. Although Fanconi syndrome can show NAGMA and hypokalemia, you would not expect to acidification of the urine as it is associated with type 2 RTA.

Hypoaldosteronism is incorrect. Hypoaldosteronism is associated with type 4 RTA and you would see hyperkalemia as aldosterone usually increases potassium excretion via the cortical collecting ducts.

Carbonic anhydrase II deficiency is incorrect. This is a very rare condition and is associated with a mixed type 3 RTA picture. It would not explain nephrolithiasis and is not the most likely answer.

Topiramate use is incorrect. This is associated with type 2 RTA. You would not expect to see acidification of the urine as it is associated with type 2 RTA.

Question 12 of 249

A 28-year-old woman attends your pre-natal endocrinology clinic. She has been taking 100 micrograms of levothyroxine daily due to hypothyroidism and is currently 6 weeks pregnant, there have been no recent changes to her dosage.

TSH	5.0 mU/L	(0.5-5.5)
Free T4	10.0 pmol/L	(9.0 - 18)

What would you recommend regarding her levothyroxine dose?

A. Continue on current levothyroxine dose, no further monitoring required during pregnancy
B. Increase levothyroxine dose to 150 micrograms daily
C. Increase levothyroxine dose to 200 micrograms once dailyimmediately
D. Reduce levothyroxine dose to 75 micrograms once daily
E. Repeat thyroid function tests at 12 weeks gestation and thenconsider adjusting levothyroxine dose

B. Increase levothyroxine dose to 150 micrograms daily

Women with hypothyroidism may need to increase their thyroid hormone replacement dose by up to 50% as early as 4-6 weeks of pregnancy

During pregnancy, the thyroid gland increases in size by 10% and the production of thyroid hormones, T3 and T4, increases by 50%.

If a patient has pre-existing hypothyroidism, we usually need to increase the dose of levothyroxine to mimic these changes.

Increase levothyroxine dose to 150 micrograms daily: an increase of levothyroxine supplementation of up to 50% mimics the physiological thyroid response to pregnancy and is the correct option for this patient.

Continue on current levothyroxine dose, no further monitoring required during pregnancy: this is incorrect. The requirement for T3 and T4 increases during pregnancy, therefore the levothyroxine dose must be increased.

Increase levothyroxine dose to 200 micrograms once daily immediately: this increase is too high, the patient does not require such an aggressive dosage adjustment.

Reduce levothyroxine dose to 75 micrograms once daily: incorrect, levothyroxine dose should not routinely be reduced during pregnancy, the requirement generally increases.

Repeat thyroid function tests at 12 weeks gestation and then consider adjusting levothyroxine dose: this would be inappropriate, thyroid abnormalities should be treated as soon as possible to minimise the risk to the foetus. Ideally, the patient should be counselled before conception. Maternal hypothyroidism is associated with low birth weight, premature labour, and neonatal respiratory problems.

Question 13 of 249

A 23-year-old woman with type 1 diabetes mellitus is brought to the emergency department by her partner after experiencing 24 hours of abdominal pain, vomiting, and increasing drowsiness. She is usually well, apart from a recent cold. She reports good compliance with insulin therapy.

On examination, she appears dehydrated. She has generalised abdominal tenderness, but there is no guarding or rigidity. Bowel sounds are normal. Her blood pressure is 102/66 mmHg, heart rate is 112 bpm, and respiratory rate is 28 breaths per minute.

Hb	145 g/L	(115-160)
WBC	7.5 * 10 /L 9	(4.0-11.0)
Na^+	140 mmol/L	(135-145)
K^+	4.8 mmol/L	(3.5-5.0)
Urea	5.2 mmol/L	(2.0-7.0)
Creatinine	90 µmol/L	(55-120)
Blood glucose	17 mmol/L	
CRP	2 mg/L	(< 5)
pH	7.08	(7.35-7.45)
Bicarbonate	8 mmol/L	(22-28)

Intravenous 0.9% sodium chloride is initiated.

What is the most appropriate management for this patient?

A. Add 10 mmol of potassium to 500 ml of saline
B. Add 40 mmol of potassium to 500 ml of saline
C. Add 40 mmol of potassium to each litre of saline
D. Do not add potassium to the fluids at this time
E. Start oral potassium supplementation

C. Add 40 mmol of potassium to each litre of saline

Diabetic ketoacidosis: 40 mmol of potassium should be added to a litre of saline if the potassium is within 3.5-5.5mmol/L

In the context of diabetic ketoacidosis (DKA), patients often present with high serum potassium levels on admission due to a shift of potassium from the intracellular to the extracellular space despite having total body potassium depletion. As insulin therapy is initiated, potassium shifts back into cells, which can lead to hypokalemia if not managed appropriately.

According to the JBDS guidelines provided in the background information, if a patient's serum potassium level is between 3.5 and 5.5 mmol/L, the correct management is to **add 40 mmol of potassium to each litre of saline**. In this case, the patient's serum potassium level is 4.8 mmol/L, which falls within this range. Therefore, adding 40 mmol of potassium to each litre of saline is appropriate to prevent hypokalemia during treatment.

It is important not to delay or omit appropriate potassium supplementation as it can lead to dangerous complications such as cardiac arrhythmias.

Add 10 mmol of potassium to 500 ml of saline is insufficient for potassium replacement in DKA when the serum potassium is within the 3.5-5.5 mmol/L range. The standard recommendation is to add 40 mmol of potassium to each litre of saline to prevent hypokalemia, which can develop rapidly as insulin treatment causes potassium to shift into cells.

Add 40 mmol of potassium to 500 ml of saline is not the recommended dose for potassium replacement in this context. The patient's serum potassium is 4.8 mmol/L, which falls within the 3.5-5.5 mmol/L range, indicating a need for 40 mmol of potassium in each litre (not 500 ml) of saline.

Do not add potassium to the fluids at this time is not the correct answer, as it risks the development of severe hypokalemia once insulin is initiated. While potassium replacement is unnecessary if the initial serum

potassium is > 5.5 mmol/L, this patient's potassium level is 4.8 mmol/L, requiring supplementation.

Start oral potassium supplementation is not suitable for the management of DKA, where rapid and adequate intravenous potassium replacement is essential due to the risk of severe hypokalemia once insulin therapy begins.

Question 14 of 249

A 45-year-old woman presents to her GP with complaints of joint pains, headaches, and excessive sweating. She has also noticed her shoe size increasing and coarsening of facial features. Her past medical history is unremarkable, and she is not taking any regular medications. On examination, her blood pressure 154/85 mmHg, she has broad hands, and a prominent forehead.

Given the likely diagnosis, what is the most appropriate first-line investigation?

A. Oral glucose tolerance test (OGTT) with growth hormone (GH) measurement
B. Pituitary MRI
C. Random growth hormone (GH) level
D. Serum IGF-1 level

E. Thyroid function tests

D. Serum IGF-1 level

Serum IGF-1 levels are now the first-line test for acromegaly

The clinical presentation of excessive sweating, joint pains, headaches, increased shoe size, and coarsening facial features suggests acromegaly. According to the Endocrine Society guidelines, **serum IGF-1 level** is recommended as the first-line test in patients with typical clinical manifestations of acromegaly. Serum IGF-1 levels are more stable than growth hormone (GH) levels throughout the day and provide a reliable indication of GH activity over time. If IGF-1 levels are elevated or equivocal, an oral glucose tolerance test (OGTT) with GH measurement can be used to confirm the diagnosis by demonstrating a lack of suppression of GH during hyperglycaemia. However, the OGTT is not used as the initial test due to its complexity compared to measuring serum IGF-1 levels.

Oral glucose tolerance test (OGTT) with growth hormone (GH) measurement is not the first-line test for acromegaly. While the OGTT with serial GH measurements is recommended to confirm the diagnosis of acromegaly when IGF-1 levels are elevated, it is not the initial investigation. In acromegaly, the OGTT fails to suppress GH to < 1 µg/L, which is diagnostic.

Pituitary MRI is used to identify a pituitary adenoma, which is often the cause of acromegaly. However, imaging is typically performed after biochemical confirmation of the diagnosis. It is not the first-line test for suspected acromegaly.

Random growth hormone (GH) level is not useful for diagnosing acromegaly, as GH levels fluctuate significantly throughout the day and are not reliable for initial assessment. GH levels alone cannot confirm or rule out acromegaly due to this variability, as normal fluctuations can overlap with pathological levels seen in acromegaly.

Thyroid function tests are not related to the diagnosis of acromegaly. Although thyroid dysfunction may occur due to the growth hormone effect on the thyroid, thyroid function tests are incorrect in this case.

Question 15 of 249

A 47-year-old man attends a diabetes follow-up appointment after being started on metformin 500mg TDS. His most recent HbA1c is 48mmol/mol and he has not been troubled by hypoglycaemia.

His past medical history includes atrial fibrillation and heart failure with preserved ejection fraction. He takes apixaban, bisoprolol, atorvastatin, and ramipril.

What would be the most appropriate course of action regarding his diabetic treatment?

A. Add in dapagliflozin
B. Add in glimepiride
C. Add in sitagliptin
D. No changes to current medications
E. Reduce metformin dose

A. Add in dapagliflozin

In patients with T2DM, SGLT-2 should be introduced at any point they develop CVD, a high risk of CVD or chronic heart failure

The stem describes a patient with well-controlled type 2 diabetes mellitus and a background of heart failure. He is also on apixaban for atrial fibrillation and so at a higher risk of cardiovascular disease. All patients with type 2 diabetes and concomitant high risk of cardiovascular disease, chronic heart failure, or new cardiovascular disease, should be commenced on an SGLT-2 inhibitor once established on metformin if there are no contraindications.

Add in dapagliflozin is correct. Patients with a high risk of cardiovascular disease should be started on an SGLT-2 inhibitor such as dapagliflozin.

Add in glimepiride is incorrect. Glimepiride is a sulfonylurea, these drugs work by increasing insulin secretion and can be helpful for patients with symptomatic, recurrent hyperglycaemia. It can be used as an adjunct if HbA1c targets are not being met, however, in this case, our patient is meeting his HbA1c target.

Add in sitagliptin is incorrect. Sitagliptin is a DDP-4 inhibitor and can be used as an adjunct if HbA1c targets are not being met, however, in this case, our patient is meeting his HbA1c target.

Reduce metformin dose is incorrect, the current metformin dose is maintaining a good HbA1c and the patient is not troubled by hypo or hyperglycaemia. There would be no indication to reduce the dose in this case.

No changes to current medications would be appropriate if this patient had not developed atrial fibrillation. Due to the development of atrial fibrillation, his cardiovascular risk is much increased and as a result, the current best practice is to add in an SGLT-2 inhibitor such as dapagliflozin.

Question 16 of 249

An 18-year-old boy attends your clinic with his parents. They have recently relocated from Bangladesh. His parents were advised to seek help after teachers at his school raised concerns about possible learning difficulties.

On examination, he is 1.42m tall and weighs 78kg. His BMI is 38.6kg/m². The cardio-respiratory examination is unremarkable, but you notice some shortening of the 5th metacarpals bilaterally when looking at his hands.

Blood tests have been completed and show:

Parathyroid hormone	78 pg/mL	(14-65)
Adjusted calcium	2.0 mmol/L	(2.2-2.6)
Phosphate	2.03 mmol/L	(0.97-1.45)

Given the above information, what is the most likely unifying diagnosis?

A. Hypoparathyroidism
B. Osteogenesis imperfecta
C. Osteomalacia
D. Pseudohypoparathyroidism
E. Pseudopseudohypoparathyroidism

D. Pseudohypoparathyroidism

Short fifth metacarpals, short stature, learning difficulties + ↑ PTH, ↓ calcium, ↑ phosphate → pseudohypoparathyroidism

The patient has short stature, short fifth metacarpals, and learning difficulties. With any presentation involving bony abnormalities, a bone profile is a helpful investigation. In this case, we see a raised parathyroid hormone, low calcium, and raised phosphate. The most likely diagnosis is, therefore, pseudohypoparathyroidism.

Pseudohypoparathyroidism is correct. The patient displays the phenotypical features of pseudohypoparathyroidism (short stature, short fifth metacarpals) in addition to learning difficulties. The blood tests also support this diagnosis. In pseudohypoparathyroidism, there is resistance to PTH. Low calcium levels promote PTH secretion, and the body responds by decreasing urinary calcium loss and releasing calcium from the bones. In pseudohypoparathyroidism, the body is not able to respond to increase calcium levels due to resistance to the PTH. We, therefore, see raised PTH (in response to low calcium), low calcium (as the body is resistant to the usual mechanism of increasing calcium levels), and raised phosphate (as PTH usually would increase phosphate excretion).

Pseudopseudohypoparathyroidism is incorrect. In pseudopseudohypoparathyroidism, you often see skeletal abnormalities and phenotypical features of type 1A pseudohypoparathyroidism, however, the biochemistry is normal. This is due to the genetic phenomenon of imprinting. Given the patient in the stem has abnormal biochemistry, the diagnosis is not pseudopseudohypoparathyroidism.

Osteomalacia is incorrect. This is a deficiency in vitamin D. Biochemical abnormalities include high PTH, and low phosphate and calcium. This is not in keeping with the biochemical abnormalities seen in this patient, and would not explain the learning difficulties and other physical abnormalities.

Osteogenesis imperfecta is incorrect. This is a genetic condition of type I collagen that predisposes to reduced bone strength. It is also termed 'brittle bone disease'. You would not see abnormalities in calcium, phosphate, or PTH levels, and so this is not the correct answer here.

Hypoparathyroidism is incorrect. In hypoparathyroidism, you would see low PTH levels and this can be caused by a number of factors e.g. autoimmune disease, inadvertent removal of the parathyroid glands during thyroidectomy, or haemochromatosis amongst others. This patient has raised PTH and so this is not the correct answer. Hypoparathyroidism would also be associated with raised calcium and low phosphate levels.

Question 17 of 249

A 27-year-old female was seen in the general medicine outpatient clinic following a referral by her GP. She had presented to her GP feeling generally tired for the last few months. Her GP organised a blood screen revealing no abnormalities except the presence of a potassium level of 2.8 mmol/l. Her GP subsequently repeated the test four weeks later revealing a level of 2.6 mmol/l, leading to the referral to the clinic. Other than the tiredness she was well in herself, denying all other symptoms. She denied specifically the presence of any cardiovascular and muscular symptoms and had never collapsed. Her past medical history was unremarkable and she was taking no prescribed medication. There was no family history of note.

Examination revealed a well 27-year-old lady. Her blood pressure was 108/78 mmHg, heart rate 82 bpm and BMI 23kg/m². Examination of her cardiovascular and respiratory systems revealed the presence of normal heart sounds, a JVP of 3cm and warm well perfused peripheries. Examination of her gastrointestinal and neurological systems was unremarkable.

Investigations conducted at the clinic revealed the following results:

Hb	122 g/l
Platelets	242 * 10 /l9
WBC	7.8 * 10 /l9
Na +	132 mmol/l
K +	2.5 mmol/l
Urea	7.2 mmol/l
Creatinine	68 µmol/l
Bicarbonate	37 mmol/l
Serum renin	824 (NR 100-500 pmol/l)
Serum aldosterone	82 (NR 55-250 pmol/l)
TSH	1.2 mu/l

24 hour urine result:

Na+	28 (NR <20mmol/L if hyponatraemia)
K+	45 (NR <10mmol/l if hypokalaemic)
Calcium	0.8 (NR <7.5 mmol/24hrs)

What is the most likely diagnosis?

A. Addison's disease
B. Gitelman's syndrome
C. Conn's disease
D. Bartter's syndrome
E. Laxative abuse

B. Gitelman's syndrome

Gitelman's syndrome: normotension, hypokalaemia + hypocalciuria

Gitelman's syndrome is an autosomal recessive disorder resulting in a normotensive hypokalaemic metabolic alkalosis with hypocalciuria and is often accompanied with hypomagnesaemia. The defect is in the thiazidesensitive sodium chloride symporter within the distal convoluted tubules, in contrast to Bartter's syndrome which presents in the same way but with hypercalciuria owing to a defect within the ascending loop of Henle. Patients with both conditions are often asymptomatic or may complain of fatigue, cramps and weakness. Conn's disease is associated with hypertension and in this instance the aldosterone level is normal with an elevated renin, making this diagnosis unlikely. Both Addison's disease and laxative abuse are associated with a metabolic acidosis; in Addison's serum potassium also tends to be elevated. The best answer is, therefore, Gitelman's syndrome.

Question 18 of 249

A surgical Foundation Year 1 doctor (FY1) asks you to review a preoperative ECG for a 19-year-old patient who has been admitted under their team with suspected appendicitis. The only abnormality is a prolonged QT and you note the adjusted calcium to be 2.02 mmol/l.

The FY1 tells you that when they looked at the patients closed fists the outer two knuckles looked like dimples. She also tells you that the patient's body mass index is 29 kg/m².

You ask her to order some blood tests which come back as follows:

Adjusted calcium	2.02 mmol/l
PTH	69 pmol/L (normal range = 0.8 - 8.5)
Phosphate	2.0 mmol/l
ALP	130 u/l

What is the most likely underlying cause for this patient's hypocalcaemia?

A. Hypoparathyroidism
B. Pseudohypoparathyroidism type 1a
C. Pseudohypoparathyroidism type 1b
D. Pseudopseudohypoparathyroidism
E. Secondary hyperparathyroidism

B. Pseudohypoparathyroidism type 1a

Short fifth metacarpals, short stature, learning difficulties + ↑ PTH, ↓ calcium, ↑ phosphate → pseudohypoparathyroidism

This patient has a high PTH, a low calcium, a high phosphate and a normal ALP. The patient is also obese and the dimples on the outer two

knuckles are likely to represent shortening of the 4th and 5th metacarpals. This biochemistry in combination of these clinical features is characteristic of pseudohypoparathyroidism Type 1a (Albright's Hereditary Osteodystrophy).

Pseudopseudohypoparathyroidism would have the same clinical features but would have normal biochemistry. Pseudohypoparathyroidism Type 1b would have the same biochemistry but lack the clinical features.

This patient has a high PTH, therefore this immediately excludes hypoparathyroidism. In secondary hyperparathyroidism the ALP would be elevated therefore this is incorrect.

an infusion of PTH followed by measurement of urinary phosphate and cAMP measurement - this can help differentiate between type I (neither phosphate or cAMP levels rise) and II (cAMP rises but phosphate levels do not change)

Question 19 of 249

A 23-year-old man with type 1 diabetes presents to the emergency department with vomiting and abdominal pain. He has been suffering from diarrhea for two days and then became severely dehydrated and started to vomit. He has not had his insulin for the last 24 hours. He normally has a basal bolus regime with Levemir as a long-acting insulin, and Humalog as short-acting. He has no other medical problems or regular prescription. On admission he is found to have acidosis, elevated serum ketones and elevated blood glucose, and was diagnosed with diabetic ketoacidosis. He is given rapid fluid infusion.

What insulin should he be prescribed?

A. Fixed rate IV insulin only
B. Fixed rate IV insulin as well as long-acting insulin
C. Fixed rate IV insulin, long-acting insulin and short-acting insulin
D. Sliding scale IV insulin only
E. Sliding scale IV insulin as well as long-acting insulin

B. Fixed rate IV insulin as well as long-acting insulin

In the acute management of DKA, insulin should be fixed rate whilst continuing regular injected long-acting insulin but stopping short actin injected insulin

This is a typical presentation for diabetic ketoacidosis in a patient with type 1 diabetes and requires urgent management which should be supported by local guidelines. It is important that IV insulin is started, but any long-acting insulin, Levemir for this patient, should also be continued. This will aid transition back to normal when the DKA resolves and the patient starts to eat and drink again. Giving him short acting insulin is not appropriate during IV insulin therapy which is also a shortacting insulin. Sliding scale IV insulin is no longer recommended in favour of fixed rate insulin.

Question 20 of 249

A 62-year-old patient presents to review in the diabetes clinic. He has had type 2 diabetes for 10 years, as well as previous appendectomy, kidney stones, previous tibial fracture and depression which is now in remission. He takes metformin and gliclazide only and has no allergies. His HbA1c is 40mmol/mol, urine dip is negative for glucose and protein but his blood pressure is 151/96mmHg. He has previously attempted to control his blood pressure by diet and exercise only. He is a white Caucasian.

What is the most appropriate plan in regards to blood pressure management?

A. Start a beta-blocker
B. Start a thiazide-like diuretic
C. Start an ACE inhibitor
D. Start an angiotensin II receptor blocker
E. Start a calcium channel blocker

C. Start an ACE inhibitor

A patient with T2DM with a new diagnosis of hypertension should be treated with an ACE inhibitor as first line treatment, regardless of age

The correct answer is to start a ACE inhibitor. This is because he has diabetes. A patient with diabetes should have an ACE inhibitor as first line treatment for diabetes regardless of their age. If the patient did not have diabetes, then a calcium channel blocker would be the most appropriate option. If the patient could not tolerate a ACE inhibitor then an angiotensin II receptor blocker should be tried. ACE inhibitors should also start for patients with diabetes if there is proteinuria, and therefore should have at least annual urinary albumin to creatinine ratios measured. Beta-blockers are only used after ACE inhibitors, calcium channel blockers, diuretics and alpha blockers have failed to controlled blood pressure. However, IV labetalol can be used in hypertensive emergencies to reduce blood pressure.

Question 21 of 249

You are called to urgently review a 45-year-old woman in the emergency department. She presented earlier in the day with fever, a productive cough and confusion. She had been diagnosed by the emergency department doctors as having community acquired pneumonia after seeing consolidation on her chest X-ray and sepsis secondary to this with associated delirium. Collateral history from her partner establishes that she had been feeling unwell for two days with a cough and become increasingly unwell, confused and agitated. The partner also explained that she had recently been diagnosed with an overactive thyroid and been told to start urgently on anti-thyroid tablets, but had not done so when she had read the label of these tablets and found that they should not be taken during pregnancy and she is trying to become pregnant. She had been taking paracetamol only for fever.

Despite treating with IV co-amoxiclav and oral clarithromycin and fluid resuscitation with three liters of fluid as well as IV paracetamol the patient has not improved.

On examination she is very agitated and aggressive and is difficult to examine. She feels very hot and clammy to touch, but peripheries are well perfused. Auscultation shows left sided crepitations. Her heart rate is 170 bpm, blood pressure 81/40 mmHg, temperature 39.2ºC, oxygen saturations 100% on 2L via nasal cannulae.

Blood tests from this admission are still pending. Out-patient blood tests show a undetectable low TSH two weeks ago, as well as a free T4 of 53mmol/ml.

What is the most appropriate immediate treatment?

A. Change to IV meropenem and monitor
B. Start carbimazole
C. Urgent referral for thyroidectomy
D. Start IV propranolol
E. IV adenosine

D. Start IV propranolol

In thyroid storm with IV beta-blockers are a important first-line treatment

This is a patient with severe confusion, fever and evidence of chest infection and untreated hyperthyroidism. The clinical history suggests that she has developed a thyroid storm as she has fever, severe tachycardia, confusion and agitation with hyperthyroidism. This is a medical emergency, and the most urgent matter is to control her heart rate which is likely the cause for her low blood pressure. IV beta-blocker should be started for this purpose, and propranolol would further help reduce the conversion of T3 into T4. In addition, fluids and dexamethasone should be given, as well as anti-thyroid medications.

Changing to meropenem is unlikely to add any further benefit as the effects of the co-amoxiclav probably have not been seen yet. As the patient is not stable it is not appropriate to refer for surgery as management, but may be a viable long-term strategy to treat her hyperthyroidism. Starting carbimazole now is not the most appropriate action as the patient is unstable and this needs to be corrected first. Adenosine is would be appropriate for a patient with SVT without infection and thyrotoxicosis.

Question 22 of 249

A 17-year-old male is referred to the endocrinology clinic due to a lack of secondary sex characteristics and low serum testosterone. He has never completely developed pubertal body hair or muscle mass, and has small testicles. His past medical history includes a cleft palate at birth. He also finds it difficult to smell things.

What is the most likely underlying diagnosis?

A. Klinefelter's syndrome
B. Androgen insensitivity syndrome
C. Pituitary adenoma
D. Testicular tumour
E. Kallman syndrome

E. Kallman syndrome

Anosmia in the context of male hypogonadism, low testosterone and normal or low FSH and LH makes Kallman Syndrome likely

The correct answer is Kallman syndrome, largely indicated from the history of primary hypogonadism in a young patient lacking secondary sex characteristics and anosmia. The history of cleft palate also is suggestive and helps differentiate from Klinefelter's syndrome. Androgen insensitivity syndrome would present as phenotypically female but genetically male.

Question 23 of 249

A 35-year-old patient presents to the emergency department after being brought in by ambulance. She had recently moved locally but had not yet registered with a GP, and therefore had an interruption in her supply of carbimazole for hyperthyroidism, diagnosed three months earlier. She is currently very unwell, complaining of palpitations. Clinically she has a raised JVP, bilateral crepitations on auscultation and severe peripheral oedema, and has become very breathless, and a temperature of 40.1 degrees C. She is started on oxygen. An ECG demonstrates a heart rate of 170bpm in AF, whilst a chest X-ray shows pulmonary oedema.

What is the most appropriate immediate management?

A. IV propranolol
B. Propylthiouracil
C. Aspirin
D. Refer for emergency thyroid surgery
E. Potassium iodide

A. IV propranolol

In thyroid storm with IV beta-blockers are a important first-line treatment

This is a patient with likely thyroid storm. The presentation suggests abrupt withdrawal of carbimazole has been the provoking factor, and now she has developed AF and heart failure. The most appropriate action is to rapidly control the heart rate by the use of IV beta-blockers. Propylthiouracil should also be started urgently, but the more critical action is to manage her heart rate. Aspirin may worsen the situation by displacing T4 from thyroid-binding globulin, causing an increase in free T4. Whilst the patient will likely need both potassium iodide and thyroid surgery, neither are as urgent as controlling the heart rate.

Question 24 of 249

A 62-year-old woman presents to hospital. She has been agitated over the last few days and her daughter has become concerned about this. She complains of nausea and has vomited once. She also has a cough which has been productive with a green sputum, and she has had some chest pain when she coughs. She also is confused and agitated. On examination, she has a heart rate of 150bpm, blood pressure of 103/65mmHg, temperature 40.1° and oxygen saturation of 94%. She has a past medical history of myocardial infarction three years ago needing cardiac stenting, polycystic ovaries and hypertension. She has also diagnosed with hyperthyroidism four months ago but she admits poor compliance with treatment. She is supposed to take aspirin, atorvastatin, amlodipine and carbimazole. Her blood tests are pending.

What feature of her presentation is most suggestive of thyroid storm?

A. Productive cough
B. History of aspirin use
C. Chest pain
D. Confusion
E. Hyperpyrexia

E. Hyperpyrexia

In thyroid storm, hyperpyrexia, tachycardia, cardiac failure and agitation are the key diagnostic elements

This patient has features of thyroid storm. The most important features are high fever and tachycardia, with temperatures becoming very high, even above 40ºC, and heart rate can be as high as 180bpm. Thyroid storm can be triggered by infection, and therefore the productive cough is relevant but not a distinguishing feature. The use of aspirin can worsen thyrotoxicosis as well by displacing T4 from thyroid binding globulin but is not a main feature. Agitation and confusion are both features but are less discriminating than hyperpyrexia.

Question 25 of 249

A 45-year-old woman develops agitation, high fever, sweating and atrial fibrillation with a heart rate of 160bpm following a CT pulmonary angiogram. She was recently seen in the emergency department and given a provisional diagnosis of pulmonary embolism and discharged with an urgent CT pulmonary angiogram and treatment dose low molecular weight heparin two days ago. She has a past medical history of breast cancer which has recently recurred with metastases to the liver, depression and COPD. She normally takes sertraline, letrozole, carbimazole and as needed salbutamol. Her original presentation to the emergency department was with shortness of breath which has now resolved. The CT scan has demonstrated no evidence of pulmonary embolism and clear lung fields. On examination, she is very agitated, has tremors, is very hot to touch and has ankle oedema. She has bilateral crepitations on chest auscultation. Her temperature is 40.1ºC. Blood tests are pending.

What is the most likely cause of her presentation?

A. Delayed autoimmune reaction secondary to contrast medium
B. Atypical pneumonia
C. Missed pulmonary embolism with right heart strain
D. Thyroid storm
E. Heparin induced thrombocytopenia

D. Thyroid storm

Iodine in CT contrast media can precipitate thyrotoxicosis or thyroid storm

The correct answer is thyroid storm. The iodine in the CT contrast medium can trigger thyrotoxicosis and thyroid storm. The features of hyperpyrexia, agitation, confusion, AF with a fast ventricular rate in a patient with known hyperthyroidism following a triggering event, such as

CT contrast, infection or surgery can thyrotoxicosis or thyroid storm. An autoimmune reaction is unlikely given the absence of rash and presence of fever and agitation. Pneumonia is unlikely without any radiological features on the CT scan and also with the absence of a cough, and missing pulmonary embolism is unlikely, but it is also unlikely to miss right heart strain on CT.

Question 26 of 249

You are asked to review an 82-year-old man on the care of the elderly ward as serum sodium results are low. The patient was admitted five hours ago with a diagnosis of community-acquired pneumonia and exacerbation of congestive cardiac failure. He presented with a productive cough, shortness of breath and leg swelling. He has a past medical history of multiple myocardial infarcts four years ago and has subsequently developed heart failure. He also has had osteoarthritis, hypertension and high cholesterol. He takes aspirin, ramipril, furosemide, bisoprolol, omeprazole and atorvastatin. On examination, he has a raised JVP and bilateral crepitations to midzones bilaterally as well as oedema to the sacrum.

Blood tests:

Hb	122 g/l
Platelets	478 * 10 /l9
WBC	13.9 * 10 /l9
Na +	125 mmol/l
K +	3.4 mmol/l
Urea	6.1 mmol/l
Creatinine	82 µmol/l

Urinary sodium: 15mmol/l

What is the most likely cause of hyponatraemia?

A. SIADH
B. Furosemide
C. Ramipril
D. Heart failure
E. Renal failure

D. Heart failure

In hyponatraemia, a low urinary sodium makes diuretics an unlikely cause

The most likely cause of this patient's hyponatraemia is heart failure. The patient is clinically hypervolaemic with hyponatraemia in the context of low urinary sodium. The low urinary sodium excludes diuretics as a cause of hyponatraemia. SIADH is also unlikely with low urinary sodium, and also cannot be diagnosed with hypervolaemia. Renal failure can cause hyponatraemia with low urinary sodium as the kidneys fail to excrete sodium, but the normal creatinine excludes this.

Question 27 of 249

A 27-year-old woman has been referred to the endocrinology clinic. She has been complaining of palpitations over the last three weeks, and an ECG during an episode of palpitation revealed sinus tachycardia. This resolved with a Valsalva manoeuvre. She was offered but declined any beta-blockers. She has no past medical history and does not take any regular medications apart from over the counter beta-blockers and an oral contraceptive pill. She does not smoke but drinks roughly two units of alcohol per week. She has a non-tender goitre on examination. Biochemical investigations reveal an undetectable TSH, free T4 of 46ng/dl and positive thyroid-stimulating hormone receptor antibodies. On noticing the goitre, she is keen to start treatment. After discussion, she opts for carbimazole treatment with a view of inducing remission. On starting carbimazole she is warned that if she experiences a sore throat or any infection she must have blood tests to exclude agranulocytosis.

What other symptoms is it important to warn her about?

A. Loss of vision
B. Loss of peripheral sensation
C. Jaundice
D. Worsening palpitations
E. Insomnia

C. Jaundice

Patients taking carbimazole need to be warned about potential hepatic impairment and the relevant symptoms

This is a patient with clinical and biochemical evidence of Graves' disease. As this has been confirmed with positive thyroid-stimulating hormone receptor antibodies it would be entirely appropriate to offer carbimazole, radioiodine or surgery. When starting carbimazole, it is very important to ensure the patient is aware of the risk of agranulocytosis, but it is also important to warn about liver dysfunction which may develop and may necessitate stopping the treatment. Rash, headache, fever and malaise commonly occur as well, but can usually be managed with analgesia and antihistamines. Loss of vision could occur in the case of moderate to severe Graves' disease with orbitopathy and starting on radio-iodine, which can worsen thyroid eye disease. Palpitations and insomnia should improve with treatment as these are symptoms of hyperthyroidism.

Question 28 of 249

A 25-year-old woman is referred urgently to the endocrinology clinic. She became pregnant three weeks ago. She had been suffering from diarrhea for the last month, and despite taking the oral contraceptive pill, became pregnant. She went to see her GP who requested blood tests as well as organizing an antenatal referral. Her full blood count, renal profile and liver function tests are all normal, but her TSH is undetectable and her free T4 levels are 52pmol/l. By the time of referral she has also had thyroid-stimulating hormone receptor antibodies which are positive. She has no past medical history.

What is the most appropriate management for her hyperthyroidism?

A. Hemithyroidectomy
B. Carbimazole
C. Propylthiouracil
D. Propranolol
E. Total thyroidectomy

C. Propylthiouracil

In pregnant woman who develop hyperthyroidism in the first trimester, propylthiouracil is preferred over carbimazole due to lower risk of foetal malformation

This patient has a new diagnosis of Graves' disease with positive thyroidstimulating hormone receptor antibodies whilst pregnant in the first trimester. The most appropriate medical treatment is propylthiouracil. This has a lower risk of foetal malformation than carbimazole in the first trimester and would, therefore, be first-line treatment. Radioiodine is contra-indicated in pregnancy, whilst propranolol would not address the underlying problem. Surgery can be an option to patients who are resistant to propylthiouracil, and would need to happy at an urgent basis. Of operations, a hemithyroidectomy is more appropriate in trying to obtain histological samples in suspicious histology, whilst a total thyroidectomy would be a definitive management. Urgent treatment is necessary as thyroid-stimulating hormone receptor antibodies cross the placenta and can cause foetal problems.

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A 59-year-old gentleman with capricious type 2 diabetes mellitus is reviewed in a community diabetic clinic. Despite optimising lifestyle and diet, glycaemic control is still poor on first line oral hypoglycaemic therapy. His diabetic consultant decides to start him on dapagliflozin 10mg daily.

Which of the following is a common side effect of dapagliflozin the patient should be warned about?

A. Pancytopaenia
B. Jaundice
C. Increased likelihood of pancreatitis
D. Increased likelihood of urinary infections
E. Increased risk of ischaemic heart disease

D. Increased likelihood of urinary infections

Dapagliflozin is a SGLT-2 inhibitor which causes increased renal glucose loss to control diabetic glycaemia. It may cause recurrent urinary infections due to high urinary glucose load

Since the regulation of glucose and insulin is affected by many physiological variables, there are many targets for drugs to influence glycaemic homeostasis. Older oral hypoglycaemic drugs such as the sulphonylureas (gliclazide, etc) work by directly stimulating insulin release from pancreatic beta cells, whereas biguanides like metformin inhibit glucose mobilisation from hepatic stores and improve glucose handling. Metformin is associated with lactic acidosis and sulphonylureas are associated with increased incidence of hepatitis and jaundice.

A class of oral hypoglycaemic agent known as the thiazolidinediones (pioglitazone, etc) also improve glucose processing by increasing upregulation of genes which augment glucose and fat metabolism. This occurs by stimulation of the nuclear signalling protein peroxisome proliferator-activated receptor gamma (PPAR-). Thiazolidinediones have fallen out of vogue of late due to concerns about heart failure and ischaemic heart disease.

A more novel treatment for hyperglycaemia is the use of incretins which are gastrically derived peptides which also stimulate insulin secretion from beta cells. The two main peptides of use are glucose like peptide 1 (GLP-1) and gastric inhibitory peptide (GIP) both of which synergistically stimulate beta cell insulin release and inhibit alpha-cell glucagon release, causing a fall in blood glucose. Incretin drugs of this class include exenatide and liraglutide and are usually given as subcutaneous daily injections. In vivo, GLP-1 and GIP are rapidly deactivated by the enzyme dipeptidyl peptidase-4 (DPP-4) which limits their use (although the synthetic incretin analogue drugs are not deactivated by this enzyme). Drugs which can inhibit DPP-4 are useful in controlling blood glucose since they allow physiological gut incretins to stimulate insulin release. Examples of these drugs include sitagliptin, saxagliptin, etc. Rarely DPP-4 inhibitors and PPAR- modulators can cause hepatitis, pancreatitis and pancytopenia. Previous pancreatitis is a relative contraindication to their use.

Dapagliflozin is a renal SGLT-2 (sodium glucose transporter) inhibitor which has only recently obtained UK and US licensing authority. It causes an increase in glucose excretion by the kidney to lower serum glucose concentrations. SGLT-2 is a sodium/glucose cotransporter protein in the nephrons proximal tubule which reabsorbs glucose from the renal filtrate. These drugs cause heavy urinary glucose loss and can cause recurrent urinary infections and candidiasis as well as hypoglycaemia, crystalluria and renal failure due to osmotic diuresis.

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A 43-year-old man presents to his general practitioner complaining of generalized aches and pains. He has a past medical history of glaucoma and takes acetazolamide. He does not smoke cigarettes or drink alcohol. He is a marketing manager for a large international fashion brand.

On examination, he has subtle proximal myopathy. There is no synovitis. Blood tests:

Hb	136 g/L	Male: (135-180)Female: (115 - 160)
Platelets	189 * 10 /L 9	(150 - 400)
WBC	8.2 * 10 /L 9	(4.0 - 11.0)
Na +	137 mmol/L	(135 - 145)
K +	2.8 mmol/L	(3.5 - 5.0)
Urea	5.2 mmol/L	(2.0 - 7.0)
Creatinine	89 µmol/L	(55 - 120)
Bicarbonate	16 mmol/L	(22-29)
CRP	4 mg/L	(< 5)
Bilirubin	14 µmol/L	(3 - 17)
ALP	165 u/L	(30 - 100)
ALT	23 u/L	(3 - 40)
Î³GT	44 u/L	(8 - 60)
Albumin	36 g/L	(35 - 50)
Parathyroid hormone	7.9 pmol/L	(1.6-6.9)
Calcium	2.08 mmol/L	(2.20-2.60)
Vitamin D	31nmol/L	(>50)

What is the unifying diagnosis that explains the presentation?

A. Osteoporosis
B. Primary hyperparathyroidism
C. Type 1 renal tubular acidosis
D. Type 2 renal tubular acidosis
E. Type 4 renal tubular acidosis

D. Type 2 renal tubular acidosis

Hypokalaemia, osteomalacia - type 2 renal tubular acidosis

Type 2 renal tubular acidosis is the correct answer. Type 2 renal tubular acidosis is characterized by the failure of proximal tubular cells to reabsorb bicarbonate, resulting in academia. It is associated with hypokalemia and the development of osteomalacia. The traditional explanation for osteomalacia in this setting is that the proximal tubular conversion of 25 (OH)-cholecalciferol to the active 1,25 (OH)2- cholecalciferol is impaired. Acetazolamide treatment is a known cause of type 2 renal tubular acidosis. This patient presents with aches and pains and proximal myopathy, low vitamin D, low calcium and raised PTH, typical of osteomalacia. Additionally, there is academia (low serum bicarbonate) and hypokalemia. The unifying diagnosis is type 2 renal tubular acidosis secondary to acetazolamide treatment.

Type 4 renal tubular acidosis is incorrect. This is usually associated with hyperkalemia rather than hypokalemia.

Type 1 renal tubular acidosis is incorrect. This is less likely to cause osteomalacia and not associated with carbonic anhydrase inhibitor use. It is associated with the development of nephrocalcinosis.

Primary hyperparathyroidism is incorrect. This patient has an appropriately raised parathyroid hormone as a consequence of vitamin D deficiency and hypocalcemia.

Osteoporosis is incorrect. Calcium is typically normal in this condition and it is characterized by fracture and low T scores, rather than proximal myopathy and generalized aches and pains.

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A 35-year-old woman presents to the emergency department with 3 days of fevers and sweating. She has a past medical history of Graves' disease and is not compliant with medication treatment. She smokes ten cigarettes daily and works in advertising.

Her observations are heart rate 146 beats per minute, blood pressure 154/99 mmHg, respiratory rate 24/minute, oxygen saturations 97% on room air and temperature 38.4ºC.

On examination, she is diaphoretic, tremulous and confused (Glasgow coma scale 14/15). Proptosis and chemosis are noted on examination of her eyes. There are bilateral crackles on chest auscultation and her JVP is elevated.

Blood tests:

Hb	124 g/L	Male: (135-180)Female: (115 - 160)
Platelets	189 * 10 /L $_9$	(150 - 400)
WBC	5.3 * 10 /L $_9$	(4.0 - 11.0)
Na $^+$	131 mmol/L	(135 - 145)
K $^+$	4.2 mmol/L	(3.5 - 5.0)
Urea	5.4 mmol/L	(2.0 - 7.0)
Creatinine	89 µmol/L	(55 - 120)
CRP	4 mg/L	(< 5)
Bilirubin	26 µmol/L	(3 - 17)
ALP	122 u/L	(30 - 100)
ALT	99 u/L	(3 - 40)
Î³GT	74 u/L	(8 - 60)
Albumin	34 g/L	(35 - 50)
TSH	0.0 mIU/L	(0.2 - 5.5)
Free T4	81 pmol/L	(10 - 24.5)

Based on the likely diagnosis, what treatment is most appropriate from the listed options?

A. Antibiotics
B. Carbimazole
C. Propylthiouracil
D. Radioactive iodine therapy
E. Surgery

C. Propylthiouracil

In thyroid storm, treat acutely with propylthiouracil rather than carbimazole or surgery

Propylthiouracil is the correct answer. The patient presents with ophthalmological features of Graves' disease in association with fever, confusion, tachycardia, tremor, hypertension and clinical evidence of heart failure. In the setting of significant hyperthyroidism this constitutes a 'thyroid storm'. Propylthiouracil has traditionally been the preferred anti-thyroid drug to be used in this setting due to its more rapid onset of action when compared with carbimazole and its additional ability to inhibit peripheral conversion of T4 to T3.

Antibiotics is not the right answer. Although this patient has a fever, she has a normal CRP and WCC and infection is less likely than thyroid storm given the clinical context.

Carbimazole is incorrect. This can be used in thyroid storm but is less preferred due to its slower onset of action.

Radioactive iodine therapy is incorrect. This can be used to manage Graves' disease but is not an option acutely for treating thyroid storm.

Surgery is incorrect. This is an option for the treatment of Graves disease but is not an option for the acute treatment of thyroid storm.

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A 65-year-woman is referred to the endocrinology department by her general practitioner. She complains of 12 months of enlarging hands and feet with an inability to put on her usual rings. She also reports that her facial appearance has changed and complains of persistent sweating and arthralgia. She has no significant past medical history and is on no regular medications.

On examination, it is noted that she has coarsened facial features and frontal bossing. There is macroglossia. Neurological examination reveals bitemporal hemianopia. Her hands and feet appear large and spade-like. There was no synovitis.

Blood tests:

Hb	138 g/L	Male: (135-180)Female: (115 - 160)
Platelets	189 * 10 /L $_9$	(150 - 400)
WBC	6.2 * 10 /L $_9$	(4.0 - 11.0)
Na $^+$	138 mmol/L	(135 - 145)
K $^+$	4.2 mmol/L	(3.5 - 5.0)
Urea	5.4 mmol/L	(2.0 - 7.0)
Creatinine	89 µmol/L	(55 - 120)
CRP	4 mg/L	(< 5)
IGF-1	38.2 nmol/L	(7.6-23.1)

An MRI head reveals a large pituitary macroadenoma.

An oral glucose tolerance test is arranged and there is a failure to suppress growth hormone with an oral glucose load.

The patient undergoes trans-sphenoidal surgery to remove the tumour.

Despite this intervention, she complains of ongoing sweating, arthralgia and fatigue.

A repeat MRI demonstrates a small amount of residual tumour that is deemed inaccessible to further surgical intervention.

A repeat IGF-1 remains elevated (35 nmol/L) and there is persistent failure to suppress growth hormone with an oral glucose load.

Given the likely diagnosis, what is the most appropriate management at this point?

A. Bromocriptine
B. Cabergoline
C. Octreotide
D. Pegvisomant
E. Radiotherapy

C. Octreotide

Acromegaly: if patients are not suitable for trans-sphenoidal surgery, or have residual symptoms, then octreotide may be used

Octreotide is the correct answer. This patient has acromegaly, which is the consequence of a growth hormone-secreting pituitary tumour. Clinical features include enlargement of hands and feet, coarsening of facial features and frontal bossing. IGF-1 is raised and the diagnosis is confirmed with the failure to suppress growth hormone secretion with an oral glucose load. Trans-sphenoidal surgery is first-line management in most instances. If there are ongoing symptoms post-surgery like there are in this case, there are a number of options. Repeat surgery may be indicated if the residual tumour is accessible. However, it is not in this case. Therefore medical management is indicated to control the symptoms. Octreotide is an analogue of the hypothalamic release inhibiting hormone, somatostatin. It results in significant tumour shrinkage although the mechanism of action by which it exerts this effect is unknown. It is the first-line medical treatment in this situation.

Radiotherapy is incorrect. This treatment is reserved for cases that are refractory to medical management.

Cabergoline is incorrect. This medication was historically used in the medical management of acromegaly. However, it is less effective than octreotide and associated with significant side effects like cardiac fibrosis.

Bromocriptine is incorrect. This medication is less effective than octreotide. It is a dopamine agonist like cabergoline and is associated with a similar side effect profile.

Pegvisomant is incorrect. This is a second line option in the UK for those patients who do not respond to either surgery, radiotherapy or somatostatin analogues. It is an analogue of human GH and a highly selective GH receptor antagonist which blocks the peripheral synthesis of IGF-1.

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A 54-year-old woman presents to the emergency department with sweating and palpitations. She has a past medical history of Graves disease. She has completed a course of carbimazole approximately six months ago. Her other past medical history includes type 2 diabetes, hypertension, ischaemic heart disease and morbid obesity. She is on metformin, aspirin, amlodipine and atorvastatin.

Her observations are as follows:

- Heart rate 111 beats per minute
- Blood pressure 124/72 mmHg
- Respiratory rate 21/minute
- Oxygen saturations 96% on room air
- Temperature 37.1C

On examination, she is diaphoretic and anxious. A tremor is noted. Ophthalmic examination is normal. There is a very small goitre. Her body mass index is 53 kg/kg/m².

Blood tests:

TSH	0.0 mIU/L	(0.2-5.5)
Free T4	42 pmol/L	(10 - 24.5)

Given the likely diagnosis, what is the optimal treatment approach to address the underlying cause?

A. Propranolol
B. Propylthiouracil
C. Radioiodine therapy
D. Repeat carbimazole treatment
E. Surgery

C. Radioiodine therapy

Radioiodine therapy is the treatment of choice for patients with a relapse of Graves disease in the absence of contraindications, such as pregnancy and active severe Graves ophthalmopathy

Radioiodine therapy is the correct answer. This patient has a relapse of Graves' disease. Guidelines recommend that in those patients who have a relapse of disease despite a course of antithyroid drug treatment, definitive management should be instituted in the form of radioiodine treatment or surgery. This patient's many medical problems including ischaemic heart disease and morbid obesity make her a significant anaesthetic risk and therefore radioiodine therapy would be preferable.

Surgery is incorrect. This is an option for the treatment of relapsed Graves' disease. However, she would be a high-risk surgical candidate and therefore radioiodine therapy would be preferable.

Repeat carbimazole treatment is incorrect. Definitive management with radioiodine therapy or surgery is preferred in the setting of relapse of Graves' disease.

Propylthiouracil is incorrect. Definitive management with radioiodine therapy or surgery is preferred in the setting of relapse of Graves' disease.

Propranolol is incorrect. This would control some of the symptoms of hyperthyroidism without treating the underlying cause.

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A 35-year-old woman presents to the emergency department complaining of shortness of breath and feeling unwell. She has a past medical history of type 1 diabetes mellitus. She is poorly compliant with insulin and has an erratic lifestyle.

Observations:

- Heart rate 89 beats per minute
- Blood pressure 111/77 mmHg
- Oxygen saturations 96% on room air
- Respiratory rate 24/minute
- Temperature 37C

VBG:

pH	7.05	(7.35-7.45)
Lactate	2.8 mmol/L	(0.0-2.0)
HCO3-	8 mmol/L	(22-26)
K+	4.2 mmol/L	(3.5-5.5)
Na+	128 mmol/L	(135-145)
Glucose	31 mmol/L	(4.0-7.8)
Ketones	4.2 mmol/L	(<0.6)

She is commenced on IV fluids (IV 0.9% sodium chloride with added potassium) and a fixed rate insulin infusion.

Repeat investigations four hours post-treatment:

VBG:

pH	7.12	(7.35-7.45)
Lactate	2.2 mmol/L	(0.0-2.0)
HCO3-	12 mmol/L	(22-26)
K+	4.6 mmol/L	(3.5-5.5)
Na+	131 mmol/L	(135-145)
Glucose	10 mmol/L	(4.0-7.8)
Blood ketones	3.2 mmol/L	(<0.6)

Given the likely diagnosis, what is the most appropriate management choice at this point?

A. Continue current management and add IV dextrose

B. No changes to current management
C. Stop FRII and continue 0.9% sodium chloride with addedpotassium
D. Stop current management and switch to IV dextrose
E. Switch to VRII, continue current IV fluids and add IV dextrose

A. Continue current management and add IV dextrose

DKA in the acute setting fixed rate insulin should be continued even if BM levels are <14; IV dextrose should be added instead

Continue FRII and add IV dextrose is correct. The patient has not yet had a resolution of ketonaemia or acidosis. The underlying pathophysiology of DKA is insulin deficiency. Without insulin, the body cannot use glucose for energy and therefore fat sources are broken down to use as fuel, prompting the generation of ketones, which in turn cause acidosis. To reverse this process and resolve ketonemia and acidosis, we need to continue insulin. Insulin therapy causes a lowering of blood glucose levels and therefore we should administer IV dextrose alongside insulin in order to prevent hypoglycaemia while allowing continued insulin administration.

No changes to current management is incorrect. Insulin drives glucose into cells and lowers blood glucose levels. We need to continue insulin until the ketonemia has resolved. However, this will cause hypoglycaemia unless we add IV dextrose.

Stop FRII and continue 0.9% sodium chloride with added potassium is incorrect. While this will result in the resolution of dehydration and avoid precipitating hypoglycaemia (as insulin has been stopped), it will not correct the underlying pathophysiological process (insulin deficiency) and the DKA will worsen.

Stop current management and switch to IV dextrose is not the right answer. Stopping insulin will resulting in worsening DKA, compounded by exogenous glucose administration.

Switch to VRII, continue current IV fluids and add IV dextrose is incorrect. It is standard practice now to administer a fixed rate insulin infusion in DKA rather than a variable rate as an FRII has been proven to be superior as it has been shown to have the following benefits:

- Faster resolution of DKA
- Avoids titration against a poor surrogate marker of blood glucose
- Confirms resolution of DKA if the FRII is stopped when blood ketone levels are < 0.6mmol/L.

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A 65-year-old man presents to his general practitioner with polyuria and polydipsia. He has a past medical history of surgically treated bladder cancer, hypertension and chronic kidney disease. His medications include amlodipine, furosemide, atenolol and ramipril.

The examination is unremarkable. Blood tests:

Na +	137 mmol/L	(135 - 145)
K +	4.2 mmol/L	(3.5 - 5.0)
Urea	28.2 mmol/L	(2.0 - 7.0)
Creatinine	312 µmol/L	(55 - 120)
eGFR	21 ml/minute	(>90)
HbA1c	52 mmol/mol	(<48)

What is the most appropriate medication to commence?

A. Empagliflozin
B. Insulin
C. Metformin
D. Pioglitazone
E. Sitagliptin

E. Sitagliptin

T2DM initial therapy: if metformin is contraindicated (and no risk of CVD, established CVD or chronic heart failure) → choice of DPP-4 inhibitor or Pioglitazone or Sulfonylurea or even SGLT-2 (if NICE criteria met)

Sitagliptin is correct. The patient has symptoms and biochemical evidence of type 2 diabetes with an hba1c level > 48 mmol/mol. Typically, patients would be treated with oral anti-diabetic medications in the first instance. Metformin is the usual choice but it is contraindicated in severe renal impairment. There are a number of other medications that can be chosen depending on the patient's profile. Many of them are contraindicated in severe renal impairment. Sitagliptin is the medication that is most appropriate as it can be started 1st line for T2DM when metformin is contraindicated. It can be started at a reduced dose when the eGFR is < 30ml/minute.

Metformin is incorrect. This is contraindicated in severe renal impairment.

Empagliflozin is incorrect. This is typically not initiated in type 2 diabetes if the eGFR is < 45ml/minute.

Insulin is incorrect. This is not typically used 1st line in with the management of type 2 diabetes as oral medications are typically trialled in the first instance.

Pioglitazone is incorrect. This is contraindicated in those patients with a history of bladder cancer.

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A 64-year-old man presents to his general practitioner with polyuria and polydipsia. He has a past medical history of ischaemic heart disease and mitochondrial encephalopathy, lactic acidosis, and stroke-like episodes (MELAS syndrome). He takes aspirin, atenolol and atorvastatin.

The examination is unremarkable. Blood tests:

Hb	133 g/L	Male: (135-180)Female: (115 - 160)
Platelets	189 * 10 /L 9	(150 - 400)
WBC	4.6 * 10 /L 9	(4.0 - 11.0)
Na +	138 mmol/L	(135 - 145)
K +	4.2 mmol/L	(3.5 - 5.0)
Urea	22 mmol/L	(2.0 - 7.0)
Creatinine	210 µmol/L	(55 - 120)
eGFR	31 mL/minute	(> 90)
CRP	4 mg/L	(< 5)
HbA1c	56 mmol/mol	(<48)

What is the most appropriate choice of management?

A. Empagliflozin
B. Gliclazide
C. Metformin
D. Pioglitazone
E. Sitagliptin

A. Empagliflozin

T2DM initial therapy: if metformin is contraindicated + patient has a risk of CVD, established CVD or chronic heart failure → SGLT-2 monotherapy

Empagliflozin is correct. The patient has developed type 2 diabetes. Metformin is contraindicated in lactic acidosis, so it would be best to avoid it in a patient with MELAS syndrome. As the patient has established CVD, empagliflozin is the most suitable choice of initial antidiabetic medication. It can be started if the eGFR is > 30ml/minute in those with established CVD.

Gliclazide is incorrect. This is a possible option for the initial treatment of T2DM where metformin is contraindicated. However, an SGLT-2 inhibitor is a preferred choice in the setting of established CVD.

Metformin is incorrect. Metformin is contraindicated in lactic acidosis, so it would be best to avoid it in a patient with MELAS syndrome.

Pioglitazone is incorrect. This is a possible option for the initial treatment of T2DM where metformin is contraindicated. However, an SGLT-2 inhibitor is a preferred choice in the setting of established CVD.

Sitagliptin is incorrect. This is a possible option for the initial treatment of T2DM where metformin is contraindicated. However, an SGLT-2 inhibitor is a preferred choice in the setting of established CVD.

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A 45-year-old man attends his general practitioner for an annual review of his diabetes for which he takes metformin. He had an NSTEMI three months and has been commenced on aspirin, clopidogrel, bisoprolol and ramipril.

Blood tests 6 months ago:

HbA1c	51 mmol/mol	(<48)

Blood tests today:

HbA1c	47 mmol/mol	(<48)

What is the correct management of type 2 diabetes in this patient at this point?

A. Add empagliflozin
B. Add gliclazide
C. Add pioglitazone
D. Add sitagliptin
E. No changes to current treatment

A. Add empagliflozin

In patients with T2DM, SGLT-2 should be introduced at any point they develop CVD, a high risk of CVD or chronic heart failure

Add empagliflozin is correct. While this patient has well-controlled diabetes on metformin, SGLT-2 inhibitors should be introduced if at any point they developed cardiovascular disease (CVD). As he has had a recent NSTEMI, empagliflozin should be commenced.

Add gliclazide is incorrect. The patient has well-controlled diabetes and so the addition of gliclazide is not warranted.

Add pioglitazone is incorrect. The patient has well-controlled diabetes and so the addition of pioglitazone is not warranted. Additionally, you may be cautious about the possibility of heart failure in this patient.

Add sitagliptin is incorrect. The patient has well-controlled diabetes and so the addition of sitagliptin is not warranted.

No changes to current treatment is incorrect. While the HbA1c is well controlled, if at any point the patient develops CVD, an SGLT-2 inhibitor should be added.

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You review a 73-year-old female in the thyroid clinic. She has a history of hypothyroidism for about 25 years. Her other co-morbidities are COPD and dyslipidaemia. She is taking levothyroxine 125 mcg daily, atorvastatin 20mg daily, budesonide/formoterol inhaler 2 puffs BD and salbutamol inhaler PRN. Her most recent thyroid function tests showed:

TSH	0.4 mIU/l
FT4	29 pmol/l

You decide to reduce the dose of levothyroxine based on these results.

What is the risk of over-replacement with levothyroxine?

A. Weight gain
B. Cognitive impairment
C. Liver failure
D. Osteoporosis
E. Agranulocytosis

D. Osteoporosis

Over-replacement with thyroxine increases the risk for osteoporosis

The major risks of over-replacement with thyroxine include osteoporosis and atrial fibrillation.

Weight gain is a feature of untreated hypothyroidism.

Cognitive impairment is not a recognised adverse effect of thyroxine. Although levothyroxine can rarely cause mild elevations in liver enzymes, there have been no reports of liver failure. Agranulocytosis is caused by anti-thyroid drugs.

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A 32-year-old pregnant woman (26 weeks) presents to her GP because she has been feeling increasingly tired. She is normally fit and well and has had one previous pregnancy which was uneventful. A fasting blood glucose is 5.4 mmol/L. Urine dip is negative and her blood pressure is 134/78 mmHg. Abdominal examination is unremarkable. Blood results are as follows:

Hb	116 g/L	Male: (135-180)Female: (115 - 160)
Platelets	270 * 10 /L 9	(150 - 400)
WBC	7.3 * 10 /L 9	(4.0 - 11.0)
Na +	136 mmol/L	(135 - 145)
K +	3.9 mmol/L	(3.5 - 5.0)
Bicarbonate	25 mmol/L	(22 - 29)
Urea	6.5 mmol/L	(2.0 - 7.0)
Creatinine	89 µmol/L	(55 - 120)
Bilirubin	4 µmol/L	(3 - 17)
ALP	170 u/L	(30 - 100)
ALT	20 u/L	(3 - 40)
Î³GT	55 u/L	(8 - 60)
Albumin	37 g/L	(35 - 50)

Which of the following is the most likely diagnosis?

A. Acute cholecystitis
B. Gestational diabetes
C. Intrahepatic cholestasis of pregnancy
D. Non-alcoholic fatty liver disease
E. Normal pregnancy

E. Normal pregnancy

Normal pregnancy can cause a raised ALP - it doesn't necessarily imply liver problems

The only abnormal finding is a slightly raised ALP. ALP is found in bone, liver, kidney, and intestinal and placental tissue, so ALP tends to rise during pregnancy. In pregnant women levels of up to twice that upper limit can still be normal.

Acute cholecystitis would usually present with abdominal symptoms and right upper quadrant tenderness. It is important to bear in mind that the risk of gallstones increases during pregnancy, as oestrogen can lead to higher cholesterol levels in bile.

Diagnosis (according to NICE) can be made based on either a fasting plasma glucose level of 5.6 mmol/L or above, or a 2-hour plasma glucose level of 7.8 mmol/L or above. Her fasting plasma glucose is below the threshold.

Intrahepatic cholestasis of pregnancy typically presents with itching (particularly in palms and soles) and can lead to complications for both mother and baby if untreated. LFTs are usually deranged but may be normal on some occasions.

Non-alcoholic fatty liver disease (NAFLD) is excessive fat build-up in the liver without another clear cause. NAFLD can progress to non-alcoholic steatohepatitis (NASH). Elevated liver enzymes are common in both but would not typically present with just an isolated rise in ALP. There is also no reason for this young fit woman to have NAFLD.

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A 45-year-old lorry driver was referred to the endocrine clinic with symptoms of reduced libido and lack of energy. He had history of a traumatic head injury 5 years previously, which needed a period of 24 hours observation in hospital. On examination, his body mass index was 42 kg/m with normal general physical and systemic examination.

Investigations:

FT4	8.1pmol/l (11.5-22.7)
TSH	0.4mU/l (0.35-5.5)
FSH	2.2U/l (1.4-18.1)
LH	3.5U/l (3.0-8.0)
Testosterone	6.8nmol/l (8.4-28.7)
IGF-1	35nmol/l (16-118)
Prolactin	880mU/l (45-375)

Which one of the following is the most likely diagnosis, based on his clinical profile?

- Microprolactinoma
- Morbid obesity
- Non-functioning pituitary adenoma
- Post traumatic pituitary apoplexy
- Primary hypothyroidism

C. Non-functioning pituitary adenoma

The presence of an elevated prolactin level along with secondary hypothyroidism and hypogonadism is indicative of stalk compression is consistent with a non-functioning pituitary adenoma

This gentleman has secondary hypothyroidism and hypogonadism in the presence of elevated prolactin levels, which are indicative of stalk compression due to a pituitary adenoma. A prolactinoma is generally associated with prolactin levels >2000 mU/L. Morbid obesity may be associated with hypogonadotrophic hypogonadism, although secondary hypothyroidism is unlikely. Pituitary apoplexy is characterized by a sudden onset of headache, visual symptoms, altered mental status, and hormonal dysfunction due to acute hemorrhage or infarction of a pituitary gland. It usually presents with biochemical features of hypopituitarism which is not presented in this scenario. Primary hypothyroidism is characterized by low FT4, low FT3 and high TSH.

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A 62-year-old man presents to hospital with a three-day history of tiredness, muscle aches, a fever and pain at the front of his neck. Two weeks ago he had an upper respiratory tract infection which he treated himself with paracetamol and oral decongestants. He did not receive any antibiotics. His only past medical history is mild arthritis of the right knee and he only takes occasional antihistamines during summer time.

On examination, he appears anxious with a fine resting tremor. He has a temperature 38.1°C, a pulse rate of 125 per minute which is regular and normal in character and a blood pressure of 131/78 mmHg. Heart sounds 1 and 2 were present with no added sounds and his chest was clear on auscultation. His abdomen was soft and non-tender with no organomegaly. Neurological examination was unremarkable apart from the resting tremor. Neck examination reveals a diffusely enlarged and tender thyroid gland.

Blood tests are requested and the results are as follows:

Hb	14.0 g/dl
Platelets	378 * 10 /l9
WBC	8.9 * 10 /l9
ESR (Westergren)	94 mm/1st hour
Free T4	214 nmol/l
Free T3	192 nmol/L
Plasma TSH	<0.05 mU/l

Which of the following investigations is likely to be most helpful in establishing the diagnosis?

A. Blood cultures
B. Serum anti-thyroid antibodies
C. Ultrasound scan of neck
D. Radioactive iodine uptake scan
E. Fine needle aspiration

D. Radioactive iodine uptake scan

In De Quervain's thyroiditis there is globally reduced uptake of iodine-131 during thyroid scintigraphy

The patients presenting symptoms and signs are typical of a diagnosis of subacute thyroiditis, of which the most likely cause of this is De Quervains thyroiditis.

Radioactive iodine uptake scan is the most suitable investigation for confirming subacute thyroiditis which demonstrates reduced intake.

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A 60-year-old man attends a medical health check-up at his GP surgery. He was fit and well with a past medical history of childhood asthma and osteoarthritis in his fingers. His observations were included a blood pressure of 129/80 mmHg, pulse of 82 bpm, and oxygen sats of 97%.

Blood tests were performed and revealed:

Hb	138 g/l	
Platelets	190 * 10 /l9	
WBC	7.6 * 10 /l9	
Na +	139 mmol/l	
K +	3.9 mmol/l	
Urea	4.1 mmol/l	
Creatinine	92 µmol/l	
Bilirubin	15 µmol/l	
ALP	52 u/l	
ALT	26 u/l	
γGT	58 u/l	
Albumin	40 g/l	
Serum corrected calcium	2.77 mmol/L	
Serum phosphate	0.90 mmol/l	
Parathyroid hormone	5.9 pmol/L	normal range 1.2-5.8 pmol/L

A 24 hour urinary calcium test was performed based on the results above and revealed a result of 0.5 mmol/24 hours (normal range 2.4-7.4 mmol/24 hours)

What is the most likely diagnosis?

A. Primary hyperparathyroidism
B. Secondary hyperparathyroidism
C. Vitamin D toxicity
D. Multiple endocrine neoplasia type I
E. Familial benign hypocalciuric hypercalcaemia

E. Familial benign hypocalciuric hypercalcaemia

Patients with familial benign hypocalciuric hypercalcaemia may have a normal or raised PTH

The most likely diagnosis in this scenario is familial benign hypocalciuric hypercalcaemia. Most cases are asymptomatic and blood test reveals hypercalcaemia with a reduced calcium urinary excretion rate (of under 0.02 mmol/L). There may also be normal to high parathyroid hormone, despite the elevated serum calcium levels.

Question 43 of 249

A 21-year-old female presents with a vague two month history of lethargy, muscle pain and weight loss. Examination is unremarkable. Observations included heart rate 72/min, respiratory rate 14/min, oxygen saturations are 99% on air, blood pressure of 110/80mmHg. She was apyrexial.

Routine bloods are sent.

Hb	150 g/l	Na +	128 mmol/l
Platelets	200 * 10 /l 9	K +	2.9 mmol/l
WBC	12.0 * 10 /l 9	Urea	7.0 mmol/l
Neuts	8.0 * 10 /l 9	Creatinine	85 µmol/l
Lymphs	4.0 * 10 /l 9	CRP	11 mg/l

What is the next most appropriate investigation?

A. Computed tomography (CT) of the thorax
B. Ultrasound of the renal tract
C. Serum aldosterone
D. Urinary electrolytes
E. Short synacthen test

D. Urinary electrolytes

The combination of lethargy, weight loss, low sodium and low potassium in a young patient suggest an inherited renal pathology such as Bartter's or Gitelman's syndrome. Liddle's syndrome is usually associated with high blood pressure.

Urinary electrolytes are a quick and simple test and would be likely to aid the diagnosis here.

Most other causes alluded to here would not cause both sodium and potassium to be low.

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A 77-year-old female is admitted to the acute medal unit with a chest infection. Her past medical history includes COPD and heart failure. Her current medications include salbutamol, tiotropium, bisoprolol and ramipril. On examination you note left basal crepitations. The JVP is at 2cm above the sternal angle. There is no peripheral oedema. Her blood pressure is 145/85 mmHg.

You note that her blood results are as follows:

Na +	120 mmol/l
Urine osmolarity	400 mosmol/l
Urine sodium	50 mosmol/l
Urea	6.2 mmol/l
Creatinine	54 µmol/l
fT3	6.2 pmol/l (normal range 3.5 - 7.8)
fT4	6 pmol/l (normal range 9.0 - 25.0)
TSH	0.2 mU/l (normal range 0.4 - 4.0)
morning cortisol	normal

You water restrict the patient to 1.5 litres per day. On day 4 her bloods are reported as follows:

Na +	118 mmol/l

How would you manage this patient?

A. Furosemide
B. Levothyroxine
C. Demeclocycline
D. Hypertonic saline (3%)
E. Hypotonic saline (0.45%)

C. Demeclocycline

If SIADH is resistant to fluid restriction then the drug demeclocycline can be used

The differential for hyponatraemia is wide. The fluid status of the patient is of paramount importance for identifying the cause. In this example, the patient is clearly euvolaemic. Causes of euvolaemic hyponatraemia include SIADH, hypothyroidism, and adrenal insufficiency. The serum and urine osmolarities, and urinary sodium are consistent with either of these diagnoses.

SIADH is effectively a diagnosis of exclusion - primary hypothyroidism and hypoadrenalism must be excluded to make the diagnosis.

In this case, the thyroid function tests are suggestive of euthyroid sick syndrome. This would not be an indication for initiating levothyroxine. Although hypothyroidism is a well-known cause of hyponatraemia euthyroid sick syndrome would not effect sodium balance. It is often stated that to make a diagnosis of SIADH relies upon 'normal thyroid function tests'. It is important to note that this is referring to primary hypothyroidism. Euthyroid sick syndrome does not preclude the diagnosis of SIADH.

The morning cortisol is normal which suggests that hypoadrenalism is not present.

Therefore the most likely diagnosis is SIADH secondary to a chest infection. The initial treatment for non-severe SIADH is fluid restriction.

This patient has been trialled with fluid restriction however the sodium has not improved and has in fact got worse. The next step in the management would be demeclocycline. Demeclocycline inhibits the effects of ADH on the renal tubules. It effectively causes a nephrogenic diabetes insipidus.

The patient's symptoms are not severe enough to warrant use of hypertonic saline. Hypertonic saline has risks associated with it, as rapid correction of serum Na can result in central pontine myelinolysis.

If the patient had been hypervolaemic then furosemide would be indicated.

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A 45-year-old female presents to the clinic with right sided flank pain. A CT KUB confirms a right sided kidney stone. She has a past medical history of asthma, rheumatoid arthritis and Sjogren's syndrome. Her drug history includes salbutamol, methotrexate and hydroxychloroquine.

Blood results are as follows:

Hb	115 g/l	Na +	136 mmol/l
Platelets	460 * 10 /l 9	K +	3.1 mmol/l
Bicarbonate	16 mEq/L (normal 22-28)	Urea	7.8 mmol/l
Chloride	115mmol/l	Creatinine	76 µmol/l

How would you prevent further kidney stones?

A. Thiazide diuretics
B. Loop diuretics
C. Penicillamine
D. Encourage increased fluid intake
E. Sodium bicarbonate

E. Sodium bicarbonate

Treatment of RTA involves correction of the acidaemia with oral sodium bicarbonate, sodium citrate or potassium citrate

The patient has a low bicarbonate which confirms a metabolic acidosis. This should prompt you to work out the anion gap which can be extremely useful diagnostically.

The anion gap in this example = sum of positive cations - sum of negative anions = (Na + K) - (HCO3 + Cl) = 9.1 We are therefore dealing with a normal anion gap metabolic acidosis (NAGMA).

High anion gap metabolic acidosis (HAGMA) is generally caused by the following conditions which can be remembered with the mnemonic KULT

- Ketoacidosis: diabetic, starvation or alcoholic
- Uraemia
- Lactic acidosis
- Toxins e.g. salicylates, ethylene glycol, methanol

Normal anion gap metabolic acidosis (NAGMA) is generally caused by the following conditions which can be remembered with the mnemonic ABCD

- Addison's disease
- Bicarbonate loss via GI tract (e.g. diarrhoea) or renal tract (e.g. renal tubular acidosis)
- Chloride excess - hyperchloraemic acidosis
- Drugs e.g. acetazolamide

In this case, the NAGMA, hypokalaemia, and renal stones should prompt you to consider renal tubular acidosis (RTA). The presence of kidney stones, and the past medical history of Sjogren's syndrome and rheumatoid arthritis make RTA type 1 (distal) the most likely diagnosis. Treatment of RTA involves correction of the acidaemia with oral sodium bicarbonate, sodium citrate or potassium citrate.

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A 27-year-old female presents with secondary amenorrhoea after stopping the oral contraceptive pill 6 months ago. She gets regular headaches and struggles to stand from seated or climb stairs.

On examination, milk could be expressed from the breasts and visual fields showed bilateral defects in the upper outer quadrants.

Prolactin	1080 mIU/L (NR<360)
FSH	0.1 IU/L (NR 1-11)
LH	0.2 IU/L (NR 20-75)
TSH	0.1 mIU/L (NR 0.3-6.0)
T4	8 pmol/L (NR 10-25)
9am cortisol	20 nmol/L (NR 140-700)

Pituitary MRI: 3cm pituitary mass with tenting of optic chiasm.

What is the next step in management?

A. Bromocriptine
B. Octreotide
C. Stereotactic radiotherapy
D. Trans-sphenoidal surgery
E. Transcranial hypophysectomy

D. Trans-sphenoidal surgery

This patient has a macroadenoma (>1cm) causing visual field defects. Trans-sphenoidal surgery is the first step in management. Raised prolactin can be secondary to blockage of the pituitary stalk with prevention of dopamine reaching the pituitary causing disinhibition of the lactotrophs.

Prolactin secreting macroadenomas secrete very high quantities and PRL is usually >6000mU/ml particularly with macro-prolactinomas (this is not the case making a prolactinoma unlikely). Prolactinomas are treated with dopamine agonists (bromocriptine, cabergoline) first-line. Octreotide is used to treat acromegaly.

Transcranial hypophysectomy is done for very large tumours that cant be removed via the trans-sphenoidal route.

Question 47 of 249

A 24-year-old lady presents to hospital with increasing confusion. Her parents describe a gradual history of weight loss, lethargy with abdominal cramping. She has no past medical history and is prescribed no regular medications. She lives with her parents. Her mother suffers from hypothyroidism and her father from hypertension which is controlled with bendroflumethiazide.

On examination she is thin, with cool skin and sunken eyes. Her capillary refill time is 3 seconds with dry mucous membranes. Auscultation of her chest reveals bilateral symmetrical vesicular breath sounds. Her abdomen is soft with normal bowel sounds. She is confused with a Glasgow Coma Scale of 14. She has no focal neurology.

Her investigations reveal;

Hb	10.4 g/dL
MCV	90 fL
WCC	6.4 *10^9/l
Platelets	170 *10^9/l
Na+	105 mmol/L
K+	5.8 mmol/L
Ur	8.8 mmol/L
Cr	90 µmol/L
Glucose	3.9 mmol/L
Urinary Osmolality	108 mmol/L
Urinary Sodium	67 mmol/L
Chest X ray	Clear
CT Head	No intracranial abnormalities

What is the most likely diagnosis?

A. Adrenal insufficiency
B. Laxative abuse
C. Hypothyroidism
D. Diuretic use
E. SiADH

A. Adrenal insufficiency

This lady has hypovolaemic hyponatraemia. Sodium can be lost through the gastrointestinal tract, skin or urinary tract. This patient is likely to have renal loss secondary to adrenal insufficiency.

The urinary sodium would be reduced if there were gastrointestinal or skin losses. Also, the patient would be hypokalaemic if there were laxative abuse. The patient would be expected to have hypokalaemia if there were abuse of bendrofumethiazide.

The patient is hypovolaemic and therefore SiADH is excluded, also, hypothyroidism and adrenal insufficiency have not been excluded which would be necessary to make the diagnosis of SiADH.

Hypothyroidism classically presents with weight gain and euvolaemic hyponatraemia.

This patient has adrenal insufficiency. It is possibly of an autoimmune nature with a probable autoimmune hypothyroidism in the immediate family. She has a gradual onset of non-specific symptoms lethargy, weight loss and abdominal symptoms. The hyponatraemia has caused confusion. The blood tests reveal hypoglycaemia, hyponatraemia with hyperkalaemia and normocytic anaemia. Examination revealed signs of hypovolaemia, it also could show skin or mucus membrane pigmentation.

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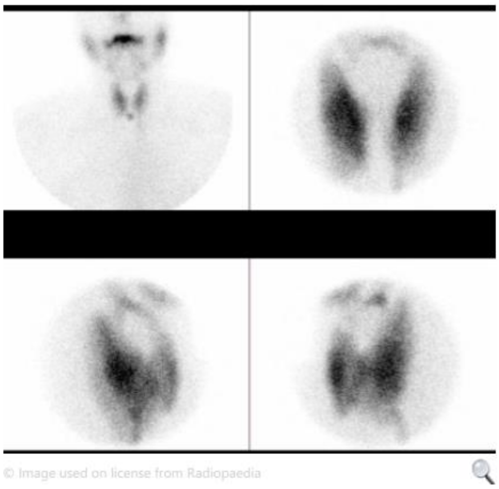
A 43-year-old woman presents to the endocrinology clinic after being referred by her general practitioner. She has suffered from a 2-month history of weight loss, sweating, and heat intolerance. She notes that she suffered from a cold-like illness a few months ago but is otherwise well and has no relevant past medical history.

Her blood tests with the general practitioner are displayed below:

TSH	0.2 mU/L	(0.4 - 2.0)
Free T4	31 pmol/L	(12 - 22)

Her thyroid scintigraphy is displayed below:



What is the most likely diagnosis?

A. Grave's disease
B. Hashimoto's thyroiditis
C. Subacute thyroiditis
D. Thyroid cancer
E. Toxic multinodular goitre

A. Grave's disease

This woman's clinical presentation and thyroid function tests are consistent with a diagnosis of hyperthyroidism. The thyroid scintigraphy shows global uptake which is consistent with Grave's disease. Grave's disease is therefore the correct answer.

Hashimoto's thyroiditis is a cause of hypothyroidism rather than hyperthyroidism. This cannot be the case as confirmed by the thyroid function tests and is therefore incorrect.

Subacute thyroiditis is a possibility due to this woman's recent illness. This often results in subacute hyperthyroidism followed by a period of hypothyroidism. Scintigraphy shows decreased uptake in this condition which is not the case in this instance. This is therefore not the correct answer.

Thyroid cancers can cause hyperthyroidism however scintigraphy would show a discrete mass with increased uptake rather than homogenous uptake. This is not the case here and therefore Grave's disease is a more likely diagnosis.

Toxic multinodular goitre is another cause of hyperthyroidism however is associated with patchy uptake on thyroid scintigraphy. As the uptake in this instance is homogenous, this is unlikely to be the underlying cause.

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A 42-year-old woman is referred to the endocrine clinic. She is treated with lithium for bipolar disorder and presents with weight gain, lethargy, a dry cough and a hoarse voice over the past 3 months. Examination reveals patchy hair loss, a smooth goitre, and she is overweight with a body mass index of 32 kg/m². Her blood pressure is 122/82 mmHg, pulse is 60 beats per minute.

Investigations:

Na+	130 mmol/l
TSH	14.2 mIU/l

Which of the following is the most appropriate way to manage her?

A. Iodine supplementation
B. Start prednisolone
C. Stop lithium
D. Surgical thyroidectomy
E. Start thyroxine

E. Start thyroxine

Lithium increases intrathyroidal iodine content, inhibits the coupling of iodotyrosine residues to form iodothyronines (thyroxine and triiodothyronine), and it also inhibits release of T4 and T3 leading to hypothyroidism, the clinical picture seen here. The most appropriate intervention is thyroxine supplementation.

There is no need acutely to stop lithium therapy, although substitution may eventually be considered after discussion with the patient's psychiatrist. Due to the fact that lithium prevents coupling of iodotyrosine, iodine supplementation is ineffective.

Prednisolone is also not of value because thyroiditis isn't the cause of the hypothyroidism seen here. Surgical thyroidectomy isn't indicated because there are no symptoms of extrinsic airway obstruction.

Question 50 of 249

A 54-year-old man presents to the diabetes clinic for review. He has had symptoms of polyuria, polydipsia and lethargy over the past few months, and his fasting glucose is elevated at 7.6 mmol/l. He has no history of diabetes in his family and is currently treated for hypertension and dyslipidaemia by his GP. On examination his blood pressure is 155/90 mmHg, pulse is 70 beats per minute and regular. His body mass index is 34 kg/m². Other blood tests of note include GAD+ antibodies, renal function is normal.

Which of the following is most appropriate with respect to managing his glucose control?

A. Gliclazide
B. Liraglutide
C. Metformin
D. Sitagliptin
E. Basal bolus insulin

C. Metformin

Up to 10% of patients who are thought to have type 2 diabetes are found to also be GAD autoantibody positive. These individuals are thought to progress faster to insulin start than patients with autoantibody negative type 2 diabetes, (within 3-5 years vs 7 years on average for 'standard' patients).

The management of these patients is the same as for patients with autoantibody negative disease, metformin as the initial therapy of choice. Weight reduction may delay progression to insulin, therefore insulin sparing strategies, at least during the first few years after diagnosis are the preferred intervention. These may include use of SGLT- 2 inhibitors or GLP-1 agonists.

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A 56-year-old man with a history of type 2 diabetes managed with Humalog mix 30 and metformin 1g BD comes to the clinic for review. HbA1c is currently 57. He has troublesome hypoglycaemia episodes in the late afternoon and early mornings and wants to know what to do about it. On examination his blood pressure is 132/82 mmHg, his pulse is 72 beats per minute and regular. His body mass index is 32 kg/m².

Investigations

Na +	139 mmol/l
K +	4.9 mmol/l
Urea	5.1 mmol/l
Creatinine	94 µmol/l
HbA1c	57 mmol/mol

Which of the following is the most appropriate next step in his management?

A. Continue current regimen, eat a snack mid afternoon and before bed time
B. Reduce the dose of metformin
C. Reduce the dose of mixed insulin
D. Switch to a basal bolus regimen
E. Switch to Humalog mix 20

D. Switch to a basal bolus regimen

This person's HbA1c is above target at 57 mmol/mol or 7.4%. In spite of this, he is still suffering from hypoglycaemia episodes as a result of delivering his insulin as a twice a day regimen. Moving him to basal bolus will allow splitting up of the short acting component of his insulin requirements, and therefore reduce the risk of hypoglycaemia at the end of each dosing period.

Reducing the dose of his mixed insulin may drive a further rise in his HbA1c, as would reducing the dose of metformin. Switching to Humalog mix 20 will increase the proportion of long-acting insulin and worsen the risk of hypoglycaemia. Eating a snack will drive weight gain and worsen his HbA1c.

Question 52 of 249

A 22-year-old woman with a history of partial Kallmann syndrome comes to the fertility clinic for review. She got married some 6 months earlier and wants to start a family. She has normal external genitalia and sparse pubic and axillary hair and has a normal body mass index of 23kg/m².

Which of the following is the most appropriate intervention?

A. Clomiphene
B. HCG and FSH then IVF
C. Metformin
D. Oestrogen
E. Referral for adoption

B. HCG and FSH then IVF

The correct answer is HCG and FSH then IVF. Restoration of ovulation in females with Kallmann syndrome is complex and often requires HCG to drive production of gonadal steroid hormones, FSH to drive ovulation, harvesting of eggs, and IVF. This process is most effective in achieving successful pregnancy.

Clomiphene does induce ovulation and is useful in patients with other conditions such as polycystic ovarian syndrome. It is however very unlikely to be effective in patients with gonadotrophin failure like Kallmann syndrome. Metformin has previously been used in the management of PCOS. Oestrogen doesn't restore ovulation in patients with Kallmann syndrome but may be considered for patients who don't want to get pregnant. In the event that fertility treatment isn't successful, adoption can be considered.

Question 53 of 249

A 35-year-old woman is referred to the endocrinology clinic after having lost 5kg without any change in diet or exercise habits. On direct questioning, she also describes that she has noticed feeling warmer, losing sleep, and a slight tremor in her hands. She has a past medical history of depression for which she takes sertraline. She has no other medical problems. On examination, she has a body mass index of 24 kg/m², a slight tremor at rest, and a large goitre. This is non-tender and there are no palpable nodules. There is no lymphoadenopathy present. Blood tests show a normal full blood count and electrolytes, but TSH is undetectable and free T4 is 39pmol/l. She is clinically diagnosed with hyperthyroidism.

What further investigation is most appropriate to determine the type of her hyperthyroidism?

A. Thyroid-stimulating hormone receptor antibodies
B. Liver function tests
C. CT neck
D. Scan of radionuclide thyroid uptake
E. US-guided fine needle aspiration cytology

A. Thyroid-stimulating hormone receptor antibodies

TSH antibodies are found in 90% of patients with Graves' disease and can help distinguish from other forms of hyperthyroidism

This patient clinically and biochemically has hyperthyroidism. To help determine the cause of hyperthyroidism, and distinguish between causes such as Graves' disease, toxic adenoma and toxic multinodular goitre. Thyroid-stimulating hormone receptor antibodies (TSH-RAb, commonly shortened to TRAb) has an up to 98% sensitivity and 99% specificity for Graves' disease. If this test is negative, then a radionuclide thyroid uptake scan can help determine the cause. In Graves' disease there is diffuse

high uptake, in thyroiditis there is low uptake, and if there are nodules then there is uneven uptake. US-guided fine needle aspiration cytology is recommended in the presence of features suggestive of malignancy. Liver function tests are important if there is a history of liver disease as carbimazole can cause further liver injury, but it would not help in determining the cause of hyperthyroidism.

Question 54 of 249

A 54-year-old woman was admitted following abnormal blood tests on routine monitoring. She is currently undergoing palliative chemotherapy with FOLFIRINOX, a combination of folinic acid, fluorouracil, irinotecan and oxaliplatin for metastatic pancreatic cancer. She has suffered from diarrhoea, vomiting, nausea and fatigue since starting treatment. She has a past medical history of hypothyroidism, epilepsy and bipolar disorder. Her regular medications include levothyroxine, levetiracetam, lithium, loperamide, paracetamol, oramorph, zomorph, movicol and ondansetron.

Na +	142mmol/l
K +	3.8mmol/l
Urea	4.4mmol/l
Creatinine	83µmol/l
Corrected calcium	3.3µmol/l

She is started on treatment with IV 0.9% saline and given 90mg of IV alendronate after further blood tests are sent for, and an ECG is done.

Which of her regular medications should not be prescribed on admission?

A. Zomorph
B. Levetiracetam
C. Loperamide
D. Levothyroxine
E. Lithium

E. Lithium

This patient has developed hypercalcaemia in the context of cancer but whilst also taking lithium. Lithium can be a cause of elevated calcium and should, therefore, be held in the acute context until the cause of hypercalcaemia is established. Note that the serum concentration of lithium does not correlate with the likelihood of it being a cause of hypercalcaemia. In the context of new confusion or dehydration, withholding the opiate medications would have been reasonable.

Question 55 of 249

A lady who is 10 weeks pregnant presents to her antenatal appointment asking for advice regarding gestational diabetes. She is a 31-year-old white woman with a BMI (body mass index) of 28.7. In terms of family history, she has a cousin who has type 1 diabetes mellitus and an aunt who is being treated for breast cancer. She has had two previous pregnancies, the first one she, unfortunately, miscarried at 8 weeks, and the second was a normal pregnancy that she took to term with a birth weight of 4.6kg. Neither of these pregnancies was complicated by gestational diabetes, and the baby is now 2 years old and has not had to be taken to see a doctor other than routine appointments.

What is the most appropriate testing regime for ruling out gestational diabetes in this woman?

A. Oral glucose tolerance test at 24-28 weeks pregnant
B. None - as she has no risk factors for gestational diabetes
C. Oral glucose tolerance test at 12-14 weeks pregnant
D. Self-monitoring of sugars and repeat appointment in 2 weeks
E. HBa1c

A. Oral glucose tolerance test at 24-28 weeks pregnant

This question requires knowledge on the risk factors for developing gestational diabetes and appropriate testing based on risk:

Risk factors for gestational diabetes include:

- BMI >30kg/m²
- Previous delivery of a baby over 4.5kg - which qualifies this patient Previous gestational diabetes
- Family history of diabetes (1st degree relative)
- Minority ethnic family origin with a high prevalence of diabetes

If any one of these risk factors is present then one should offer testing for gestational diabetes. The gold standard testing for patients with risk factors is 2-hour 75g oral glucose tolerance test (OGTT) at 24-28 weeks gestation. If the patient has had gestational diabetes in a previous pregnancy then early-self monitoring of blood glucose or OGTT as soon as possible after booking could also be used for diagnosis.

A diagnosis of gestational diabetes is made if the patient has either:

- Fasting glucose of 5.6 mmol/L or above OR
- A 2-h plasma glucose of 7.8 mmol/L or above

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A 19-year-old woman presents for review. Her past medical history includes 11-beta-hydroxylase deficiency and hypertension, which is managed with ramipril and indapamide. The 11-beta-hydroxylase deficiency was identified at birth upon identifying clitoromegaly.

Which of the following is likely to be raised most markedly?

A. 17-OH pregnenolone
B. Oestradiol
C. 11-deoxycortisol
D. 17-OH progesterone
E. Oestrone

C. 11-deoxycortisol

11 beta-hydroxylase is responsible for the conversion of 11-deoxycorticosterone and 11-deoxycortisol to corticosterone and cortisol. In patients with 11-beta-hydroxylase deficiency, this conversion does not occur in sufficient amounts and levels of these steroids accumulate in the patient. Therefore, although 17-OH hormones may also be raised, the 11-deoxycortisol is more significantly raised than the others.

Question 57 of 249

A 24-year-old woman presents to hospital after collapsing while out shopping. On taking her observations, she has a temperature 37.2°C, a pulse rate of 78 per minute which is regular and normal in character and a blood pressure of 164/92 mmHg. Heart sounds 1 and 2 were present with no added sounds and his chest was clear on auscultation. Her abdomen was soft and non-tender with no organomegaly. Neurological examination was unremarkable. She has no past medical history of note and is on no regular medications.

Further blood tests reveal low renin and aldosterone levels, hypokalaemia and a serum bicarbonate of 30 mmol/l.

Which of the following is the most appropriate treatment for her condition?

A. Angiotensin converting enzyme inhibitor therapy
B. Bumetanide
C. Potassium replacement
D. Spironolactone
E. Amiloride

E. Amiloride

This woman has presented with the combination of hypokalaemic alkalosis, suppressed renin and aldosterone levels in the presence of hypertension indicates a diagnosis of Liddle syndrome. Hypertension and hypokalaemia respond well to amiloride. Spironolactone is not as effective as this medication acts on the mineralocorticoid receptor, as opposed to amiloride, which acts directly on the sodium channel.

Question 58 of 249

A 24 year-old man presents with a five week history of increasing thirst and frequency of urinating. The GP suspects diabetes and performs two fasting blood tests on separate days which reveal blood glucose results of 8.7 mmol/l and 9.2 mmol/l. Urinalysis does not detect any ketones or protein in the urine. The patient's mother had a diagnosis as type 1 diabetes at the age of 22 and his maternal grandfather and aunt also have type 1 diabetes. Due to the family history, the patient's c-peptide is measured and found to be consistently high on two occasions.

Given the likely diagnosis, what is the most appropriate first treatment for managing this condition?

A. Gliclazide
B. Metformin
C. Pioglitazone
D. Insulin
E. Sitagliptin

A. Gliclazide

The most likely diagnosis in this scenario is maturity onset diabetes of youth, due to the persistently raised c-peptide and strong family history. It normally presents in early adulthood and 1/3 of cases can be treated with oral hypoglycaemics, such as sulphonylureas. It is therefore important to see the response to sulphonylureas first before commencing insulin therapy.

Question 59 of 249

A 49-year-old man presents with feeling under the weather. On further questioning he reports that he has been feeling tired and weak for the past few weeks and his wife has noticed that he as lost some weight, although he states that his appetite has not decreased and if anything he is feeling more thirsty and going to the toilet several times a night. Over the last couple of days he has noticed a rash develop around his groin, which is now present on his buttocks. On examination, there are patches of red with irregular borders and crusting.

A fasting blood test is arranged and reveals a blood glucose of 9.2 mm/l.

What is the next most appropriate investigation?

A. Plasma insulin level
B. Tissue transglutaminase antibody (TTA) test
C. Plasma glucagon level
D. Skin biopsy
E. Plasma zinc level

C. Plasma glucagon level

This patient has a diagnosis of a glucagonoma. The polydipsia and polyuria, coupled with the fasting blood glucose result suggest diabetes and the rash is necrolytic migratory erythema a symptom which is the presenting problem in up to 70% of cases of glucagonoma.

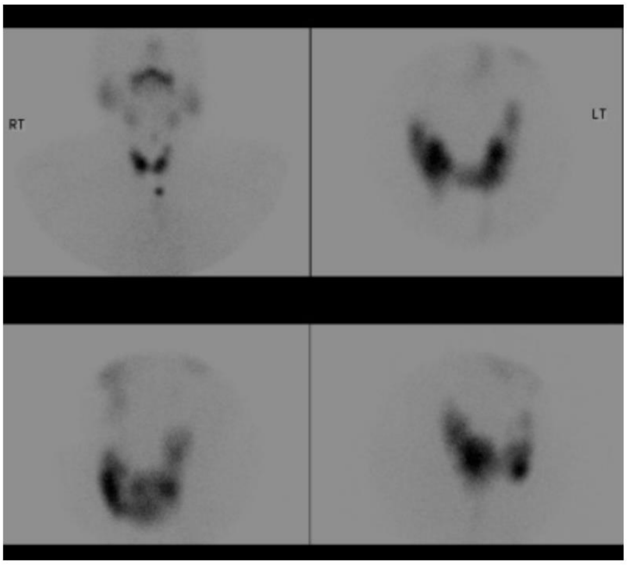
Question 60 of 249

A 54-year-old woman has attended a follow-up endocrinology clinic after undergoing investigations for hyperthyroidism. She initially presented with a 4-month history of intermittent palpitations, sweating, and fatigue. She had noticed that over this time she had developed a tremor and had lost approximately 6kg in weight.

Thyroid function tests from today's clinic are shown below.

TSH	0.01 mU/L (0.5-5.5)
Free T4	28.3 pmol/L (9.0 - 18)

Images from her nuclear scintigraphy are also available.



Which of the following is the most likely cause for this woman's presentation?

A. Grave's disease
B. Hashimoto's thyroiditis
C. Solitary toxic nodule
D. Follicular thyroid cancer
E. Toxic multinodular goitre

E. Toxic multinodular goitre

The nuclear scintigraphy images show multiple localised areas of increased uptake throughout both lobes of a normal-sized thyroid gland. This, along with the symptoms and thyroid function tests, would be in keeping with toxic multinodular goitre.

The appearance of Grave's disease commonly displays homogenous uptake throughout the whole thyroid gland, which itself will be enlarged.

Hashimoto's thyroiditis can vary with its appearance on nuclear scintigraphy, thus being an option using images alone. However, this patient has symptoms of hyperthyroidism along with thyroid function tests that confirm this. Hashimoto's thyroiditis is more likely to lead to symptoms of hypothyroidism due to autoimmune destruction of the thyroid gland.

A solitary toxic nodule shows a single, defined area of increased iodine uptake on nuclear scintigraphy.

Follicular thyroid cancer is not correct as it does not commonly show evidence of increased uptake of iodine on nuclear scintigraphy. Symptoms are also rare with the most common being that of a noticeable lump in the thyroid gland.

Question 61 of 249

A 78-year-old man is admitted with severe diarrhoea. He states that he has had profuse diarrhoea for the past 6 days. He denies blood or mucus in his stools. On examination he is peripherally shut down. His blood pressure is 85/45 mmHg and heart rate is 125 beats per minute. Blood results are as follows:

Hb	128 g/l	Na^+	112 mmol/l
Platelets	$488 * 10^9/l$	K^+	3.2 mmol/l
WBC	$14.8 * 10^9/l$	Urea	18.8 mmol/l
Neuts	$10.8 * 10^9/l$	Creatinine	212 µmol/l
Lymphs	$2.2 * 10^9/l$	CRP	78 mg/l

You diagnose hypovolaemic shock and resuscitate him with 0.9% saline boluses.

Later on in the day the patient presents with muscle weakness. On examination you note spastic quadriparesis.

What is the most likely cause?

A. Cerebral oedema
B. Central pontine myelinolysis (CPM)
C. Cerebral vasospasm
D. Cerebral hypoxia secondary to prolonged hypotension
E. Brain stem infarct

B. Central pontine myelinolysis (CPM)

Rapid correction of hyponatraemia can result in central pontine myelinolysis (CPM). CPM features are variable, however the classical presentation is spastic quadriparesis, pseudobulbar palsy, and emotional lability (pseudobulbar affect)

The patient has marked hypovolaemic hyponatraemia secondary to profuse diarrhoea. Large volumes of saline has resulted in rapid correction of the hyponatraemia. Sudden rises in plasma osmolarity can result in fluid shift from the cerebral intracellular space to the extravascular space resulting in cerebral shrinking and demyelination. This end result is central pontine myelinolysis (CPM).

CPM features are variable, however the classical presentation is spastic quadriparesis, pseudobulbar palsy, and emotional lability (pseudobulbar affect).

Question 62 of 249

A 48-year-old female is admitted with an overdose of gliclazide. She states that she has taken ten 40mg tablets approximately three hours ago after getting into an argument with her boyfriend. She has a past medical history of type 2 diabetes mellitus, psychotic depression, and self-harm. Her drug history includes gliclazide and venlafaxine.

On examination, she appears sweaty and clammy and her heart rate is 120 bpm. Her Glasgow coma score is 13 (M6, V4, E3). Plasma glucose is 3.5 mmol/l.

You administer 100 ml of 20% dextrose. Her blood glucose initially rises to 5.5 mmol/l, however it rapidly drops back to 3.2 mmol/l shortly after, despite a continuing infusion of 20% dextrose.

What treatment will you give?

A. Lucozade
B. Complex carbohydrate
C. Reassurance
D. Insulin
E. Octreotide

E. Octreotide

In sulphonylurea overdoses, if the patient remains hypoglycaemic despite infusion of sufficient glucose, consider administration of octeotride

An overdose of sulphonylureas such as gliclazide can lead to severe and prolonged hypoglycaemia. The elimination half-life of gliclazide is long and varies between 12 and 20 hours. Gliclazide is readily absorbed from the gastrointestinal tract with peak plasma concentration occurring within 4 to 6 hours.

Octreotide is a somatostatin analogue which lowers insulin levels and thus raised blood glucose.

The patient had a reduced consciousness level, and it would, therefore, be inappropriate to administer Lucozade or complex carbohydrate.

Insulin would be entirely inappropriate as this would worsen the hypoglycaemia.

Question 63 of 249

A 59-year-old man comes to the GP surgery due to facial changes over the last 6-months. He has a history of hypertension which remains uncontrolled despite compliance with medications, a low-salt diet, and regular exercise.

His observations are taken as follows: temperature is 36.7ºC, blood pressure 146/98mmHg, heart rate 90/min, and respiratory rate 14/min. On examination, facial features appear coarse and differ significantly from those on his driver's license photograph taken 3 years ago. His fingers are swollen, and his skin appears to have thickened.

His IGF-1 levels are raised, and an MRI shows a pituitary adenoma. The patient undergoes trans-sphenoidal surgery for the resection of his tumour.

On follow-up 3 months later, his serum IGF-1 levels are still raised. He still has the same symptoms as before. He is referred for a repeat MRI that shows no residual tumour.

Which of the following is the best next step in the management of this patient?

A. Refer for stereotactic radiotherapy
B. Repeat transsphenoidal surgery
C. Start octreotide
D. Repeat IGF-1 levels in 12 weeks
E. Repeat IGF-1 levels in six months

C. Start octreotide

Acromegaly: if patients are not suitable for trans-sphenoidal surgery, or have residual symptoms, then octreotide may be used

This patient's facial changes over time, coarse facial features on examination and elevated IGF-1 levels point towards a diagnosis of acromegaly. Since his MRI showed a pituitary adenoma, he was treated with transsphenoidal surgery. However, he remains symptomatic, and his IGF-1 levels have not reduced even after 3 months. Patients with residual symptoms need to repeat transsphenoidal surgery or start medical therapy with octreotide.

Since this patient has no residual tumour on his repeat MRI, he is not a candidate for repeat transsphenoidal surgery. Therefore, the most appropriate step for this patient is to start medical therapy with octreotide. Octreotide is a somatostatin analogue. It tends to decrease the size of the adenoma and its secretion of growth hormone.

Stereotactic radiotherapy is recommended for patients who fail medical therapy and is therefore not the best next step in the management of this patient.

Repeating IGF-1 levels again in 12 weeks is inappropriate as the IGF-1 level should have normalised at this follow-up 12 weeks after the transsphenoidal surgery. His repeat IGF-1 levels at 12 weeks are raised. He requires further medical or surgical management instead of follow-up in 12 weeks.

Patients with normal IGF-1 levels on a 12-week follow-up should have repeat IGF-1 levels at six months and then, annually.

Question 64 of 249

A 23-year-old female presented with acne and hirsutism worsening over the last 3 years. She attained menarche aged 10 and has irregular periods.

On examination, body mass index 29kg/m², heart rate 80/min, blood pressure 135/85 mmHg. Hirsutism and acanthosis nigricans are noticed along with mild clitoromegaly.

Bloods on 6th day after menstruation:

Estradiol	300 pmol/L (early follicular NR<300 pmol/L)
17OH-progesterone	20 nmol/L (NR<10 nmol/L)
Free Testosterone	3 nmol/L (NR<3 nmol/L)
LH	4 IU/L (NR 1-9 IU/L)
FSH	3 IU/L (NR 1-13 IU/L)
9am cortisol	150 nmol/L (NR 200-700 nmol/L)

What is the single most useful test?

A. CT adrenals
B. Karyotype
C. Pelvic USS
D. Short synacthen test
E. MRI pituitary

D. Short synacthen test

ACTH stimulation testing may be used to diagnose congenital adrenal hyperplasia

This patient has hyperandrogenism (acne, hirsutism, high 17OHprogesterone) indicative of non-classical congenital adrenal hyperplasia.

CAH is caused by:

1. 21 hydroxylase mutation (90% of CAH).

This mediates conversion of 17OH-progesterone to 11-deoxycortisol and progesterone to deoxycorticosterone. Loss of function results in reduced cortisol and aldosterone and subsequent elevation in ACTH. This causes overstimulation of the adrenal cortex (hyperplasia). The steroid precursors are forced down the sex hormone pathway leading to androgen excess (ambiguous genitalia, salt wasting, hypovolaemia and shock i.e classic type). Presentation of 21 hydroxylase deficiency can also be asymptomatic with androgen excess become a problem in late childhood i.e. non-classic (premature pubarche, accelerated bone age, acne, hirsutism, oligomenorrhoea and this mimics polycystic ovarian syndrome).

2. 11-beta hydroxylase mutation (5% of CAH).

Raised BP as 11-deoxycortisol has some aldosterone activity. Also raised androgens.

3. 17-alpha hydroxylase deficiency Raised aldosterone but low androgens. 4. 3-beta steroid dehydrogenase Low aldosterone and low androgens.

The short synacthen test is helpful when 17OH-progesterone is only modestly elevated (if 17OH-progesterone is very high then it is diagnostic). Measuring 17OH-progesterone at 0 and 60 mins after ACTH causes elevated responses in patients with CAH (>35 nmol/L).

Question 65 of 249

A 61-year-old man with type 2 diabetes mellitus (T2DM) attends a diabetic review. He has been treated with metformin 1g twice daily for the last six months. He has been compliant with the metformin and had no side effects. An HbA1c is performed.

HbA1c	61mmol/mol	(<48)

What is the next step in management?

A. Add second oral agent
B. Increase metformin to three times daily
C. No change required
D. Start insulin
E. Switch metformin to modified release

A. Add second oral agent

T2DM on metformin, if HbA1c has risen to 58 mmol/mol then one of the following should be offered depending on the individual clinical scenario:

DPP-4 inhibitor

pioglitazone

sulfonylurea

SGLT-2 inhibitor (if NICE criteria met)

As this man has an HbA1c > 58mmol/mol despite taking metformin therapy for several months, **adding a second oral agent** is required to improve glycaemic control.

The maximum daily metformin dose is 2g. **Increasing the metformin dose to three times daily** is therefore inappropriate.

This patient has been on metformin for several months so the HbA1c should be reflective of his glycaemic control. As per the guidance, further optimisation of therapy is required if the HbA1c is > 58, Therefore it is incorrect to suggest that **no change is required**.

Starting insulin is only indicated once at least two oral agents have not achieved glycaemic control. As the patient is only on one agent at present, it would be premature to start insulin at this stage.

Switching metformin to modified release may help patients to tolerate the drug but would not affect glycaemic control. This is therefore not sufficient to address the high HbA1c.

Question 66 of 249

A 23-year-old woman is brought into the emergency department by ambulance with her mum, who found her unconscious on the bathroom floor just half an hour ago. The mum had last seen her 4 hours previously and reports she was well at this time. She has a past medical history of depression and was last admitted 5 months ago with a paracetamol overdose. She has otherwise been well in herself and suffers no other medical conditions that mum is aware of. Her GCS on arrival is 8. She is found to be severely hypoglycaemic at 2.2mmol/L and is treated with IV dextrose.

You are the medical registrar on call and have been asked to assess her, she has now been on a dextrose infusion for 45 minutes with no minimal improvement. The examination is grossly normal besides a tachycardia of 110bpm and GCS of 10/15 (M4, E3, V3). She is maintaining her own airways. Up-to-date glucose is 2.9 mmol/L, and her C-peptide and insulin are both elevated. You ask that she be given 50mL of 50% glucose IV, but she remains hypoglycaemic despite this.

Which of the following should be considered as a treatment for this woman?

A. Activated charcoal
B. Cyproheptadine
C. Dialysis
D. Gliclazide
E. Octreotide

E. Octreotide

In sulphonylurea overdoses, if the patient remains hypoglycaemic despite infusion of sufficient glucose, consider administration of octeotride

At this degree of hypoglycaemia, insulin levels should be undetectable. An insulin overdose would only cause raised insulin level; C-peptide is indicative of endogenous insulin release. Therefore the raised levels of both C-peptide and insulin suggest either an insulinoma or sulfonylurea use (triggering endogenous insulin release). Considering her past medical history, sulfonylurea overdose should be considered the most likely cause. Though it is unlikely that she is taking a sulfonylurea, it is quite possible that a family member is taking this medication, and that she might have easy access to it.

An overdose of sulphonylureas such as gliclazide can lead to severe and prolonged hypoglycaemia. The elimination half-life of gliclazide is long and varies between 12 and 20 hours.

Octreotide should be considered if hypoglycaemic despite the infusion of sufficient glucose. Octreotide is a somatostatin analogue and inhibits the secretion of many hormones, including insulin. It would be worth considering in this woman, due to her hypoglycaemia despite dextrose. IM glucagon may also be considered, particularly if IV access has proven difficult but its effects are dependent on available glycogen stores. Once euglycaemia is achieved, the patient ought to regain consciousness. Failure to do so ought to make one consider cerebral oedema.

It is too late to be considering activated charcoal as this is only considered 1-hour post-ingestion, and it is likely she ingested the sulfonylurea hours ago. Cyproheptadine is used in serotonin syndrome and carcinoid tumours. Dialysis is not indicated in sulphonylurea poisoning. Gliclazide would make things very much worse for this woman.

Question 67 of 249

A 29-year-old woman is referred to the Endocrinology clinic as she has just found out she is pregnant. She was diagnosed with hypothyroidism three years ago and is currently stable on a dose of levothyroxine 75mcg od. She has also been taking folic acid 400mcg od for the past 6 months. Her last bloods taken 6 months ago show the following:

TSH	1.4 mU/l

You request a repeat TSH and free T4 measurement.

What is the most appropriate next step?

A. Decrease levothyroxine to 50mcg od
B. Keep levothyroxine at 75mcg od
C. Increase levothyroxine to 100mcg od

D. Keep levothyroxine at 75mcg od + increase folic acid to 5mg od
E. Stop levothyroxine until TSH known

C. Increase levothyroxine to 100mcg od

Pregnant with hypothyroidism → immediately increase levothyroxine and monitor TSH closely

The correct answer is to **increase levothyroxine to 100mcg od**. In pregnant women with hypothyroidism, it is essential to maintain adequate thyroid hormone levels for the developing fetus. The requirement for levothyroxine often increases during pregnancy due to increased thyroxine-binding globulin (TBG) and placental degradation of maternal T4. According to UK guidelines, pregnant women with preexisting hypothyroidism should have their levothyroxine dose increased by approximately 30% as soon as pregnancy is confirmed, even before obtaining repeat TSH and free T4 measurements. This ensures that both mother and baby have sufficient thyroid hormone levels during the critical early stages of fetal development.

The option to **decrease levothyroxine to 50mcg od** would not be appropriate, as reducing the dose could lead to inadequate thyroid hormone levels in the mother and potentially harm fetal development.

Keeping the **levothyroxine at 75mcg od** might seem reasonable if her TSH was well-controlled prior to pregnancy; however, this does not account for the increased requirements during pregnancy. It would be safer and more appropriate to increase the dose proactively.

As for **keeping levothyroxine at 75mcg od + increasing folic acid to 5mg od**, while it is important for pregnant women to take folic acid supplements, increasing the dose of folic acid alone without adjusting the levothyroxine dosage does not address her increased thyroid hormone needs during pregnancy.

Finally, choosing to **stop levothyroxine until TSH is known** would put both mother and fetus at risk of complications related to untreated or inadequately treated hypothyroidism. It is crucial that patients with preexisting hypothyroidism continue their levothyroxine therapy during pregnancy, with appropriate dose adjustments as needed.

Question 68 of 249

A 47-year-old woman is admitted to the surgical ward with severe loin to groin abdominal pain. A CT-KUB reveals a right-sided renal calculus. When you clerk her in she admits to you that she has not felt herself for the past few weeks with polyuria, polydipsia, constipation and altered mood.

Blood tests show:

Estimated glomerular filtration rate	>60 ml/min
Adjusted calcium	3.1 mmol/l (2.1-2.6 mmol/l)
Phosphate	0.6 mmol/l (0.8-1.4 mol/l)
Parathyroid hormone	5.1 pmol/l (1.2-5.8 pmol/l)

Which of the following is the most likely cause for her symptoms?

A. Primary hyperparathyroidism
B. Secondary hyperparathyroidism
C. Sarcoidosis
D. Tertiary hyperparathyroidism
E. Type 1 renal tubular acidosis

A. Primary hyperparathyroidism

The PTH level in primary hyperparathyroidism may be normal

The most likely diagnosis here is primary hyperparathyroidism caused by parathyroid adenoma or hyperplasia. The classical biochemical findings are a high serum calcium and low phosphate. The parathyroid hormone level is either high or inappropriately normal.

Secondary hyperparathyroidism is caused by chronic hypocalcaemia (e.g. chronic kidney disease). Serum calcium is low or normal whilst parathyroid hormone levels are high.

Tertiary hyperparathyroidism develops from secondary hyperparathyroidism and results in autonomous parathyroid production. It is usually seen patients with end-stage renal disease.

Sarcoidosis and type 1 renal tubular acidosis are rare causes of hypercalcaemia.

Question 69 of 249

A 67-year-old man with a history of ischaemic heart disease and type 2 diabetes mellitus is noted to have non-visible haematuria during an annual review. He is currently feeling well and is asymptomatic. The urine dipstick showed blood ++, with no protein and no leucocytes. This result is repeated one week later.

His current medications include aspirin, bisoprolol, atorvastatin, ramipril, metformin and pioglitazone.

Which one of the following drugs should be stopped whilst awaiting further investigations?

A. Aspirin
B. Ramipril
C. Atorvastatin
D. Metformin
E. Pioglitazone

E. Pioglitazone

Thiazolidinediones are associated with an increased risk of bladder cancer

The correct answer is **Pioglitazone**. Pioglitazone, an oral antidiabetic agent used in the management of type 2 diabetes mellitus, has been associated with an increased risk of bladder cancer. In the presence of non-visible haematuria (blood in urine), this medication should be discontinued until further investigations are completed.

Aspirin is not typically associated with haematuria and its use as antiplatelet therapy in a patient with ischemic heart disease should not be interrupted without a compelling reason.

Ramipril, an angiotensin-converting enzyme (ACE) inhibitor, is used for hypertension and cardiac conditions. While it can occasionally cause renal impairment leading to proteinuria, it does not typically cause haematuria.

Atorvastatin, a statin used for hyperlipidaemia and cardiovascular risk reduction, may have some side effects such as myopathy or liver dysfunction but it is not commonly associated with haematuria.

Finally, **Metformin**, another oral antidiabetic agent, can lead to lactic acidosis in cases of renal insufficiency but it does not usually cause haematuria. Therefore, there would be no need to stop metformin based on the given clinical scenario.

Question 70 of 249

A 70-year-old man with a history of smoking 15 cigarettes/day presents with drowsiness, weight loss and a persistent cough. His investigations show:

Na +	115 mmol/l	135-145 mmol/l
K +	5.1 mmol/l	3.5 - 5.0 mmol/l
Urea	3 mmol/l	2.0-7 mmol/l
Creatinine	74 µmol/l	55-120 µmol/l
Plasma osmolality	270 mOsm/kg	285-295 mOsm/kg
Urine osmolality	1210	500 - 800 mOsm/kg

What is the most likely diagnosis?

A. Small cell lung cancer
B. Hypothyroidism
C. Encephalitis
D. Congestive cardiac failure
E. Squamous cell carcinoma

A. Small cell lung cancer

A common endocrine complication of small cell lung cancer is SIADH

Hyponatraemia, reduced plasma osmolality and increased urine osmolality are suggestive of syndrome of inappropriate ADH secretion (SIADH).

The increase in ADH causes more aquaporin utilisation in the collecting duct system of the kidney. This causes more water to be retained,

diluting the electrolytes in the blood and making the electrolytes in the urine more concentrated.

Small cell lung cancer is a common cause of SIADH and is the most likely diagnosis in this man with an extensive smoking history, cough and weight loss.

Question 71 of 249

A 21-year-old woman presents to the Emergency department following a collapse at the local supermarket. She tells you that she always feels weak and washed out, and hardly ever has any energy. She takes no regular medication and virtually never sees the doctor. On examination her blood pressure is 100/70 mmHg, pulse is 80 beats per minute and regular. She is slim with a body mass index of 21 kg/m², no abnormal physical signs are noted.

Investigations

Na +	140 mmol/l
K +	3.1 mmol/l
HCO3 -	32 mmol/l
Urea	5.9 mmol/l
Creatinine	85 µmol/l

Which of the following is the most likely diagnosis?

A. Conn's syndrome
B. Cushing's syndrome
C. Gitelman's syndrome
D. Liddle's syndrome
E. Renal tubular acidosis type 1

C. Gitelman's syndrome

Gitelman's syndrome: normotension, hypokalaemia + hypocalciuria

This patient has hypokalaemic metabolic alkalosis with normal / low blood pressure. This fits best with a diagnosis of Gitelman's syndrome, Bartter's syndrome, or diuretic abuse. Bartter's syndrome often presents in infancy as failure to thrive, and isn't listed as a distractor. Diuretic

abuse presents with a similar clinical picture although it too isn't listed here as an option. Gitelman's is caused by a mutation in the gene coding for the thiazide-sensitive sodium-chloride co-transporter, and results in a clinical picture similar to that expected with thiazide diuretic use.

Conn's syndrome is associated with hypertension, as is Liddle's syndrome, effectively ruling them out as diagnoses here. Renal tubular acidosis type 1 is associated with hypokalaemia and metabolic acidosis, not with a bicarbonate of 32.

Question 72 of 249

You are asked to review a 42- year- old alcoholic who has been admitted to the medical ward following 48hrs of vomiting, generalised muscle weakness and palpitations. Despite two calcium infusions, the most recent measured calcium is still 1.89 mmol/l. On examination his blood pressure is 95/60 mmHg, pulse is 95 beats per minute and regular. You note intermittent runs of SVT on his cardiac monitor.

Other urea and electrolytes are shown below:

Na 132 +	mmol/l
K 3.7 +	mmol/l
Urea 5.4	mmol/l
Creatinine 82	µmol/l
Glucose 5.2	mmol/l

Which of the following is the most appropriate next step?

A. IV calcium
B. IV magnesium
C. IV potassium
D. IV phosphate
E. IV glucose

B. IV magnesium

Magnesium deficiency causes hypocalcaemia

Long term alcoholism as well as leading to falls in serum calcium, can also lead to significant falls in magnesium, which can account both for the persistently decreased calcium despite replacement, and for the runs of SVT seen here. The most appropriate next step is magnesium replacement, which is likely to facilitate both an improvement in calcium and resolution of SVT.

Further IV calcium will be ineffective without first replacing magnesium, and potassium is within the normal range, as is glucose. Phosphate replacement may be required in the treatment of alcoholism, but this is usually in the context of refeeding syndrome.

Question 73 of 249

A 32-year-old man is reviewed in the ischaemic heart disease clinic having suffered an inferior myocardial infarction. He has been diagnosed with heterozygous familial hypercholesterolaemia and started on 80mg per day of atorvastatin. His LDL cholesterol is still 3.5 mmol/l.

Which of the following is the most appropriate next intervention?

A. Add cholestyramine
B. Add evolocumab
C. Add fenofibrate
D. Add nicotinic acid
E. Change atorvastatin to rosuvastatin

B. Add evolocumab

Although the 80mg of atorvastatin has clearly brought the LDL cholesterol much closer to target, but LDL of 3.5 mmol/l is still suboptimal given the history of an inferior myocardial infarction. Evolocumab, a PCSK9 inhibitor which interferes with degradation of the LDL receptor is the most appropriate intervention and can reduce LDL by a further 50%. Given his relatively young age and high lifetime risk of cardiovascular disease, he is exactly the type of patient likely to benefit from PCSK9 inhibition.

Cholestyramine was formerly used in the treatment of hypercholesterolaemia, it is a cholesterol binding resin, but is less effective in lowering cholesterol than a statin. Both fenofibrate and nicotinic acid have most effect in lowering triglycerides, and changing atorvastatin to rosuvastatin is likely to have limited benefit in further lowering LDL.

Question 74 of 249

An 18-year-old man comes to the endocrinology clinic for review. He has been transferred from the paediatric clinic with a diagnosis of congenital hypoparathyroidism and is treated with vitamin D and calcium supplementation. He has had one episode of symptomatic renal stones over the past 3 years and his creatinine is elevated at 125 micromol/l.

Which of the following is the most appropriate target with respect to serum calcium?

A. 1.85 mmol/l
B. 2.10 mmol/l
C. 2.25 mmol/l
D. 2.60 mmol/l
E. 2.85 mmol/l

B. 2.10 mmol/l

This 18-year-old man is at risk of symptomatic hypocalcaemia because of congenital hypoparathyroidism, although elevating his calcium too much with vitamin D supplementation runs the risk of symptomatic renal stones. As such guidelines recommend aiming towards a calcium just below the lower end of the normal range and 2.10 is an appropriate target for serum calcium.

1.85 mmol/l is considered too low and puts the patient at risk of muscle weakness, paresthesias, tetany and cardiac arrhythmia. Maintenance of calcium either at the upper end of the normal range, (2.60 mmol/l), or 2.85 mmol/l (above the normal range), is associated with symptomatic renal stones and progressive deterioration in renal function.

Question 75 of 249

An 18-year-old woman is brought into the hospital at midnight with generalised weakness and mild difficulty breathing.

On examination, there is weakness in the upper and lower limbs with proximal predominance. Reflexes in the upper limbs are reduced. Muscle tone is normal. Chest sounds clear but expansion is reduced bilaterally. GCS is 15/15.

The patient describes similar episodes in the past that have lasted for only minutes, the episodes decreased in frequency after commencing a ketogenic diet. They had been attributed to hemiplegic migraines, though she had had bilateral symptoms.

From the following options, what investigation will be most helpful in confirming the likely diagnosis?

A. CT head
B. Electromyography
C. Lumbar puncture
D. MRI brain and spinal cord
E. Urea & electrolytes

E. Urea & electrolytes

Episodes of weakness, hypokalaemia, symptoms triggered by carbohydrate meals → hypokalaemic periodic paralysis

The most likely diagnosis is a periodic paralysis of which hypokalaemic periodic paralysis is the most common. Attacks can be precipitated by high-carbohydrate meals which explains why the number of episodes reduced for this patient when she commenced on a low-carbohydrate ketogenic diet.

Differentials might include myasthenia gravis (though this is commonly associated with fatigable weakness rather than 'attacks'), metabolic myopathies (which involve weakness typically worsened by exercise), or in a first attack Guillain-Barre, transverse myelitis, of infective paralysis (tick paralysis, botulism) may be considered.

Urea and electrolytes is the correct answer. A low potassium level would make the diagnosis here clearer. Average potassium levels in hypokalaemic periodic paralysis are around 2.4mmol/L. If hypokalaemia is significantly low, less than 2.0mmol/L for example, you should think about secondary causes of hypokalaemia such as losses from the gut, and renal losses.

MRI brain and spinal cord is incorrect. This would be a reasonable investigation to investigate the possibility of multiple sclerosis (MS). Here, the pattern of weakness does not fit a particular cerebral or spinal territory which you would expect with MS.

Electromyography is incorrect. Although this would be able to demonstrate myopathy during an acute attack, it would not assist us in determining the specific underlying diagnosis as there are many causes of myopathy.

Lumbar puncture is incorrect. A lumbar puncture would be helpful in looking for inflammatory or infective causes of neurological symptoms, but would not be able to confirm a diagnosis of hypokalaemic periodic paralysis. Given the episodic nature of the attacks, transverse myelitis or infection, for example, would not explain the symptoms here.

CT head is incorrect. This patient's symptoms are recurrent and do not correspond to a particular cerebral territory. A CT head would be unhelpful in confirming the likely diagnosis.

Question 76 of 249

You are called to review a 43-year-old woman who has received 48 hours of treatment for diabetic ketoacidosis. She has had diarrhoea and abdominal discomfort for the past 12 hours is now become increasingly confused with visual hallucinations and aggressive behaviour.

She has a history of type 1 diabetes mellitus and Grave's disease which has been in remission and not requiring treatment.

Observations: SpO2 97% room air, respiratory rate 18 breaths per minute, heart rate 145 beats per minute, blood pressure 170/110 mmHg, temperature 40.2ºC.

On examination, she has generalised abdominal pain, mild pitting oedema, and an irregularly irregular pulse.

Hb	135 g/L	Male: (135-180)Female: (115 - 160)
Platelets	422 * 10 /L 9	(150 - 400)
WBC	8.5 * 10 /L 9	(4.0 - 11.0)
Na +	135 mmol/L	(135 - 145)
K +	4.0 mmol/L	(3.5 - 5.0)
Urea	7.8 mmol/L	(2.0 - 7.0)
Creatinine	65 µmol/L	(55 - 120)
CRP	24 mg/L	(< 5)
TSH	0.4 mIU/L	(0.5-5)
FT4	32 pmol/L(12-22)	

What is the most appropriate acute treatment for the likely underlying cause of this patient's current presentation?

A. 300mg aspirin
B. Carbimazole
C. IV antibiotics
D. Propylthiouracil
E. Thyroidectomy

D. Propylthiouracil

In thyroid storm, treat acutely with propylthiouracil rather than carbimazole or surgery

The stem describes a classic case of thyroid storm. It is most commonly seen in patients with a background of Grave's disease but can occur in other scenarios too. There is usually a precipitating insult such as diabetic ketoacidosis or a heart attack.

The likelihood of a thyroid storm can be calculated using the Burch and Wartofksy thyroid storm diagnostic criteria. This attributes points to the presence of pyrexia, tachycardia, signs of heart failure, confusion, coma, seizures, gastrointestinal disturbance etc.

In this case, the patient scores 90. A score of >45 indicates a likely thyroid storm. The patient also has a suppressed TSH level with raised T4, this cements the diagnosis.

In an ideal situation treatment for presumed thyroid storm should not be delayed whilst awaiting blood results as this condition has significant mortality (>10%). Blood tests may be in keeping with hyperthyroidism, but there is no specific cut-off to diagnose thyroid storm, the diagnosis is made on clinical findings.

Propylthiouracil is the correct answer. Patients should be assessed with an ABCDE approach and stabilised. IV fluids and cooling are important aspects of treatment. Tachyarrhythmias should also be managed according to local guidelines. The most appropriate treatment for the underlying thyroid storm is propylthiouracil. This inhibits thyroid hormone synthesis and also inhibits the conversion of T4 to T3.

Carbimazole is incorrect. Although carbimazole can be used, propylthiouracil is the traditionally preferred treatment as it has a faster onset of action and also inhibits the conversion of fT4 to T3.

Thyroidectomy is incorrect. This can be used as a definitive treatment for Grave's disease and can also be used in the acute setting if a patient has had a previous poor reaction to thionamides such as propylthiouracil. Usually, the first-line treatment would be stabilisation with beta-blockers, supportive treatment and oral thionamides.

IV antibiotics is incorrect. In this case, the patient is pyrexial and has new-onset atrial fibrillation with a rapid ventricular response. Diarrhoea and abdominal pain could indicate an intra-abdominal infection. It would not be unusual for the patient to receive IV antibiotics in this scenario to cover for potential infection, but given the blood results here - normal white cell count, normal CRP, and deranged thyroid function - we can be quite sure of a diagnosis of thyroid storm which wouldn't respond to IV antibiotics. The concomitant atrial fibrillation with signs of heart failure (pedal oedema), extreme pyrexia, and delirium, are better explained by thyroid storm.

300mg aspirin is incorrect. This would be the treatment of stroke. There is an altered conscious level with behavioural changes and pyrexia, both of which can be seen in acute stroke. There are, however, no focal neurological findings described and a stroke would not explain the change in bowel habit and abdominal pain.

Question 77 of 249

A 26-year-old woman presents to the endocrine clinic. She has a background of Graves' disease, previously well-controlled on carbimazole alone. At the onset, she was in atrial fibrillation and she remains on apixaban for this.

In the past month, she has developed a new tremor, palpitations and weight loss. Observations are within normal limits. Her most recent blood results confirm a return to a hyperthyroid state.

A pregnancy test in the clinic is negative and she has no plans to become pregnant. Examination of the eyes is unremarkable but you find a mild goitre on examination.

What is the best course of action?

A. Add Lugol's iodine
B. Add high dose oral prednisolone
C. Organise thyroidectomy
D. Start radioiodine treatment
E. Switch to propylthiouracil

D. Start radioiodine treatment

Radioiodine therapy is the treatment of choice for patients with a relapse of Graves disease in the absence of contraindications, such as pregnancy and active severe Graves ophthalmopathy

The correct answer is **start radioiodine treatment**. Radioiodine is the treatment of choice for relapse of Graves' disease. The negative pregnancy test and lack of significant ophthalmopathy rule out the most important contraindications.

Add Lugol's iodine is incorrect. Iodine-containing solutions can block the release of T3 and T4. However, the main place is in the treatment of thyroid storms, generally in combination with other agents such as beta blockers and thionamides.

Add high dose oral prednisolone is incorrect. Steroids are mainly used in severe cases such as those with thyroid storm, although the evidence is limited. This patient is symptomatic but with normal observations and no evidence of thyroid storm.

Organise thyroidectomy is incorrect. Thyroidectomy is a very effective treatment option and may be used to treat relapse in some patients but is generally unpopular. However, it is favoured in those with large goitre or contraindications to radioiodine. These include pregnancy or severe eye disease. In the absence of such contraindications, radioiodine would be favoured here. Her anticoagulation would also increase the complexity and risk of surgery here.

Switch to propylthiouracil is incorrect. This patient has relapsed despite appropriate first-line therapy with one thionamide - switching to another drug of the same class is unlikely to offer any benefits.

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A 37-year-old man is being investigated for a chronic metabolic acidosis. He has an anion gap of 9 mEq/L and your consultant suspects he may have a renal source of his acidaemia.

Which of the following can lead to renal tubular acidosis (type 2)?

A. Sjogren's syndrome
B. Wilson's disease
C. Haemochromatosis
D. NSAIDs
E. Nephrocalcinosis

B. Wilson's disease

Type 2 renal tubular acidosis may be caused by Fanconi syndrome

The answer is Wilson's disease.

Renal tubular acidosis (RTA) is a renal cause of a metabolic acidosis. It has 3 main categories:

- Type 1: Distal (failure of proton secretion)
- Type 2: Proximal (failure of bicarbonate reabsorption) Type 4: Aldosterone deficiency/insensitivity
- Type 3 is a combination of 1 and 2.

Types 1 & 2 are characterised by hypokalaemia and type 4 is characterised by hyperkalaemia. Sjogren's syndrome is one of the classic causes of Type 1 RTA, as well as nephrocalcinosis being both a common cause and consequence of long standing RTA.

NSAIDs lead to aldosterone resistance, causing a Type 4 RTA.

Wilson's disease causes a form of Type 2 RTA called Fanconi syndrome. It can also be caused by other toxic heavy metals (lead & mercury) as well as expired tetracyclines. It leads to proximal loss of protons, phosphate, amino acids and glucose.

Haemochromatosis is a distractor.

Question 79 of 249

A 71-year-old gentleman presents to clinic for review. He has recently been diagnosed with type two diabetes following screening by his GP. His HbA1c has not responded to dietary changes that he was advised on. He has a past medical history of bladder cancer for which he had chemotherapy, hypertension, macular degeneration, eczema and chronic kidney disease. His baseline eGFR is 28ml/min/1.73m2.

Blood tests:

Na 139 mmol/l

K 4.4 mmol/l

Urea 6.2 mmol/l

Creatinine 214 μmol/l

What is the most appropriate medication to start?

A. Metformin
B. Sulfonylurea
C. Pioglitazone
D. Insulin
E. GLP-1 mimetic

B. Sulfonylurea

The correct answer is a sulfonylurea. This is a patient with new type two diabetes and initial treatment would normally be metformin, but since the eGFR is <30ml/minute/1.73m2 metformin is contraindicated. The next step would be either a a dipeptidyl peptidase4 inhibitor, pioglitazone or a sulfonylurea. However, pioglitazone should not be offered to a patient with a history of bladder cancer. Therefore that leaves sulfonylurea as the correct answer. Insulin and GLP-1 mimetic could be used further down in the treatment algorithm.

Question 80 of 249

A 59-year-old man presents to the medical clinic. He has a past history of type 2 diabetes mellitus, gout, obesity and cholecystectomy. He takes metformin and allopurinol. He has no allergies as far as he is aware. His total cholesterol is 4.8mmol/mol. He is concerned that his brother recently had a heart attack and was wondering if there is any medication that he should take to further reduce his risk of heart attacks. He smokes roughly five cigarettes per day, of which he has been trying to reduce. His blood pressure is 132/71mmHg.

What is the most appropriate action?

A. Start atorvastatin 20mg ON
B. Start atorvastatin 80mg ON
C. Start aspirin 75mg OD
D. Start clopidogrel 75mg OD
E. Start ezetimibe 10mg OD

A. Start atorvastatin 20mg ON

Patients with T2DM should not be routinely started on statins; their QRISK2 score should be calculated

This is a patient with high risk in terms of cardiovascular disease due to the fact that he is male, has obesity, is a current smoker and has a strong family history of heart disease. Therefore it is very important to manage his risk factors. In patients with type 2 diabetes, risk should be assessed using the QRISK2 method. This formula uses cardiovascular risk factors and estimates risk of cardiovascular ischaemia within the next 10 years. If you were to assess this patient, his risk would be far greater than 25%. NICE guidelines advises that if the risk is greater than 10% then Atorvastatin 20mg at night should be started, and increased if the cholesterol does not fall. Starting 80mg of Atorvastatin would be appropriate in secondary prevention, such as following a myocardial infarction. NICE does not advise starting aspirin or clopidogrel as primary prevention unless there is a high risk for an ischaemic event, as determined by QRISK scoring, and the patient is hypertensive or being treated for hypertension. This is because the risk of gastrointestinal bleeding is thought to outweigh the benefit of reduction in cardiovascular events in such patients.

Question 81 of 249

A 22-year-old gentleman presents to endocrinology clinic. He was referred by his GP as he was complaining of low energy and a morning testosterone was low. He immigrated from Albania one year ago and has been noticed to be always tired and lacking energy. He has also found to have low sexual desire after starting a new relationship in the UK. On examination he is tall and slim, with slight gynaecomastia.

What is the most appropriate investigation after confirming the low morning testosterone?

A. Prolactin
B. LH and FSH
C. MRI pituitary
D. Morning cortisol
E. Synacthen test

B. LH and FSH

A patient with low testosterone should have LH and FSH tested as first line investigation to separate primary and secondary hypogonadism

This is a patient with biochemical and clinical evidence of low testosterone. He is symptomatic with low energy and low sexual desire, and most likely would have erectile problems if questioned. The history of immigration is important as it raises the possibility of missed childhood diagnoses, in this case implying a missed chromosomal disorder. The most appropriate investigation to differentiate primary from secondary hypogonadism is LH and FSH. If LH and FSH are elevated this would suggest a primary hypogonadism such as Klinefelter's syndrome, as is suggested by the description of this patient, but if it is not then a secondary cause should be found. If that were the case, then MRI of pituitary can be necessary, as well as morning cortisol and prolactin. Synacthen testing should be done if morning cortisol is low to confirm adrenal insufficiency.

Question 82 of 249

A 42-year-old man presents to endocrinology clinic. He has been seeing his GP due to developing gynaecomastia. At first, this was thought to be due to alcohol excess and possible liver involvement, but after stopping alcohol completely and normal liver function tests this diagnosis was excluded. He has no previous medical history apart from having had a tibial fracture a year ago, and having been diagnosed with migraines two months ago. He takes paracetamol for the migraines but finds this to be not very helpful, especially with the headaches at night. On more direct questioning he has also developed problems maintaining an erection. On examination he does have gynaecomastia, but apart from this he appears normal. On repeating blood tests in the clinic his liver function tests are normal, but morning serum testosterone is low, with normal FSH and LH.

What further investigation is likely to be diagnostic?

A. Liver biopsy
B. Transferrin saturations
C. Serum prolactin
D. CT brain
E. Thyroid function tests

C. Serum prolactin

Hypogonadism secondary to prolactinaemia would be associated with low testosterone and low or normal FSH and LH

This patient has symptoms of hypogonadism with erectile dysfunction and gynaecomastia with a suggested evidence of a headache suspicious for an intracranial lesion, especially with a new diagnosis of headaches at night. The most likely explanation is a prolactinoma, which can be detected by very high prolactin levels. After this, an MRI of the pituitary would be appropriate. A CT brain is unlikely to be able to demonstrate any lesions. A liver biopsy could be considered if there was liver failure and it was unexplained. Transferrin saturation could be considered if haemochromatosis was considered.

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A 62-year-old man presents to the emergency department with vomiting and abdominal pain. He has been becoming worse over four days since eating a barbecued piece of chicken he suspects was undercooked. Since then he has had diarrhoea and vomiting and has recently been noticing abdominal pain. His wife brought him to the emergency department as she felt he was starting to become drowsy. He has a past medical history of type 2 diabetes mellitus, osteoarthritis, steatohepatitis and stable angina. He normally takes metformin, dapagliflozin, aspirin, omeprazole, paracetamol and bisoprolol. Investigations reveal that he has a serum glucose of 12.1 mmol/L, a pH of 6.9 and urine testing reveal +++ of ketones. He is diagnosed with euglycaemic diabetic ketoacidosis.

What factor has most likely contributed to the development of diabetic ketoacidosis?

A. Drug history of dapagliflozin
B. Drug history of metformin
C. Drug history of bisoprolol
D. Medical history of steatohepatitis
E. Medical history of angina

A. Drug history of dapagliflozin

SGLT2 inhibitors can cause normoglycaemic ketosis in type 2 diabetes

The most likely explanation is the drug history of dapagliflozin. Dapagliflozin is a SGLT2 inhibitor which reduces glucose reabsorption in the urine and increases urinary glucose excretion. This patient has developed one of the side effects of dapagliflozin; DKA. DKA secondary to SGLT2 dapagliflozin is more likely to be euglycaemic than DKA in general. Metformin is associated with metabolic acidosis, especially in the context of reduced renal function or alcohol, and bisoprolol can cause hypoglycaemia. Steatohepatitis can eventually result in hepatic failure causing impaired gluconeogenesis, but this is more likely to result in hypoglycaemia rather than DKA. Angina is unlikely to be relevant.

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A 73-year-old woman presents to the general medical clinic. She has been investigated for hyponatraemia and has come for discussion of her results. She was incidentally found to have low sodium after having blood tests during a community-acquired chest infection two weeks ago. The chest infection has entirely resolved. She was found to have radiological evidence of small cell lung cancer on CT scan of her chest during investigations for her chest infection as well. She declined further biopsy or any investigation for this finding. Clinically she is euvolaemic. She has had serum sodium and osmolality measured. She is suspected of having SIADH.

Na +	122 mmol/l
K +	4.1 mmol/l
Urea	4.8 mmol/l
Creatinine	69 µmol/l
Serum osmolality	240 mmol/kg

What further investigation would be necessary to diagnose SIADH?

A. Random serum cortisol
B. Echocardiogram
C. Lung biopsy
D. Urinary electrolytes and osmolality
E. Water deprivation test

D. Urinary electrolytes and osmolality

SIADH criteria includes Na < 135, serum osmolality <271 and urinary osmolality >100 in a euvolaemic patient

This patient has established hyponatraemia with low serum osmolality. She needs to have elevated urinary osmolality to confirm the diagnosis of SIADH in the context of hyponatraemia and no other cause such as hypervolaemia. A morning cortisol would be helpful in diagnosing Addison's disease, and if suspected would be a useful investigation, but a random serum cortisol would not be very helpful. An echocardiogram could exclude heart failure in the context of fluid overload as a cause of hyponatraemia, but in the absence of peripheral oedema, a raised JVP and pulmonary oedema this is unlikely to be necessary. Water deprivation test is done to confirm diabetes insipidus.

Question 85 of 249

A 52-year-old patient is referred to the endocrinology clinic due to excessive sweating, which occurs over several months, and was concerned when the GP was unable to reassure him. On further questioning he has persistent sweating which is out of keeping with his activities, and sometimes develops headaches. He has found this to be a worsening problem over the last months. He has found that he has low energy as well. On examination he has a large face and tongue. He has a past medical history of hypertension and type 2 diabetes.

What is the most appropriate first-line investigation

A. Serum growth hormone
B. Serum IGF-1
C. Visual field testing
D. Oral glucose tolerance test measuring serum glucose
E. Pituitary MRI

B. Serum IGF-1

Serum IGF-1 levels are now the first-line test for acromegaly

The most appropriate first-line investigation for this patient is **serum IGF-1**. The patient's symptoms and physical examination findings are suggestive of acromegaly, which is caused by excessive secretion of growth hormone (GH) from the pituitary gland. Acromegaly can lead to hypertension, type 2 diabetes, and other complications. Serum IGF-1 (insulin-like growth factor 1) levels are a reliable indicator of GH activity and are more stable than serum GH levels throughout the day. An elevated serum IGF-1 level would support the diagnosis of acromegaly.

Serum growth hormone measurement is not the best first-line investigation because GH secretion is pulsatile, with peaks and troughs throughout the day. This can lead to false-negative results if the blood sample is taken during a period when GH levels are low. Serum IGF-1 levels are less variable and provide a more accurate assessment of overall GH activity.

Visual field testing could be useful in assessing potential compression of the optic chiasm by a pituitary tumour causing acromegaly; however, it is not an appropriate first-line diagnostic test for confirming the presence of acromegaly itself. It would be more relevant once biochemical evidence has been obtained to support the diagnosis.

An **oral glucose tolerance test measuring serum glucose** may be helpful in evaluating a patient's diabetes status but does not directly address the suspicion of acromegaly. In patients with confirmed acromegaly, an oral glucose tolerance test measuring serum growth hormone may be used to assess disease activity, as normal individuals should have suppressed GH levels after glucose ingestion, while those with acromegaly will not suppress their GH adequately.

A **pituitary MRI** could potentially identify a pituitary adenoma as the cause of acromegaly; however, it is not the best first-line investigation. Biochemical confirmation of GH excess should be obtained before performing imaging studies. Once a diagnosis of acromegaly has been established through elevated serum IGF-1 levels and/or an abnormal oral glucose tolerance test measuring serum growth hormone, a pituitary MRI would then be appropriate to identify the underlying cause and guide further management.

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A 19-year-old woman is brought to the endocrinology clinic. She has had episodes of feeling weak and needing a sweet drink to improve. This has been occurring frequently since she was rejected from applications to medical school. She has had problems with anxiety previously, but the parents have become concerned as they have once used the patient's sister's glucose monitoring equipment, as she has type 1 diabetes, and found the patient to have capillary glucose as low as 2mmol/l. They have become concerned that the patient may have an insulinoma after reading about this on the internet, and therefore insisted on an endocrinology referral from the GP.

Which investigation is best suited to exclude insulinoma?

A. CT abdomen
B. Capillary blood glucose monitoring over 48 hours
C. Post-prandial serum insulin and C-peptide
D. Supervised fasting with serum insulin and C-peptide
E. Synacthen test

D. Supervised fasting with serum insulin and C-peptide

Elevated C-peptide in fasting hypoglycaemia is suggestive of insulinoma

This patient has episodes of hypoglycaemia. There are several causes for hypoglycaemia, including insulinoma, liver failure, Addison's disease, alcohol and use of oral hypoglycaemic agents and insulin. Whilst insulinomas can cause episodes of hypoglycaemia, commonly associated with rapid weight gain and MEN-1, other explanations should also be considered. As this patient has a relative with type 1 diabetes she may have an opportunity to acquire insulin and use this herself. The most appropriate investigation to differentiate between the two is supervised fasting with monitoring of blood glucose, insulin and C-peptide can demonstrate or exclude insulinoma. If the patient were to have an insulinoma, then during fasting the blood glucose would become low, with elevated serum insulin and C-peptide, as C-peptide corresponds to endogenous rather than exogenous insulin. If the patient had hypoglycaemia during a fast with elevated insulin but low C-peptide the diagnosis would more likely be exogenous insulin use. A CT scan of the abdomen can be used if insulinoma is suspected following supervised fasting. Synacthen testing should be used to suspected Addison's disease.

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A 29-year-old woman is referred to the endocrinology clinic. She was concerned about the loss of her period with a negative pregnancy test. She underwent blood tests which demonstrated an undetectable TSH and a free T4 of 48ng/dl. She has a past medical history of hepatitis C but without chronic liver disease. Further tests demonstrate a negative thyroid-stimulating hormone receptor antibodies and a radionuclide thyroid uptake scan demonstrates diffuse thyroid uptake. She is diagnosed with Graves' disease. Due to the previous history of hepatitis C she opts for radioiodine treatment. She is warned regarding avoiding children during treatment, but wonders about her own conception following treatment.

What is the most appropriate advise?

A. Any future pregnancy will have increased risk of foetal malformation
B. She should avoid becoming pregnant for six months aftertreatment
C. She should avoid becoming pregnant for four months aftertreatment
D. Fertility may be adversely affected due to treatment
E. She should always avoid breastfeeding

B. She should avoid becoming pregnant for six months aftertreatment

Following radioiodine treatment, patients should avoid becoming pregnant for at least 6 months

This patient has a diagnosis of Graves' disease based on the biochemical evidence of hyperthyroidism and the diffuse radioiodine uptake scan. Radioiodine is an appropriate treatment choice, but in the absence of severe hepatic dysfunction carbimazole can also be used. During radioiodine treatment and for three weeks following the treatment, she should avoid contact with small children and pregnant women. She should avoid becoming pregnant for six months following treatment.

Following this, the radioiodine carries no risk for her foetus or children. A man on radioiodine treatment should avoid fathering a child for up to four months following treatment. It is important that Graves' disease is biochemically controlled during pregnancy as thyroid-stimulating hormone receptor antibodies can cross the placenta.

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A 34-year-old patient is brought by ambulance to the emergency department with a decreased level of consciousness. She has a past medical history of type 2 diabetes and emotionally unstable personality disorder. She takes metformin and gliclazide. She smokes ten cigarettes daily. She is unemployed.

Her observations are heart rate 111 beats per minute, blood pressure 101/55 mmHg, respiratory rate 21/minute, oxygen saturations 96% on room air and temperature 37ºC.

On examination, her Glasgow coma scale is 9/15 (E1 V3 M5). The cardiorespiratory examination is normal. Abdominal examination is normal. Neurological examination reveals normal tone, downgoing plantars. Her pupils are equal and reactive to light.

A bedside blood glucose level is 1.8 mmol/L.

Despite 2 x 100ml boluses of 10% dextrose and a subsequent dextrose infusion, the patient experiences recurrent hypoglycaemia.

What is the next most appropriate pharmacological treatment?

A. Activated charcoal
B. Enteral supplementation
C. Octreotide
D. 10% dextrose
E. 50% dextrose

C. Octreotide

In sulphonylurea overdoses, if the patient remains hypoglycaemic despite infusion of sufficient glucose, consider administration of octeotride

Octreotide is correct. This patient has recurrent hypoglycaemia in the context of known sulphonylurea use an emotionally unstable personality disorder. It is likely that she has taken a sulphonylurea overdose. Despite dextrose supplementation, she experiences recurrent hypoglycaemia. Octreotide blocks insulin secretion. The rationale being that dextrose precipitates further insulin release, causing a vicious cycle of insulin secretion. Octreotide prevents further insulin release in sulphonylurea overdose and is an option if the patient remains hypoglycaemic.

Activated charcoal is incorrect. This typically needs to be given within 1-2 hours of administration of sulphonylureas and we do not know the time of ingestion.

10% dextrose is incorrect. Despite sufficient dextrose administration, the patient remains hypoglycaemic and therefore an alternative treatment is needed.

Enteral supplementation is incorrect. The patient has a GCS of 9/15 and therefore enteral supplementation would be inappropriate.

50% dextrose is incorrect. The patient has already had a typically sufficient administration of dextrose. The patient remains hypoglycaemic, likely because of ongoing endogenous secretion of insulin in response to the glucose load and needs alternative treatment that blocks insulin secretion.

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A 32-year-old woman presents to her general practitioner with reports of fatigue. She is 30 weeks pregnant with her first child. She has no past medical history and is not on any medications.

Examination reveals a distended abdomen with a symphysis-fundal height that is consistent with gestational age. Foetal movements are noted.

Blood tests:

Hb	136 g/L	Male: (135-180)Female: (115 - 160)
Platelets	189 * 10 /L ^{9}	(150 - 400)
WBC	4.2 * 10 /L ^{9}	(4.0 - 11.0)
Na $^+$	138 mmol/L	(135 - 145)
K $^+$	4.2 mmol/L	(3.5 - 5.0)
Urea	5.2 mmol/L	(2.0 - 7.0)
Creatinine	88 µmol/L	(55 - 120)
CRP	4 mg/L	(< 5)
Bilirubin	12 µmol/L	(3 - 17)
ALP	145 u/L	(30 - 100)
ALT	32 u/L	(3 - 40)
Albumin	36 g/L	(35 - 50)

What is the most likely explanation for her symptoms?

A. Acute fatty liver of pregnancy
B. HELLP syndrome
C. Intrahepatic cholestasis of pregnancy
D. Normal pregnancy
E. Primary biliary cirrhosis

D. Normal pregnancy

Normal pregnancy can cause a raised ALP - it doesn't necessarily imply liver problems

Normal pregnancy is correct. This woman is 30 weeks pregnant and therefore fatigue is not an unexpected complaint. A raised ALP is common in late pregnancy and is usually of placental origin. In the absence of any other signs or symptoms suggesting underlying liver disease, the most likely explanation is that these findings are consistent with normal pregnancy.

HELLP syndrome is incorrect. This stands for haemolysis, elevated liver enzymes and low platelets. She does not have evidence of either haemolysis or low platelets. While fatigue is a possible symptom, typically other symptoms are also present including abdominal pain, nausea, vomiting and backache.

Intrahepatic cholestasis of pregnancy is incorrect. This is typically characterized by itch, which is absent in this case.

Primary biliary cirrhosis is incorrect. While this can cause fatigue and a raised ALP, the median age of diagnosis is 65 and it is relatively uncommon in young people. A placental cause of the raised ALP is far more likely.

Acute fatty liver of pregnancy is incorrect. This is typically characterized by nausea, vomiting and abdominal pain. The ALT level is typically in the 300-500 IU/L range rather than an isolated rise in ALP. The patient will appear unwell, jaundice is common and there may be disseminated intravascular coagulation.

Question 90 of 249

A 65-year-old man presents is referred to the urology clinic with three weeks of macroscopic haematuria. He has a past medical history of type 2 diabetes. He recently underwent treatment for a threadworm infection. His medications include linagliptin, metformin and pioglitazone. He does not smoke cigarettes or drink alcohol. He works as a dairy farmer. He is originally from Jamaica.

He undergoes a cystoscopy, which demonstrates an exophytic lesion in the bladder.

Given the likely diagnosis, what is the patient's most significant risk factor for the development of this condition?

A. Ethnicity
B. Threadworm infection
C. Metformin
D. Occupation
E. Pioglitazone

E. Pioglitazone

Thiazolidinediones are associated with an increased risk of bladder cancer

Pioglitazone is correct. Macroscopic haematuria and an exophytic lesion identified on cystoscopy suggest bladder cancer. Thiazolidinediones are a well-recognized risk factor for the development of bladder cancer.

Ethnicity is incorrect. Bladder cancer more commonly develops in those of white ethnicity rather than black. However, black patients are twice as likely to die from bladder cancer.

Occupation is incorrect. Typically, those employed in industries such as textile, rubber, leather, dye, paint, and print are at increased risk of bladder cancer due to chemical exposures. Dairy farming is not a typical risk factor.

Metformin is incorrect. The use of this medication is not associated with the development of bladder cancer.

Threadworm infection is incorrect. This parasite is not associated with bladder cancer. The parasitic infection associated with the development of bladder cancer is schistosomiasis. This is endemic to various tropical and subtropical regions. It is not typically found in Jamaica.

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A 43-year-old woman is referred to the endocrinology outpatient service after a finding of incidental hypercalcaemia on a routine blood test. She has no past medical history.

The examination is unremarkable. Blood tests:

Parathyroid hormone	7.9 pmol/L	(1.6 - 6.9)
Calcium	2.74 mmol/L	(2.20-2.6)
Vitamin D	75 nmol/L	(>50)
Urea	4.2 mmol/L	(2.0 - 7.0)
Creatinine	66 µmol/L	(55 - 120)

What is the most likely explanation for the findings from the list below?

A. Familial benign hypocalciuric hypercalcaemia
B. Multiple myeloma
C. Sarcoidosis
D. Secondary hyperparathyroidism
E. Tertiary hyperparathyroidism

A. Familial benign hypocalciuric hypercalcaemia

Patients with familial benign hypocalciuric hypercalcaemia may have a normal or raised PTH

Familial benign hypocalciuric hypercalcaemia is the correct answer. The patient has hypercalcaemia with a raised PTH level. Plausible differentials include primary hyperparathyroidism, tertiary hyperparathyroidism and familial benign hypocalciuric hypercalcaemia. Tertiary hyperparathyroidism develops in patients with longstanding secondary hyperparathyroidism (typically as a consequence of CKD, which is absent here). Primary hyperparathyroidism is not listed as an option. Therefore, familial benign hypocalciuric hypercalcaemia is the correct answer.

Multiple myeloma is incorrect. Classically, malignancies cause a suppressed PTH level if they are the cause of hypercalcemia. This is true of multiple myeloma.

Sarcoidosis is incorrect. Hypercalcemia in sarcoidosis is due to the uncontrolled production of 1,25-dihydroxy vitamin D3 by macrophages. This causes hypercalcemia and a suppressed PTH. unlike in some forms of hyperparathyroidism, the negative feedback mechanism whereby high calcium causes a low PTH is maintained.

Secondary hyperparathyroidism is incorrect. This form of hyperparathyroidism occurs as an appropriate reaction to low vitamin D or calcium. In this case, the vitamin is normal and the calcium is high.

Tertiary hyperparathyroidism is incorrect. Tertiary hyperparathyroidism develops in patients with longstanding secondary hyperparathyroidism (typically as a consequence of CKD, which is absent here). Therefore this is not likely.

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A 17-year-old woman is referred to the endocrinology outpatient clinic with primary amenorrhoea. She has no past medical history and does not take any medications.

On examination, there is no axillary or pubic hair. There are swellings palpable in the groins bilaterally. There is normal breast development.

Testosterone	1100 ng/dl	Male: (300-1200) Female : (20-75)

What is the likely diagnosis?

A. 17-alpha-hydroxylase deficiency
B. Androgen insensitivity syndrome
C. Klinefelter syndrome
D. Pituitary tumour
E. Turner syndrome

B. Androgen insensitivity syndrome

Primary amenorrhoea, little or no axillary and pubic hair, elevated testosterone → androgen insensitivity syndrome

Androgen insensitivity syndrome is the correct answer. This is an Xlinked recessive condition caused by end-organ resistance to testosterone causing genotypically male children (46XY) to have a female phenotype. Patients often present with primary amenorrhoea along with absent pubic/axillary hair. Bilateral groin swellings may reflect undescended testes and breast development may occur as a result of the conversion of testosterone to oestradiol. Testosterone is typically in the upper normal range for a post-pubertal male.

17-alpha-hydroxylase deficiency is incorrect. This is an uncommon form of congenital adrenal hyperplasia that may also present with primary amenorrhoea and external female genitalia. However, in this condition, testosterone will be low as a lack of androgens are characteristic of this disease.

Klinefelter syndrome is incorrect. This is a syndrome where a patient that is phenotypically male has an extra X chromosome (i.e. 47 XXY). Patients may have small testicles and infertility as predominant features. The patient in this scenario is phenotypically female.

Pituitary tumour is incorrect. This may cause delayed puberty if it causes impaired secretion of gonadotrophins (LH and FSH) i.e. hypogonadotropic hypogonadism. However, breast development may also be impaired with a pituitary tumour and testosterone will be in the normal range for a female, in contrast to this case where it is in the highnormal range for a male.

Turner syndrome is incorrect. This can cause primary amenorrhoea as a consequence of primary ovarian insufficiency. Breast development will be abnormal but pubic and axillary hair development will typically be normal. Unlike this case, testosterone will be in the normal range for a female. Patients have a 45XO karyotype.

Question 93 of 249

A 34-year-old lady with no previous medical history presents to her GP. She has felt anxious and on edge for the past month. She also reports feeling sweaty and developing shakes in her hand that are impairing her work as a teacher. She has occasional abdominal bloating but manages this with herbal tea.

On examination, you notice she is flushed and tremulous but alert. She has no goitre and no rashes across her skin. There is a small mass palpable in the left side of the pelvis that is non tender and there are normal bowel sounds overlying. Her visual fields and cranial nerve exams are normal. A thyroid scintigraphy shows normal uptake. An MRI scan is awaited.

TSH	5.0 mU/L (range 0.4-4.0)
T4	28 pmol/l (range 9-24)
Urine HCG	negative

What is the most likely diagnosis?

A. Jod-Basedow phenomenon
B. Exogenous iodine intake
C. Pituitary adenoma
D. Molar pregnancy
E. Struma ovarii

E. Struma ovarii

Ovarian teratomas can produce exogenous TSH

This lady has clear secondary hyperthyroidism. There are no signs of pituitary disease so we have to consider other sources of TSH production. In pregnancy, HCG can act as an agonist at the TSH receptor but the test here is negative. The pelvic mass and TSH rise can be explained by ovarian teratomas that rarely can secrete TSH.

Question 94 of 249

You are called to see a 35-year-old man on the Acute Medical Unit who is having a seizure.

He was admitted 2 days ago having presented acutely agitated to the Emergency Department. He gave a 10-year history of alcohol overuse and at that time had not had an alcoholic drink in 24 hours following an argument with his girlfriend where he vowed to give up alcohol. Prior to this he had been drinking 4-6 litres of cider per day plus additional spirits in variable amounts. He was admitted for detox and prescribed chlordiazepoxide, pabrinex and fluids. He has no other past medical history.

Nursing staff inform you that the patient has had no other seizures on this admission and has been eating and drinking small amounts today. However, he has been complaining of all over body pain and became confused 2-3 hours ago. The seizure is generalised tonic-clonic and self terminates after 3 minutes.

On examination post-seizure the patient is drowsy but responding to voice. His saturations are 100% on 15 litres oxygen via non-rebreathe mask and his temperature is 37.2 ºC. His heart rate is 110 beats per minute and blood pressure is 126/72 mmHg. His chest is clear, abdomen soft and non-tender and there is no focal neurology.

Repeat blood tests and arterial blood gas are taken.

Which electrolyte abnormality is most likely to have caused his seizure?

A. Hypocalcaemia
B. Hypoglycaemia
C. Hypokalaemia
D. Hypomagnesaemia
E. Hypophosphataemia

E. Hypophosphataemia

Hypophosphataemia is the most common electrolyte abnormality during alcohol withdrawal and is a recognised cause of seizures. It is often present at baseline in alcoholic patients and can further decrease in withdrawal. It is thought that this is due to effects on the proximal renal tubule. It can cause muscle pain and disorientation at very low levels (< 0.3 mmol/l).

Hypoglycaemia would also be possible but in the absence of diabetes, and with the patient eating and drinking, this is less likely. Hypoglycaemia is common in patients intoxicated with alcohol and these patients should be monitored with regular capillary blood glucose testing.

Low magnesium and low calcium both cause seizures but are unlikely to be lowered sufficiently in this patient to result in seizures in the absence of other clinical signs.

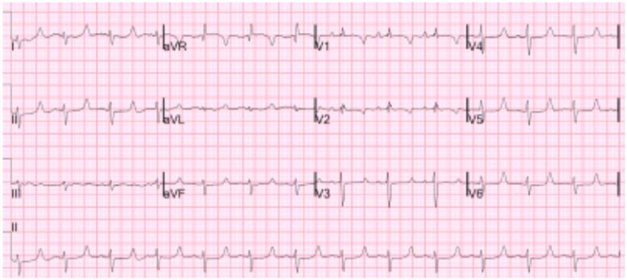
Low potassium is unlikely to result in seizures.

Question 95 of 249

A 40-year-old woman presents to the ambulatory clinic with a 4-day history of lethargy and weakness. There is also a history of intermittent cramps, particularly when trying to use her hands. She reports having had a recent gastrointestinal infection prior to the onset of her symptoms and has only just begun to eat and drink normally. Her past medical history consists of coeliac disease and vitiligo and her regular medications include folic acid and ferrous sulphate.

On examination, her observations are within normal limits. Her cardiorespiratory and abdominal examinations are unremarkable.

A set of routine blood tests are requested and an ECG is taken, as shown below:



What is the most appropriate intervention to improve this patient's symptoms?

A. 0.9% sodium chloride with potassium supplementation
B. Calcium chloride
C. Hartmann's solution
D. Magnesium sulphate
E. Sodium bicarbonate

B. Calcium chloride

Calcium chloride is correct. This patient has a history of lethargy and muscle cramping along with an ECG that demonstrates a prolonged QT interval. A combination of these features is highly suspicious for hypocalcaemia. Given the past medical history of coeliac disease, it is quite possibly a malabsorptive cause of hypocalcaemia contributing to this patient's symptoms. In the context of significant hypocalcaemia, intravenous replacement should be given with either calcium chloride or calcium gluconate.

0.9% sodium chloride with potassium supplementation is incorrect. There are no features of hypokalaemia (e.g. U waves) on this patient's ECG.

Hartmann's solution is incorrect. There is nothing to suggest that this patient would benefit from intravenous fluid resuscitation.

Magnesium sulphate is incorrect. This is classically used in the management of Torsades de Pointes. However, it is also commonly given in the treatment of other arrhythmias (e.g. atrial fibrillation). Given that this patient has a prolonged QTc interval, and not yet an arrhythmia (such as Torsades de Pointes that is associated with QTc interval prolongation), the priority should be to reduce the QTc interval which is with the administration of intravenous calcium. Furthermore, calcium would also help to improve the patient's physical symptoms.

Sodium bicarbonate is incorrect. This is not indicated in the management of hypocalcaemia or QTc interval prolongation.

Question 96 of 249

A 46-year-old man visits his general practitioner with an 8-week history of reduced libido. He has never experienced this before and is becoming very distressed. There is no history of recent illness or change in weight and he denies any headaches or visual symptoms.

On examination, his observations are taken which are all within normal range. He has a soft and non-tender abdomen with no stigmata of liver disease. His testicular examination is unremarkable and there is no evidence of scrotal swelling. There is a visible milky discharge from both nipples.

What is the most likely cause of this patient's presentation?

A. Carbamazepine
B. Lithium
C. Metoclopramide
D. Ropinirole
E. Spironolactone

C. Metoclopramide

Metoclopramide may result in galactorrhoea

This patient has reduced libido and galactorrhoea secondary to increased prolactin levels. Other features of raised prolactin levels include impotence in men and amenorrhoea in women. There are multiple causes of raised prolactin levels such as prolactinoma, PCOS, hypothyroidism and medication use. Dopamine inhibits the release of prolactin from the anterior pituitary gland and therefore drugs that decrease dopamine levels increase the amount of prolactin release. Of the drugs given in the question, metoclopramide is the only dopamine antagonist. It is a D2 and serotonin 5-HT3 receptor antagonist in the chemoreceptor trigger zone.

Carbamazepine is an anti-convulsant medication. Although it's mechanism of action is still widely debated, it is thought that it works by inhibiting sodium channels. Carbamazepine has multiple side effects including dizziness, drowsiness, hyponatraemia and thrombocytopenia. In rare cases, it can cause gynaecomastia. However, it does not cause galactorrhoea.

Lithium is a mood stabiliser used in the treatment of mania, bipolar disorder and recurrent depression. Lithium has multiple potential side effects including alopecia, arrhythmias, dizziness, seizures and coma. It does not cause galactorrhoea.

Ropinirole is a dopamine agonist used in the treatment of Parkinson's disease. Raised dopamine levels would decrease the amount of prolactin and, therefore, dopamine agonists are used to control galactorrhoea.

Spironolactone is an aldosterone antagonist. A side effect of spironolactone that is commonly tested in exam questions is the development of gynaecomastia. However, galactorrhoea is not seen.

Question 97 of 249

A 47-year-old woman has attended the endocrinology clinic for a review of hypothyroidism. She has been well maintained on 75micrograms of levothyroxine, once daily, for the past 4 years but describes worsening symptoms over the last 6 weeks.

She describes fatigue, low mood, constipation, and weight gain but is adamant she has not missed any doses of her medication.

She explains that she has recently been commenced on a few new medications by her GP including citalopram, ferrous fumarate, ramipril, propranolol, and metformin. All of her medications are taken in the morning.

Thyroid function tests today:

Thyroid-stimulating hormone (TSH)	12.4 mU/L	(0.5-5.5)
Free T4	3.6 pmol/L	(9.0 - 18)

Thyroid function tests 3 months ago:

Thyroid-stimulating hormone (TSH)	2.6 mU/L	(0.5-5.5)
Free T4	14.1 pmol/L	(9.0 - 18)

Which of her medications would explain her blood test results?

A. Citalopram
B. Ferrous fumarate
C. Metformin
D. Propranolol
E. Ramipril

B. Ferrous fumarate

Iron / calcium carbonate tablets can reduce the absorption of levothyroxine - should be given 4 hours apart

Ferrous fumarate is the correct answer. Iron tablets commonly affect levothyroxine absorption which can lead to symptomatic hypothyroidism. It is best to leave a 4-hour gap between consuming iron and levothyroxine.

Citalopram does not have any interactions with levothyroxine.

Metformin does not affect levothyroxine, however, levothyroxine can interfere with blood glucose and affect the ability of metformin.

Propranolol does not interact with levothyroxine.

Ramipril does not have any proven interactions with levothyroxine, however, studies are currently ongoing.

Question 98 of 249

A 22-year-old female, who is known to have type 1 diabetes mellitus, presents with weight loss, anorexia and fatigue for the last six months.

Her diabetes was well controlled with soluble insulin three times daily and long acting insulin in the evening but during the last six months she noticed that her insulin requirement has generally decreased and on three occasions she had hypoglycaemic attacks.

During the same time period she had lost approximately 7 Kg in weight and had generally lost her appetite. She had also been amenorrhoeic over the last three months.

On examination, she is thin (BMI 18), with a pulse rate of 70 beats per minute and a blood pressure of 110/70 mmHg with a postural drop.

Investigations reveal:

Serum sodium	125mmol/L
Serum potassium	5.3mmol/L
Serum urea	7.4mmol/L
Serum creatinine	100 mol/L
Serum glucose	7.5mmol/L
HbA1c	6.0%
Serum free T4	7.5 pmol/L
Serum TSH	5.5 pmol/L
Serum oestradiol	70 pmol/L (130-850)
Serum LH	2.5 mU/L (2-10)
Serum FSH	2.2 mU/L (2-10)
Serum prolactin	400 mU/L (50-450)
Serum calcium	2.9 mmol/l
Serum phosphate	0.8 mmol/l

What is the most appropriate investigation for this patient?

A. Thyroid autoantibodies
B. PTH concentration
C. Short synacthen test
D. Pregnancy test
E. Random cortisol concentration

C. Short synacthen test

This known type 1 diabetic female has developed Addisons disease on top of her diabetes. This explains the hypoglycaemic attacks, the decrease in her insulin requirement, the fatigue and weight loss.

Both type 1 DM and Addisons disease are features of Schmidt's disease (type 2 autoimmune polyendocrine syndrome) which is the diagnosis in this case.

The low T4, raised TSH, high calcium, low FSH, low LH, low oestradiol (hypogonadotrophic hypogonadism) are all features of Addisons disease.

The best investigation to diagnose Addisons disease is the short synacthen test.

It is of paramount importance when treating these patients is not to replace thyroxine before hydrocortisone because this will induce Addisonian crisis.

Question 99 of 249

A 60-year- old female presented with a six month history of polyuria, polydipsia and generalised aches and pains.

She is a known hypertensive for ten years and is taking bendroflumethiazide 2.5 mg daily. She has been taking calcium and vitamin D supplements for the last two years as she has a strong family history of osteoporosis.

On examination, her pulse rate is 80 beats per minute and her blood pressure is 150/90 mmHg. Cardiovascular, respiratory and abdominal examination were normal.

Investigations reveal:

Serum sodium	130 mmol/L
Serum potassium	3.1 mmol/L
Serum urea	7.7 mmol/L
Serum creatinine	88 mol/L
Serum corrected calcium	2.9 mmol/L
Phosphate	0.8 mmol/L
PTH	4.5 pmol/L (0.9-5.4)
Urinalysis	glycosuria ++

What is the most likely cause of this ladys symptoms?

A. Primary hyperparathyroidism
B. Vitamin D excess
C. Bendroflumethizide induced hypercalcaemia
D. Familial hypocalciuric hypercalcaemia
E. Diabetes mellitus

A. Primary hyperparathyroidism

The PTH level in primary hyperparathyroidism may be normal

This lady has hypercalcaemia which may be due to bendroflumethiazide or vitamin D excess, but the PTH level is inappropriately normal in the context of hypercalcaemia which indicates primary hyperparathyroidism rather than any other cause of hypercalcaemia in which the PTH would be suppressed by homeostatic mechanisms.

In addition, the phosphate level is low which is typical of primary hyperparathyroidism. The glycosuria is a distractor.

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A 20-year-old female admits to abusing codeine and diclofenac, up to 30 tablets per day. She attends the emergency department demanding help with her addiction. Her baseline bloods include:

pH	7.28
PCO2	2.9 kPa
PO2	8.5 kPa
HCO3-	16 mmol/l
Na+	130 mmol/l
K+	6 mmol/l
Cl-	110 mmol/l
HCO3-	16 mmol/l

What is the most likely diagnosis?

A. Lactic acidosis
B. Type 1 renal tubular acidosis
C. Type 2 renal tubular acidosis
D. Type 4 renal tubular acidosis
E. Ketoacidosis

D. Type 4 renal tubular acidosis

Type 4 renal tubular acidosis is due either to a deficiency of aldosterone or to a resistance to its effects.

Causes include:

- Aldosterone deficiency (hypoaldosteronism): Primary vs.
- hyporeninaemic
- Aldosterone resistance
- → 1.Drugs: Non-steroidal anti-inflammatories, angiotensin converting enzyme inhibitors, angiotensin 2 receptor blockers, eplerenone, spironolactone, trimethoprim, pentamidine
- → 2.Pseudohypoaldosteronism

Question 101 of 249

A 30-year-old south Asian woman is admitted to the accident and emergency department with abdominal pain. She is thought to be constipated. Initial blood results with subsequent tests are listed below. Urine is clear and an ECG performed is normal. Examination is unremarkable with no oedema, and blood pressure 105/68 mmHg.

pH	7.250
Bicarbonate	18.0 mmol/l
Base excess	-8.0 mmol/l
Anion gap	Normal
Potassium	7.2 mmol/l
Creatinine	56 mmol/l
Glucose	5.3 mmol/l
Thyroid function	Normal
Aldosterone	Normal
Renin	Normal
Protein electrophoresis & immunoglobulins	Normal
Urinary sodium	94 mmol/l (normal range >20mmol/L)
Urinary potassium	26.8 mmol/l (normal range >25 mmol/L)
17- hydroxyprogesterone	Normal
Short synacthen test (basal)	320 nmol/l
Short synacthen test (30 mins)	750 nmol/l

What is the likely diagnosis?

A. Renal tubular acidosis type 1
B. Renal tubular acidosis type 2
C. Renal tubular acidosis type 4
D. Gitelman syndrome

E. Adrenal insufficiency

C. Renal tubular acidosis type 4

Renal tubular acidosis type 4 is a condition associated with increased urinary ammonia secondary to hypoaldosteronism or pseudohypoaldosteronism. This leads to hyperkalaemia and a hyperchloraemic metabolic acidosis with a normal anion gap.

Both renal tubular acidosis type 1 & 2 lead to low potassium in the context of acidosis.

Gitleman: An autosomal recessive kidney disorder characterized by hypokalemic metabolic alkalosis with hypocalciuria and hypomagnesemia.

Adrenal insufficiency is effectively ruled out with a normal short synacthen test. Candidates would be expected to understand how to perform a short synacthen test and to interpret the results. Laboratory references vary, but in general a basal plasma cortisol should exceed 170 nmol/L and should rise to above 580 nmol/L.

Question 102 of 249

A 45-year-old gentleman is admitted to the psychiatry ward with a two day history of visual hallucinations. His past medical history is relevant for renal transplant last year, indicated because of membranous glomerulonephritis. He is on an established regime of immunosuppressants.

On examination, there is no evidence of focal neurology. Cranial nerves I to XII are normal, with equal and reactive pupils. There are no cerebellar signs. Plantars are equivocal on the left and downgoing on the right. Abbreviated mental test score (AMTS) is 8/10. An urgent CT head is unremarkable.

Which medication may be implicated as a cause of this patient's presentation?

A. Mycophenolate mofetil
B. Azathioprine
C. Prednisolone
D. Tacrolimus
E. Ciclosporin

C. Prednisolone

This patient is likely to be experiencing steroid-induced psychosis, and therefore, prednisolone is the correct answer. Most immunosuppressants have side-effects, and it is important to be aware of common associations. In addition to psychosis, steroids such as prednisolone may cause weight gain, secondary diabetes, osteoporosis and myopathy.

Ciclosporin and tacrolimus are both calcineurin inhibitors. Ciclosporin is associated with nephrotoxicity, gum hypertrophy and hirsutism. Tacrolimus may cause tremor and secondary diabetes.

Mycophenolate mofetil and azathioprine are both anti-metabolites. Mycophenolate mofetil is associated with mucositis.

Azathioprine is steroid-sparing but can induce rashes, hepatitis and pancreatitis.

Question 103 of 249

You are asked to review a 67-year-old man who is currently an inpatient on a surgical ward with new paraesthesia in his fingers. He was admitted for an elective parathyroidectomy three days ago for fairly long-standing hyperparathyroidism and subsequent hypercalcaemia. He had a single parathyroid adenoma excised which had been identified on preoperative MIBI scanning. The procedure was without complications but he is now complaining of a tingling sensation in his fingers that he first noticed about twelve hours ago. He also complains of new severe pain in both of his ankles which is worse when he walks, but also present at rest. The surgical SHO has already arranged x-rays of the patient's ankles and these reveal multiple osteolytic lesions which have been reported as being suspicious for metastatic disease. He is otherwise fit and well and his only regular medications are paracetamol, tramadol and prophylactic dalteparin. His blood tests are as follows.

Adjusted Calcium	1.84 mmol/L
Magnesium	0.7 mmol/L

What is the most likely explanation for his current symptoms?

A. Metastatic parathyroid cancer
B. Secondary hyperparathyroidism
C. Hypomagnesaemia
D. Hungry bone syndrome
E. Secondary hypoparathyroidism

D. Hungry bone syndrome

Hypocalcaemia after parathyroid surgery is relatively common and usually 'benign' and associated with a transient hypoparathyroidism. However, it can sometimes be more marked and give rise to symptoms such as perioral or finger paraesthesia. This state alone would not, however, explain his ankle pain or x-ray findings.

Although hypomagnesaemia may also be present and should be treated, it does not explain the symptoms. Metastatic parathyroid cancer is a possibility given the x-ray findings, but is very uncommon and is less likely given that his hyperparathyroidism and hypercalcaemia was long standing (i.e. indolent). Secondary hyperparathyroidism is the syndrome of appropriately raised parathyroid hormone in response to hypocalcaemia, usually secondary to chronic kidney disease. Secondary hypoparathyroidism describes the normal parathyroid hormone suppression that occurs in hypercalcaemia secondary to non-parathyroid causes, such as malignancy.

Question 104 of 249

A 75-year-old man presents to referred to the diabetic clinic by his general practitioner with newly identified hyperglycaemia. He had presented with a two-month history of polyuria, polydipsia and diarrhoea and was found to have a blood sugar of 18.4 mmol/L. Over this time period he had lost 6kg and now weighed 61kg. His past medical history includes hypertension and a deep vein thrombosis which was diagnosed three months ago. He takes amlodipine 10mg and warfarin.

What is the diagnosis?

A. Glucagonoma
B. Type one diabetes mellitus

C. Cushing's syndrome
D. Drug induced diabetes
E. Type two diabetes mellitus

A. Glucagonoma

Glucagonoma is an uncommon tumour of the pancreatic alpha cells. It can present with new or worsening diabetes mellitus, venous thromboembolism, the classic rash of necrolytic migratory erythema (a painful, pruritic maculopapular rash occuring typically at sites of friction with clothing...) and other symptoms of hyperglucagonaemia (diarrhoea, weight loss, anaemia). Type one diabetes mellitus can of course present with the osmotic symptoms of hyperglycaemia and weight loss but would be unlikely in a patient of this age with no history of auto-immune disease. Type two diabetes mellitus would be uncommon in a non-obese older patient and is less likely to present with osmotic symptoms.

Question 105 of 249

A 25-year-old woman is brought to the emergency department by ambulance after being found unwell by friends. Collateral history reported by the paramedics indicated that the patient had been unwell for 3 days with vomiting and diarrhoea. Her housemate said that the patient had been unable to eat since becoming unwell and that he did not think she had been taking her regular insulin during that time. The patient herself was too disorientated to give any history. The paramedics had found both novorapid and lantus insulin pen devices in the patients fridge.

General examination indicated a drowsy and dehydrated patient with generalised abdominal tenderness but no evidence of focal peritonism.

Please see below for selected investigation results.

Observations: blood pressure 86 / 57 mmHg; heart rate 127 beats per minute; respiratory rate 28 per minute; O2 saturations 100 % (room air); Temperature 37.1 oC.

Fingerpick blood glucose	38.2 mmol / L
Fingerpick blood ketones	8.7 mmol / L
Urea	12.5 mmol / L
Creatinine	123 micromol / L
Sodium	148 mmol / L
Potassium	3.7 mmol / L
Haemoglobin	156 g / dL
White cell count	14.3 x 10>3 / microlitre
Neutrophils	11.3 x 10>3 / microlitre
Platelets	453 x 10>3 / microlitre

Arterial blood gas (room air)

pH	7.05
PaCO2	15 mmHg (reference 32-43)
PaO2	99 mmHg (reference 70-100)
Bicarbonate	12.3 mmol / L (reference 20.0-26.0)
Chloride	111 mmol / L (reference 99-108)
Lactate	7.5 mmol / L

What is the appropriate strategy for intravenous insulin treatment in this patient?

A. Variable rate insulin infusion without initial insulin bolus, converting to subcutaneous insulin once acidosis resolved
B. Fixed rate insulin infusion without initial insulin bolus, convertingto subcutaneous insulin once patient is eating and drinking normally
C. Fixed rate insulin infusion following initial insulin bolus, converting to subcutaneous insulin once patient is eating and drinking normally
D. Variable rate insulin infusion with initial insulin bolus, converting to subcutaneous insulin once acidosis resolved
E. Variable rate insulin infusion without initial insulin bolus, converting to subcutaneous insulin once ketonaemia resolved

B. Fixed rate insulin infusion without initial insulin bolus, converting to subcutaneous insulin once patient is eating and drinking normally

The patient is presenting in diabetic ketoacidosis due to vomiting, dehydration and omission of prescribed insulin.

The Joint British Diabetes Society recommend an insulin infusion at rate 0.1 units / kg / h. An initial bolus of insulin is not advised due to a randomised controlled trial that found no benefit. Fixed rate insulin infusions are now preferred over titration of insulin dose against blood sugar levels (sliding scale). This is due to the fact that blood glucose may correct more quickly than ketoacidosis and so ensures adequate insulin to eradicate ketones.

There is no consensus between expert bodies as to biochemical endpoint of DKA, therefore it is advised that patients are transferred onto subcutaneous insulin once they are eating and drinking normally. It is vital to ensure an overlap between the administration of intravenous and subcutaneous insulin to avoid recurrent ketogenesis.

Question 106 of 249

A 72-year-old woman is recovering on the neurosurgical unit following a subdural hemorrhage. Four days earlier she underwent Burr hole surgery. You are asked to see her due to a persistently low sodium for the past three days. You note the following investigations:

Day 2 post-surgery

Serum Na+	116 mmol/l

Day 3 post-surgery

Serum Na+	117 mmol/l

Day 4 post-surgery

Serum Na+	115 mmol/l
Urinary Na+	25 mmol/l
Serum osmolality	280 mmol/l

Examination of the patient demonstrates dry mucous membranes and delayed capillary refill time.

What is the most likely diagnosis?

A. SIADH
B. Diabetes insipidus
C. Cerebral salt wasting syndrome
D. Renal tubular acidosis type IV
E. Sheehan's syndrome

C. Cerebral salt wasting syndrome

Diabetes insipidus is classically associated with hypernatraemia. Sheehan's syndrome refers to the specific situation of pituitary necrosis following childbirth. The cardinal feature of renal tubular acidosis type IV is hyperkalaemia.

This leaves SIADH and cerebral salt wasting syndrome. The hydration status in this patient can be considered hypovolaemic making SIADH unlikely (typically euvolaemic). Additionally, this diagnosis should only be made in the absence of hypothyroidism and adrenal dysfunction.

Cerebral salt wasting syndrome can occur following neurosurgery. It occurs due to sodium wasting in the urine. Comparatively, it is treated with replacing fluid and sodium losses.

Question 107 of 249

A 68-year-old gentleman presents with severe abdominal pain. He has a past medical history of depression and type 2 diabetes. His medications include; levemir, sitagliptin and gliclazide. He denies smoking, alcohol or illicit drug use. He also claims he has been taking all his diabetic medications.

On examination he is overweight. He has dry mucus membranes. He has generalised abdominal pain with no rebound or guarding. His bowel sounds are present and normal. His blood pressure is 101/76 mmHg, his pulse rate is 113 beats per minute and his temperature is 37.8ºC.

Investigations;

Erect Chest X Ray	Clear
Ultrasound scan of the abdomen	Biliary duct 3mm Normal appearance of gallbladder Diffuse increase echogenicity of liver, normal size Pancreas no visible abnormality
Hb	134 g/dL
WCC	13.1 *10^9/l
Platelets	234 *10^9/l
MCV	89 fL
Sodium	148 mmol/L
Potassium	5.6 mmol/L
Creatinine	165 µmol/L
Urea	10.4 mmol/L
Alkaline Phosphatase	76 IU/L
Alanine Transaminase	42 IU/L
Gamma-glutamyl transpeptidase	60 IU/L
Amylase	1378 IU/L
Glucose	38 mmol/l
Urinalysis	WCC + RBC ve Ketones +

What is the likely underlying diagnosis?

A. Diabetic ketoacidosis
B. Perforated small bowel
C. Drug induced pancreatitis
D. Acute hepatitis
E. Alcohol induced pancreatitis

C. Drug induced pancreatitis

Answer: Sitagliptiin induced pancreatitis.

Patients with perforated small bowel or DKA can present with a raised amylase. The level suggests the cause is most likely pancreatitis. A perforated small bowel would more likely show free gas on a Chest X ray, diminished bowel sounds and guarding. Diabetic ketoacidosis would show more ketones on urinalysis and is rare, although still possible, in type 2 diabetics.

The gentleman has no evidence of gallstones or biliary obstruction. This combined with a normal liver function tests makes gallstone unlikely. The patient denies alcohol intake and non-alcoholic fatty liver can produce the appearances on ultrasound scan.

Both dipeptidyl peptidase 4 inhibitors (sitagliptin) and glucagon like peptide-1 agonists have been linked to pancreatitis. They are still both under investigation but is to be discontinued in the event of an episode of pancreatitis.

Question 108 of 249

A 51-year-old lady librarian attends outpatient clinic with painful eyes. She reports that her vision has deteriorated over the past four weeks. On examination, she has proptosis, periorbital oedema and a painful complex ophthalmoplegia. She appears anxious and is worried about not coping at work. At present she smokes ten cigarettes daily.

What would be the most appropriate next step in managing this patient?

A. IV methylprednisolone
B. Surgical decompression
C. Smoking cessation advice
D. Total thyroidectomy
E. Artificial tear drops

A. IV methylprednisolone

IV methylprednisolone is the treatment of choice for moderately severe active Graves' ophthalmopathy. IV steroids have fewer side effects than oral steroids. If symptoms or vision do not improve then urgent surgical decompression should be considered.

Artificial tear drops are useful for symptomatic relief.

Total thyroidectomy has shown no benefit in the treatment of thyroid eye disease.

Outcomes have been shown to be worse in those patients who smoke, therefore smoking cessation advice should be given.

Question 109 of 249

A 31-year-old female presents with a 2-day history of abdominal pain, a 5-day history of diarrhoea and vomiting and reduced appetite. She is a known type one diabetic with background diabetic retinopathy and stage 3 chronic kidney disease. She usually takes 32 units lantus at night and variable doses of Novomix with meals however due to her poor appetite she has not taken these for 2 days. On examination, she looks unwell. The airway is patent and chest is clear. Respiratory rate is 26/min with normal oxygen saturations on

air. Pulse is 120/min and thready with a capillary refill of 3 seconds centrally. Blood pressure is 103/45 mmHg with a temperature of 36.7 C. Abdomen is generally tender without guarding. Capillary blood glucose is 26 and ketones are 4.9. Arterial blood gas is as follows:

pH	7.32
pO2	11.6 kPa
pCO2	3.32 kPa
Bicarbonate	14 mmol/l
Base Excess	-6.5 mmol/l
Lactate	2.1 mmol/l

The patient is currently being fluid resuscitated appropriately.

What form of insulin therapy would you advise?

A. Dose of novomix now and increase usual lantus by 8 units
B. Variable rate i.v insulin, continue all s/c insulins
C. Recommence normal s/c insulin regimen
D. Fixed rate i.v insulin, continue lantus
E. Fixed rate i.v insulin, stop all s/c insulin

D. Fixed rate i.v insulin, continue lantus

This patient has a compensated metabolic acidosis and despite her reasonably preserved pH, a bicarbonate of 14 suggests significant metabolic acidosis. In the presence of high blood ketones and hyperglycaemia, the diagnosis is diabetic ketoacidosis (DKA). Fluid therapy and potassium supplementation is a key part of DKA management but will not be dealt with in this question. Current recommendations advise fixed rate insulin with the addition of 10 % dextrose when capillary blood glucose drops below 14. The aim is to correct the underlying ketoacidosis brought about by a hypoinsulinaemic state. Patients should be continued on their long acting insulin but short/medium term insulins should be held. This ensures a smooth transfer from fixed rate to usual insulin regimen when the patient has clinically and biochemically improved.

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A 48-year-old man who was diagnosed with type 2 diabetes mellitus presents for review. During his annual review he was noted to have the following results:

Total cholesterol	5.3 mmol/l
HDL cholesterol	1.0 mmol/l
LDL cholesterol	3.1 mmol/l
Triglyceride	1.7 mmol/l
HbA1c	6.4%

A QRISK2 score is calculated showing that he has a 12% 10-year risk of developing cardiovascular disease. His current medication is metformin 500mg tds.

According to recent NICE guidelines, what is the most appropriate action?

A. Simvastatin 40mg on
B. Lifestyle advice, repeat lipid profile in 3 months
C. Atorvastatin 40mg on
D. Atorvastatin 20mg on
E. Increase his metformin slowly to 1g tds

D. Atorvastatin 20mg on

NICE recommend the following when considering the use of statins in patients with type 2 diabetes mellitus:

> *Offer atorvastatin 20 mg for the primary prevention of CVD to people with type 2 diabetes who have a 10% or greater 10-year risk of developing CVD.*

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You review a 38-year-old woman with type 1 diabetes mellitus in clinic. Her diabetes is currently controlled with a basal-bolus regime. She takes no other medication apart from citalopram 20mg od for depression. She was diagnosed with type 1 diabetes at the age of 13 years. Her most recent bloods show:

Na +	142 mmol/l
K +	3.9 mmol/l
Urea	4.9 mmol/l
Creatinine	79 µmol/l
Total cholesterol	4.4 mmol/l
HDL cholesterol	1.2 mmol/l
LDL cholesterol	1.8 mmol/l
Triglyceride	1.3 mmol/l

Urine dip: No protein or blood

What is the most appropriate management with regards to lipid modification?

A. Start atorvastatin 10mg on
B. Start atorvastatin 20mg on
C. Start atorvastatin 40mg on
D. Perform a QRISK2 assessment
E. Reassure her that lipid modification therapy is not required atthis stage

B. Start atorvastatin 20mg on

NICE specifically state that we should not use QRISK2 for type 1 diabetics. Instead, the following criteria are used:

- older than 40 years, or
- have had diabetes for more than 10 years or
- have established nephropathy or
- have other CVD risk factors

This patient has had diabetes for 25 years so we should start atorvastatin 20mg on.

Question 112 of 249

A 64-year-old man is reviewed in clinic. He has a history of ischaemic heart disease and was diagnosed with type 2 diabetes mellitus around 12 months ago. At this time of diagnosis his HbA1c was 7.6% (60 mmol/mol) and he was started on metformin which was titrated up to a dose of 1g bd. The most recent bloods show a HbA1c of 6.8% (51 mmol/mol). He has just retired from working in the IT industry and his body mass index (BMI) today is 28 kg/m². His other medication is as follows:

Atorvastatin 80mg on Aspirin 75mg od Bisoprolol 2.5 mg od Ramipril 5mg od

What is the most appropriate next step?

A. Add sitagliptin
B. Make no changes to his medication
C. Add empagliflozin
D. Add pioglitazone
E. Add exenatide

C. Add empagliflozin

Normally NICE recommend we only add another drug if the HbA1c has risen to >= 58 mmol/mol (7.5%) at this stage. However, as this patient has established cardiovascular disease he should also be given an SGLT-2 inhibitor (e.g. empagliflozin) in addition to metformin.

Question 113 of 249

A 56-year-old man with a history of hypertension presents for review. As part of his annual health check he has a U&E, HbA1c and cholesterol check done. The following results are obtained:

His blood pressure today is 128/78 mmHg. His only regular medication is ramipril 5mg od.

Na +	142 mmol/l
K +	4.6 mmol/l
Urea	5.2 mmol/l
Creatinine	88 µmol/l
Total cholesterol	5.2 mmol/l
HbA1c	45 mmol/mol (6.3%)

His 10-year QRISK2 score is 7%.

What is the most appropriate action following these results?

A. Start atorvastatin 20mg on
B. Arrange a fasting glucose sample
C. Diagnose type 2 diabetes mellitus
D. Increase the dose of ramipril
E. Add amlodipine 5mg od

B. Arrange a fasting glucose sample

His QRISK2 score is < 10% so no action needs taking about his cholesterol. His blood pressure is also well controlled.

His HbA1c is on the higher side and currently resides in the pre-diabetes range (42-47 mmol/mol). A HbA1c reading cannot however be used to exclude diabetes - a fasting sample should therefore be arranged.

Question 114 of 249

A 72-year-old female presents with 5 days of general decline following a recent urinary tract infection, treated with oral antibiotics in the community by the GP. She is known to be a type 2 diabetic, diagnosed 28 years ago and insulin dependent for the past 6 years. She is normally on 46 units Lantus, 23 units TDS Novorapid. On examination, she is not orientated in time or place, GCS 14/15. She has no focal neurology, chest and cardiovascular auscultation are unremarkable. You demonstrate suprapubic tenderness on deep palpation but the abdomen is other soft and non-tender, bowel sounds are present. She appears extremely dehydrated: her mucous membranes are dry, peripheries cool with capillary refill time of 4 seconds and JVP +1 cm above the angle of Louis. Her blood sugar is 31mmol/L and a venous blood gas demonstrates pH 7.22, lactate 2 mmol/l, ketones 5 mmol/l. A urine dip is awaited.

What is the most likely diagnosis?

A. Hyperglycaemic hyperketotic state
B. Hyperglycaemia secondary to poor medical compliance duringrecent acute illness
C. Urosepsis secondary to inadequately treated UTI
D. Diabetic ketoacidosis
E. Dehydration secondary to poor oral intake

D. Diabetic ketoacidosis

The patient is acidotic with ketones >3 mmol/l demonstrated, on a background of known insulin dependence. Although she is known to be a type 2 diabetic, it should be remembered that both types of diabetics can present as DKA, particularly advanced T2 DM who produce little to no endogenous insulin and are hence unable to shut down ketogenesis. Treatment should be as per DKA protocols, with intravenous fluids, fixed rate insulin infusion @ 0.1 unit/kg/hour, thromboprophylaxis, broad spectrum antibiotics and appropriate K+ replacement with insulin1.

Question 115 of 249

A 29-year-old female is admitted to the Emergency Department following an episode of collapse. She denies prodromal symptoms and woke up after an undetermined period to find herself lying face down on the ground. She has recently consulted her GP regarding feelings of generalised weakness, intermittent palpitations and dizziness.

Her past medical history is remarkable for hypothyroidism and rheumatoid arthritis, previously managed with infliximab, and complicated 3 months ago by a diagnosis of TB. Her regular medications include methotrexate, folic acid, levothyroxine, artificial tears, rifampicin, and isoniazid.

During the examination, the patient complains of recurrent palpitations. The cardiac monitor shows broad-complex tachycardia. Her blood pressure is stable at 117/68mmHg and she is given a bolus of amiodarone.

Her venous blood gas is as follows:

pH	7.31
pCO2	4.3 kPa
pO2	6.3 kPa
Na+	137 mmol/l
K+	2.1 mmol/l
Cl-	114 mmol/l
iCa2+	1.05 mmol/l
Glucose	5.4 mmol/l
HCO3	15.6 mmol/l
BE	-9.5 mmol/l

Some additional investigations are requested:

Urine dipstick	pH 5.0 glucose ++ protein +

XR chest	unremarkable
XR abdomen	normal bowel gas pattern, no evidence of abnormal renal calcification

What is the most likely cause of the metabolic abnormalities described?

A. Addison's disease
B. Fanconi syndrome
C. Rheumatoid arthritis
D. Sjogren's syndrome
E. Liver cirrhosis

B. Fanconi syndrome

The patient has a normal anion gap hyperchloraemic metabolic acidosis associated with hypokalaemia. This, in combination with the absence of GI symptoms, should alert you to the possibility of underlying renal tubular acidosis.

Fanconi syndrome is a disorder of proximal renal tubular function that leads to abnormal loss of bicarbonate, glucose, potassium, phosphate, uric acid and amino acids in the urine. Patients have features of type 2 renal tubular acidosis, accompanied by hypophosphataemia, glycosuria, and aminoaciduria. Presenting features include polyuria, polydipsia, osteomalacia (rickets and growth failure in children) and symptoms secondary to the electrolyte abnormalities associated with the disorder. Rifampicin has recently been implicated in the development of Fanconi syndrome and is the most likely cause in this case.

Rheumatoid arthritis and Sjogren's syndrome are causes of type 1 (distal) renal tubular acidosis; a failure of hydrogen ion secretion in the distal tubule that causes hypokalaemia and nephrocalcinosis. The absence of abnormal renal calcification on the patient's x-ray points away from this as the diagnosis.

Addison's disease typically causes hyperkalaemia, often associated with other electrolyte abnormalities including hyponatraemia and hypoglycaemia. It would, therefore, be inconsistent with the biochemistry provided.

Liver cirrhosis does not typically cause renal tubular acidosis unless it is itself a manifestation of Wilson's disease. There is nothing in the question to suggest Wilson's disease as the underlying diagnosis, however, and the occurrence of this pattern of metabolic abnormality would still be predicated on the development of a complicating Fanconi syndrome.

Question 116 of 249

A 54-year-old man with a history of type 2 diabetes is recovering on the surgical ward having suffered an episode of acute pancreatitis some 4 days earlier. Medication for glucose control includes metformin, dapagliflozin and liraglutide. On examination his blood pressure is 135/80 mmHg, pulse is 72 and regular. His body mass index is 35 kg/m². A recent HbA1c is 63 mmol/mol, renal function is reported as normal.

Which of the following is the correct course of action with respect to his long term blood glucose lowering medication?

A. Stop metformin
B. Stop dapagliflozin
C. Stop liraglutide
D. Continue usual medication
E. Stop metformin and liraglutide

C. Stop liraglutide

This patient is now stable following his episode of pancreatitis, but in view of a number of studies suggesting an association between GLP-1 agonist prescription and pancreatitis, it should be withdrawn in patients who suffer pancreatitis whilst on therapy, and not instituted in patients who have a history of the disease. For this reason liraglutide should be withdrawn.

With respect to metformin, it should potentially be discontinued during periods of increased risk of tissue hypoxia, (e.g. during the acute period of pancreatitis), but there is no indication it should be omitted over the longer term. In regards to metformin and renal impairment, guidance has recently been revised to support metformin initiation at glomerular filtration rate as low as 45ml/min.

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A 22-year-old student comes to the Emergency department with a cough productive of rusty coloured sputum. She has been suffering from increased shortness of breath, night sweats and fevers for the past 48 hours. Current medication includes daily hydrocortisone for congenital adrenal hyperplasia and the combined oral contraceptive pill. Current bloods are shown below:

Hb	131 g/l	Na^+	134 mmol/l
Platelets	201 * 10^9/l	K^+	4.1 mmol/l
WBC	14.9 * 10^9/l	Urea	7.0 mmol/l
Neuts	10.1 * 10^9/l	Creatinine	82 µmol/l
Lymphs	1.2 * 10^9/l	CRP	185 mg/l
Eosin	0.4 * 10^9/l		

Which of the following is the most appropriate way to manage her steroid hormone replacement?

How should you manage her steroid replacement?

A. Convert to 200mg hydrocortisone IV BD
B. Increase the daily dose by 50%
C. Increase the daily dose by 100%
D. Reduce the daily dose by 50%
E. Keep the daily dose the same

C. Increase the daily dose by 100%

Patients with congenital adrenal hyperplasia who are managed with steroid hormone replacement should be managed in the same way as patients with Addison's disease. In other words, during a period of significant acute infection like the pneumonia here, the dose of corticosteroid should be doubled.

With the patient able to swallow, having presented early with her pneumonia, switching to IV hydrocortisone would represent excess steroid replacement and is not appropriate here. The other options including only small increases in steroid dose, reductions or maintaining the status quo, run the risk of adrenal crisis.

Question 118 of 249

A 29-year-old woman comes to the clinic with feelings of anxiety, palpitations and a resting tremor present over the past 2 weeks. She also has symptoms of a flu-like illness and pain over her anterior neck. On examination,you reveal tenderness over the thyroid. Her blood pressure is 115/88 mmHg, she has a fine tremor at rest, sweaty palms and a tachycardia of 88 beats per minute. TSH is <0.05 U/ml.

Which of the following is the most appropriate intervention?

A. Thyroxine
B. Carbimazole
C. Propylthiouracil
D. Carbimazole and thyroxine
E. Propranolol

E. Propranolol

The short history of flu-like symptoms, coupled with pain over the thyroid gland is consistent with a diagnosis of subacute thyroiditis. The presentation with thyrotoxicosis is caused by a transient increase in thyroid hormone release, rather than over production, hence anti-thyroid drugs are not indicated. Symptomatic relief with propranolol is indicated, as are non-steroidal anti-inflammatory agents for management of pain and inflammation. A period of hypothyroidism may follow, with later recovery to normal thyroid function.

Anti-thyroid drugs are not needed, carbimazole and propylthiouracil are therefore incorrect, as is the block-replace regimen option. Although a transient period of hypothyroidism may be seen after the thyrotoxicosis subsides, thyroxine replacement is usually not needed.

Question 119 of 249

A 19-year-old woman comes to the endocrine clinic for review. She has problems with hirsutism and irregular periods, and troublesome weight gain. Her GP has just stressed the need to lose weight and offered no pharmacological intervention. She takes no medication from the doctor and is currently studying law. Examination reveals a blood pressure of 135/85 mmHg, pulse is 65 beats per minute and regular. body mass index is 32kg/m². You confirm extensive hirsutism affecting the beard line, upper lip and the nipples. there is acne over the face and the upper chest. Relevant bloods include:

testosterone	4.8 nmol/l (upper limit of normal 2.1 nmol/l)
LH:FSH ratio	2.1
fasting glucose	5.0 mmol/l

Her main concern is hirsutism.

Which of the following is the most appropriate intervention?

A. Co-cyprindiol
B. Clomiphene
C. Levonorgestrel
D. Metformin
E. Pioglitazone

A. Co-cyprindiol

Co-cyprindiol contains both cyproterone, an anti-androgen, and ethinylestradiol, (a synthetic oestrogen). In combination, used for the treatment of polycystic ovarian syndrome, the most likely diagnosis here, co-cyprindiol significantly reduces symptoms of hirsutism and acne, both related to androgen excess.

Clomiphene is the preferred option for inducing ovulation, and is preferred to metformin for this purpose, although the two are sometimes used in combination in the obese population. Pioglitazone is also effective in reducing ovarian insulin resistance, and inducing ovulation, but is not used due to its adverse event profile. Progesterone, (levonorgestrel), is ineffective in managing hirsutism.

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A 37-year-old female presents to the medical outpatient department with a progressive loss of libido. She attributes this to persistent diarrhoea, which she has noted over the last 6 months. She has also lost 16kg of weight and feels fatigued. She has noticed that her eyes have become grossly protuberant and she has double vision on looking towards either the right or left. She also experiences painful watering of her eyes.

On examination she has a marked tremor in both hands, her heart rate is irregularly irregular and she has marked exophthalmos. There is an audible bruit on auscultation of the thyroid gland.

Her laboratory investigations reveal:

Hb	130 g/l
MCV	77 fl
MCH	29 pg
WBC	7.4 * 10 /l9
Plt	430 * 10 /l9
TSH	0.03 mU/l (0.4 3.6 mU/l)
Total T4	302 nmol/l (68 174 nmol/l)

CT scan of the orbits reveals taut optic nerves and retro-orbital oedema.

Which of the following would be the most appropriate management of her eye condition?

A. Treatment with a block and replace regimen
B. Treatment with radioactive iodine
C. Treatment with IV methylprednisolone
D. Surgical removal of the thyroid gland
E. Orbital decompression surgery

C. Treatment with IV methylprednisolone

The question aims to address the ophthalmopathy associated with Graves disease and tests the candidates understanding of the correct approach to a patient with painful and significant eye disease. The treatment of choice is the administration of systemic steroids to lessen the inflammation and provide symptomatic relief. Treatment of the underlying thyrotoxicosis is essential, but it will not directly result in an improvement in the ophthalmopathy.

Radioactive iodine therapy may worsen Graves ophthalmopathy and should not be the initial treatment option. Treatment-induced hypothyroidism must be avoided as this may also worsen the eye problems.

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A 32-year-old female presents to the infertility clinic with an inability to conceive. She is overweight, with a body-mass index of 32 kg/m², and has noticed increased hair growth over her face and chest over the last 12 months. Her periods are irregular and she has also noticed a deepening of her voice. An ultrasound of the pelvis has revealed the presence of multiple cysts in both ovaries. She has been treated with cyproterone acetate for her hirsutism but was informed that she should not attempt conception whilst on the drug. She now wishes to conceive.

On examination, she has a cushingoid appearance, with abdominal striae and her blood pressure is 140/85 mmHg.

Laboratory investigations reveal:

9:00 am Cortisol	710 nmol/l (170 700 nmol/l)
LH	28 iU/l (1 20 iU/l)
Basal FSH	4.7 iU/l (1.0 8.8 iU/l)
DHEAS	509 µg/dl (31 228 µg/dl)
Prolactin	602 mU/l (<360 mU/l)
17 OH Progesterone	54 ng/dl (<80 ng/dl)

Which of the following treatment options would be most appropriate for the treatment of infertility?

A. Metformin
B. Spironolactone
C. Reverse circadian rhythm steroids
D. Clomiphene citrate
E. Cabergoline

D. Clomiphene citrate

The Rotterdam criteria for the diagnosis of PCOS requires at least two of the following

- Clinical or biochemical evidence of hyperandrogenism.
- Evidence of oligo- or anovulation.
- Presence of polycystic ovaries on ultrasound.

Multiple clinical trials have been conducted to assess which drug is the most appropriate in aiding fertility. An article published in the New England journal of Medicine entitled Clomiphene, Metformin, or Both for Infertility in the Polycystic Ovary Syndrome concluded that Clomiphene is superior to metformin in achieving live birth in infertile women with the polycystic ovary syndrome, although multiple birth is a complication(N Engl J Med 2007; 356:551-566 February 8, 2007).

Another article Status of clomiphene citrate and metformin for infertility in PCOS (Trends Endocrinol Metab. 2012 Oct;23(10):533-43) published the following results:

'Though widely used, there is uncertainty about the effectiveness and adverse effects of metformin and clomiphene citrate (CC) for infertility in polycystic ovary syndrome (PCOS). A systematic review (SR) of the best available evidence suggests that both CC and metformin are better than placebo for increasing ovulation and pregnancy rates, but CC is more effective than metformin for ovulation, pregnancy and live-birth rates, in PCOS patients with body mass index (BMI) >30.'

In PCOS, serum prolactin may also be marginally raised, but the levels seldom exceed 1500 mU/l.

Reverse circadian rhythm steroids are used in the treatment of congenital adrenal hyperplasia, whilst cabergoline is used for the medical management of hyperprolactinemia.

Spironolactone has antiandrogenic activity and can cause improvements in hirsutism in PCOS but has no bearing on fertility.

Question 122 of 249

A 56-year-old man is referred to clinic by his General Practitioner as his GP had performed some routine blood tests which showed a K+ of 2.8 mmol/l. The patient feels well in himself. His past medical history includes angina and renal stones. On examination his chest is clear and his abdomen is soft and non-tender.

Observations are as follows: temperature 36.3, blood pressure 132/86 mmHg, heart rate 78/min, respiratory rate 16/min, saturations 95% on air

His ECG shows normal sinus rhythm. Investigations are as follows:

Na+	142 mmol/l
K+	2.8 mmol/l
Creat	117 µmol/l
Urea	9.6 mmol/l
Urinary K+	26 mmol/l (normal <20)
PaO2	11.2 kPa
PaCO2	3.6 kPa
pH	7.32
HC03-	18 mmol/l
Base excess	-3 mmol/l

What is the most likely cause of his hypokalaemia?

A. Barter's Syndrome
B. Liddle's Syndrome
C. Renal tubular acidosis type 1
D. Renal tubular acidosis type 2
E. Renal tubular acidosis type 4

C. Renal tubular acidosis type 1

All of these diagnoses could cause hypokalaemia except renal tubular acidosis type 4 which causes a hyperkalaemia.

When faced with a patient with hypokalaemia first check the urinary K+, if this is low you should consider gastrointestinal losses such as diarrhoea or vomiting or decreased intake of K+. If the urinary K+ is high check the blood pressure, which is this case is normal. Liddle's Syndrome would cause hypertension.

If the patient is not hypertensive check the bicarbonate if it is low such as in this case the diagnosis is renal tubular acidosis. The clue here is that the patient has a history of renal stones meaning that the diagnosis is renal tubular acidosis type 1.

Renal tubular acidosis type 2 is associated with conditions such as Wilson's disease, lead poisoning and myeloma.

Question 123 of 249

A 78-year-old female attends the diabetes clinic. She has longstanding type 2 diabetes. Over the last few years she has become increasingly frail. Her main complaint is recurrent nausea and vomiting. Earlier this year she underwent endoscopy and gastric emptying studies which confirmed gastroparesis. She has since been started on metoclopramide which has had minimal effect on her symptoms. Her weight has decreased by 10% over the past year with a current BMI of 26 kg/m².

Her HbA1c today at the clinic is 44 IFCC mmol/l (6.2%) having been 60 IFCC mmol/l (7.6%) this time last year.

Her past medical history includes chronic kidney disease stage 3 and aortic stenosis.

Her current therapy is Humulin M3 22 units at breakfast and dinner, metformin 500mg BD, ramipril 5mg OD, bendroflumethiazide 2.5mg OD, aspirin 75 mg OD.

She lives alone and is still driving. She denies the need for carers, however, she has had 3 falls in the past month. She describes particular difficulty getting up in the morning and says her mood can often be low in the mornings.

She checks her blood sugar once daily in the morning with the following results.

Saturday	3.1 mmol/l
Sunday	4.0 mmol/l
Monday	4.1 mmol/l
Tuesday	3.2 mmol/l
Wednesday	14.6 mmol/l
Thursday	3.5 mmol/l
Friday	16.1 mmol/l

What is the correct step in the management of her diabetes?

A. Change Humulin M3 to a glucagon-like-peptide 1 receptor agonist
B. Stop metformin
C. Add sitagliptin
D. Change Humulin M3 to 20 units in the morning and 10 units inthe evening
E. Change Humulin M3 to 30 units once daily

D. Change Humulin M3 to 20 units in the morning and 10 units in the evening

This lady's fasting blood sugars are too low. The 2 high readings raise the concern of overnight hypoglycaemia with reflex hyperglycaemia in the morning. Her recent weight loss will mean her insulin resistance will have decreased (as reflected in her decreasing HbA1c) and she will require smaller doses of insulin. Changing her insulin from a total of 44 units daily to 30 units daily offers protection against hypoglycaemia. Furthermore altering her split of insulin to the conventional 2/3rd in the morning and 1/3rd in the evening reduces the risk of overnight hypos.

The risks of tight glycaemic control in this lady vastly outweigh any benefits. She should be advised to check her sugar more regularly for the short-term and given advice regarding driving. Close follow-up with the diabetic nurses should be arranged until her hypo risk is reduced.

There is no indication for a GLP1 agonist (BMI 26) and metformin should continue (assuming eGFR remains above 30) to allow the reduction in her insulin doses to lowest effective levels.

Question 124 of 249

A 35-year-old man presents to the Emergency Department with a 24-hour history of nausea, vomiting, and increasing lethargy. He has a known diagnosis of type 1 diabetes mellitus but reports non-adherence to his insulin therapy. On examination, he is dehydrated with dry mucous membranes, Kussmaul breathing, and weighs 63 kg. His vital signs are as follows:

Blood pressure: 70/45 mmHg Pulse: 116 bpm Respiratory rate: 26/min

Laboratory investigations reveal:

Hb	140 g/L	Male: (135-180)
Platelets	220 * 10 /L 9	(150 - 400)
WBC	14.5 * 10 /L 9	(4.0 - 11.0)
Random blood glucose	18 mmol/L	
pH	6.9	(7.35 - 7.45)
pCO2	2.6 kPa	(4.5-6.4)
Na +	140 mmol/L	(135 -145)
K +	4.1 mmol/L	(3.5-5.0)
Blood ketones	3 mmol/L	
Bicarbonate	9 mmol/L	(22-29)
Creatinine	105 µmol/L	(55-120)

What is the most appropriate management plan for this patient?

A. IV normal saline, 5.0 units/h insulin infusion and 40 mmol/L potassium
B. IV normal saline, 6.3 units/h insulin infusion and 40 mmol/L potassium
C. IV normal saline, 63 units/h insulin infusion and 40 mmol/Lpotassium
D. IV normal saline, IV antibiotics, 5.0 units/h insulin infusion and 40mmol/L potassium
E. IV normal saline, IV antibiotics, 6.3 units/h insulin infusion and 40mmol/L potassium

B. IV normal saline, 6.3 units/h insulin infusion and 40 mmol/L potassium

Diabetic ketoacidosis: the IV insulin infusion should be started at 0.1 unit/kg/hour

IV normal saline, 6.3 units/h insulin infusion and 40 mmol/L potassium is the correct answer. The clinical presentation is highly suggestive of diabetic ketoacidosis (DKA), characterised by metabolic acidosis, hyperglycaemia, and ketonaemia. Management involves intravenous normal saline for rehydration, insulin therapy at a rate of 6.3 units/h (based on the patient's body weight of 63 kg), and potassium replacement at 40 mmol/L because the patient's serum potassium is within the range of 3.5-5.5 mmol/L.

IV normal saline, 5.0 units/h insulin infusion, and 40 mmol/L potassium are incorrect because the appropriate insulin infusion rate should be calculated as 0.1 unit/kg/hour; for a patient weighing 63 kg, this equates to an infusion rate of 6.3 units/hour.

IV normal saline, 63 units/h insulin infusion, and 40 mmol/L potassium represents a significant overdose of insulin. The correct dosage should be based on the standard DKA management protocol of administering insulin at a dose of 0.1

unit/kg/hour, resulting in an infusion rate of 6.3 units/hour for this patient.

IV normal saline, IV antibiotics, 5.0 units/h insulin infusion and 40 mmol/L potassium is not the appropriate choice here as it underestimates the required rate of insulin infusion (should be set at 6.3 units/h instead). Moreover, leukocytosis can occur associated with hyperglycaemia and DKA; thus, it should not automatically prompt antibiotic administration without further evidence suggestive of infection.

IV normal saline, IV antibiotics, 6.3 units/h insulin infusion and 40 mmol/L potassium is incorrect due to the unnecessary inclusion of antibiotics in the treatment regimen. As previously stated, leukocytosis can be associated with both hyperglycaemia and DKA; therefore, it does not justify empirical antibiotic therapy without other clinical indications pointing towards an infectious process.

Question 125 of 249

A 50-year-old gentleman presents to review in the endocrine clinic. He was normally taking metformin and gliclazide, but over the last six months has also been prescribed a GLP-1 mimetic. He was started on a GLP-1 mimetic because of poor diabetic control and concerns about starting insulin. He does not want to start insulin because he is a truck driver and worried about losing his driving license. He had previously tried the combination of metformin, gliclazide and pioglitazone, but this had also failed to control his HbA1c.

Over the last six months his HbA1c has reduced from 81mmol/ mol to 80mmol/ mol, and he has lost two kg in weight.

What is the most appropriate action?

A. Continue current treatment and review in one year
B. Continue current treatment and review in six months
C. Change to metformin, gliclazide and pioglitazone
D. Offer to add on insulin
E. Stop GLP-1 mimetic and offer insulin treatment

E. Stop GLP-1 mimetic and offer insulin treatment

The correct answer is to stop GLP-1 mimetic and offer insulin treatment. This is a patient with diabetes whose diabetic control has not improved even when on a combination of metformin and two other drugs. He was then started on GLP-1 mimetic therapy, but nice advice that this should only be continued if after six months HbA1c reduces by at least 11 mmol/mol, which has not happened. Therefore continuing the current treatment is incorrect. Insulin should not be combined with a GLP-1 mimetic.

Question 126 of 249

An 82-year-old man is reviewed on the ward. He was admitted with recurrent falls four days ago. He has been found to have symptomatic postural hypotension with a drop of 50mmHg on standing in systolic blood pressure.He has a past medical history of hypertension, osteoporosis, radial fracture and COPD. He was taking ramipril and amlodipine, as well as inhalers and calcium supplements. He normally has a good functional baseline and is independent.

Despite stopping all antihypertensives on admission the physiotherapists are unable to mobilise him due to worsening of his symptoms on standing. At rest, he has a blood pressure of 150mmHg systolic.

What is the most appropriate plan?

A. Fludrocortisone
B. Hydrocortisone
C. Discharge bedbound and continue with physiotherapy at home
D. Discharge bedbound with restarting antihypertensives
E. Inpatient rehabilitation

A. Fludrocortisone

The correct answer is fludrocortisone. Fludrocortisone is a corticosteroid with minimal glucocorticoid activity but very high mineralocorticoid activity which can aid in symptomatic postural hypotension and can significantly reduce symptoms. It may take several days to have an effect but can restore mobility in some patients. Continuing with rehabilitation without addressing his treatable cause would not be optimal, whilst reintroducing his antihypertensives would reduce the risk of stroke and cardiovascular disease but would also reduce his independence significantly.

Question 127 of 249

A patient with type 1 diabetes mellitus is referred urgently from a Dose Adjustment For Normal Eating (DAFNE) course, a course designed to help patients with type 1 diabetes correctly assess carbohydrate contents, to the endocrinology consultant. The nurse in the course was concerned because she found that the patient has had three episodes of hypoglycaemia within the last nine months where he required aid from his wife in order to increase his blood glucose levels. He takes a basal bolus regime of long acting insulin once a night as well as short acting insulin three times a day. He works in a restaurant. He is an ex-smoker who drinks very little alcohol.

Apart from adjusting the insulin dose, what is the most appropriate action?

A. Advise the patient to inform the DVLA and to not drive
B. The consultant should inform the DVLA
C. Advise the patient to start checking blood glucose prior todriving
D. Advise the patient to always carry a snack when driving
E. Advise the patient to attend retinal screening prior to drivingagain

A. Advise the patient to inform the DVLA and to not drive

Patient with diabetes who have had two hypoglycaemic episodes requiring help needs to surrender their driving licence

This patient has had hypoglycaemic episodes needing help multiple times. If a patient needs help to correct the hypoglycaemic episode then it is termed severe hypoglycaemia and is of concern to the DVLA. If a patient has two or more episodes of severe hypoglycaemia then they need to inform the DVLA and not drive.

Patients who take insulin should have their licence for driving assessed on an annual basis. For this they will be required to submit three months

of blood glucose readings. Patients are advised to always check blood glucose before driving and to always have a snack in their vehicle as well, not just if having hypoglycaemic episodes.

When the DVLA is informed, the patient should be advised to inform the DVLA themselves rather than breaking patient confidentiality, but if the patient repeatedly fails to follow this advise then the doctor should inform the DVLA after telling the patient that he or she is doing so.

Question 128 of 249

A 45-year-old gentleman presents to clinic for review. Two weeks ago he presented to the emergency department with renal colic. A spiral CT KUB confirmed nephrolithiasis and he was managed conservatively with IV fluids, analgesia and an alpha-blocker. His symptoms resolved entirely and he was discharged.

Blood tests:

Hb	142 g/l
Platelets	329 * 10 /l9
WBC	6.6 * 10 /l9
Na +	141 mmol/l
K +	3.8 mmol/l
Urea	6.2 mmol/l
Creatinine	71 µmol/l
Corrected calcium	2.71 mmol/l
Parathyroid hormone	10.2 pmol/l (1.0-7.0 pmol/l)

How should he be further managed?

A. Annual monitoring of calcium and renal function
B. Encourage oral fluids
C. Bisphosphonates
D. Vitamin D supplementation
E. Parathyroidectomy

E. Parathyroidectomy

The correct answer is parathyroidectomy. This is a patient who has developed renal colic secondary to likely primary hyperparathyroidism, as is suggested by his hypercalcaemia and elevated parathyroid hormone. The mainstay of management of primary hyperparathyroidism is parathyroidectomy, but cases have to be appropriately identified as

surgical candidates. This patient developed renal stones as a likely complication and therefore would benefit from surgery. If the blood tests been an incidental finding, then monitoring and oral fluids both would have been more appropriate.

Question 129 of 249

A 44-year-old woman is admitted to hospital complaining of a swollen breast for three days. She is otherwise well, having no medical problems. She is diagnosed by the surgical team with a breast abscess, which is drained and she is started on antibiotic treatment. Before being discharged, she is found to have elevated corrected calcium (2.79 mmol/L) and elevated parathyroid hormone (9.5 pmol/L).

She is reviewed by the endocrine team. She does not have any symptoms apart from those related to her breast abscess, and additional examination is unremarkable. Further tests are requested, showing that vitamin D levels are normal, 24-hour urine calcium is normal, and a DEXA scan is normal as well. She is advised to see her GP for annual blood tests for calcium levels and renal function.

She is diagnosed with primary hyperparathyroidism.

What additional investigation should be used to monitor her?

A. 24-hour urine calcium annually
B. Breast ultrasound annually
C. Abdominal X-ray annually
D. Abdominal ultrasound every three years
E. DEXA scan every one to two years

E. DEXA scan every one to two years

The correct answer is a DEXA scan. This patient has been incidentally found to have primary hyperparathyroidism and has no evidence of indications for parathyroidectomy. Monitoring should include renal function and DEXA scanning to identify any decline in renal function, worsening hypercalcaemia or osteoporosis. Any of these changes would be indications for surgery. Abdominal X-rays and ultrasound scanning may be useful in the acute setting to detect renal stones but are not recommended as monitoring. Urinary calcium useful at diagnosis to exclude hypocalciuric hypercalcaemia.

Question 130 of 249

A 68-year-old Indian patient presents to the emergency department with facial tetany, muscle cramps and paraesthesia of her fingers and toes. This is her second admission with similar symptoms. Her past medical history includes diffuse cutaneous systemic sclerosis with gastrointestinal, cutaneous and pulmonary manifestations. She was also diagnosed with vitamin D deficiency two years ago and receives regular vitamin D supplements. Her blood tests are as follows:

Hb	124 g/l
WBC	8.0 * 10 /l9
Na +	141 mmol/l
K +	4.3 mmol/l
Urea	6.5 mmol/l
Creatinine	90 µmol/l
CRP	15 mg/l
Corrected calcium	1.68 mmol/l
Phosphate	1.4 mmol/l
Magnesium	0.28 mmol/l
PTH	2 pmol/L (normal range = 8.5-12)
Amylase	14 u/l

Her symptoms improve with intravenous calcium replacement and intravenous magnesium replacement, correcting both electrolytes to within normal range.

What is the underlying cause for these metabolic disturbances in this patient?

A. Hypomagnesaemia
B. Primary hypoparathyroidism

C. Insufficient vitamin D supplementation
D. Chronic kidney failure
E. Chronic pancreatitis

A. Hypomagnesaemia

Magnesium deficiency causes hypocalcaemia

This complex picture investigates the underlying cause of hypomagnesaemia and hypocalcaemia in a patient with significant GI disease. With regular vitamin D supplementation, it is unlikely this is the cause. Her renal function is also within normal range. Although her parathyroid hormone levels are low, the likely underlying cause is due to insufficient magnesium absorption due to GI systemic sclerosis, which results in reduces parathyroid hormone release. There is nothing in the history to suggest a primary hypoparathyroidism or chronic pancreatitis.

Question 131 of 249

A 37-year-old female presents with 4 days of generally unwell and a recent dysuria. Her urine is foul smelling and dark. She is a known type 1 diabetic with a long-standing subcutaneous insulin regime. Her pH on admission was 7.24, bicarbonate 8 mmol/l and blood glucose 32 mmol/l. Urinary dip leucocytes 2+, nitrites2+ and 4+ ketones. She was started on treatment for diabetic ketoacidosis with intravenous fluids and fixed rate insulin. She also has intravenous antibiotics for a urinary source of sepsis. You are asked to review her blood sugars at 4 hours after treatment was initiated.

What should be the aim in managing hyperglycaemia in a diabetic ketoacidosis patient?

A. Reduce blood glucose to under 14 mmol/l as quickly as possible
B. Reduce blood glucose by 3mmol/l per hour
C. Reduce blood glucose by 6 mmol/l per hour
D. Aim blood glucose above 18 mmol/l
E. Blood glucose does not require monitoring if insulin infusion isrunning

B. Reduce blood glucose by 3mmol/l per hour

The most recent guidelines by the Joint British Diabetes Societies Inpatient Care Group in September 2012 recommends a reduction in blood glucose of 3 mmol/l per hour until BM reaches 14 mmol/l, at which point 5% dextrose should be considered as the intravenous fluid of choice. Rapid glucose lowering should be avoided: the rapid flux in osmolality can result in significant cerebral oedema and resultant cerebral damage.

Question 132 of 249

A 16-year-old male presents with 3 months of chronic headaches and visual blurring. He has no past medical history and no known family history. On examination, his heart sounds are normal with no added sounds and the respiratory examination is unremarkable. He has no focal neurological signs. Fundoscopy reveals papilloedema, hard exudates and flame haemorrhage. His blood pressure is 226/160mmHg. His blood tests and arterial blood gas are as follows:

Na +	145 mmol/l
K +	2.9 mmol/l
Urea	5.4 mmol/l
Creatinine	72 µmol/l
pH	7.49
PaO2	13kPa
PaCO2	3.4 kPa
Bicarbonate	34 mmol/L
Serum ambulatory renin activity	0.2 pmol/L @ 3-4 hours (normal range 0.8-3.5 pmol/ml/hr)
Serum ambulatory aldosterone	24 pmol/L@ 3-4 hours (normal range 100-800)

What is the optimal long-term treatment?

A. Amlodipine
B. Ramipril
C. Atenolol
D. Doxazosin
E. Amiloride

E. Amiloride

The patient is young and presents with grade 4 hypertensive retinopathic changes associated with a systolic of over 200mmHg, associated with hypokalaemia and a metabolic alkalosis. Importantly, both renin and aldosterone are decreased, ruling out primary hyperaldosteronism. The diagnosis is thus likely to be Liddle's syndrome, a genetic disorder of ENaC channels in the collecting duct, leading to increased sodium reabsorption and increased potassium excretion. The treatment is with amiloride, a potassium sparing diuretic that directly blocks collecting tubule sodium channels and resolves hypertension.

Question 133 of 249

A 28-year-old pregnant lady presents to the Emergency Department with palpitations and sweating. She mentions that she has had these symptoms on and off for the past 4 months but that they have worsened over the past few weeks. Now she is feeling worried and wanted to be assessed medically due to her concern she was having a miscarriage. She looks particularly anxious to be in hospital. This is her first pregnancy. She is 7 weeks pregnant. She has had no vaginal bleeding or discharge during the course of her pregnancy. She is normally fit and well.

Initial observations reveal a blood pressure of 130/85 mmHg, a heart rate of 110 beats per minute, a respiratory rate of 19/min, oxygen saturations of 99% on air and a temperature of 37.5ºC. Examination findings reveal a resting tachycardia and a subtle goitre is noted.

Blood test results are as follows:

Hb	110 g/l
Wcc	12 x10^9/l
Plt	245 x10^9/l
CRP	12 mg/l
Na+	140 mmol/l
K+	5.0 mmol/l
Ur	5.7 mmol/l
Cr	110 µmol/l
D-dimer	490 ng/ml
T4	21 mU/l

TSH	<0.05 pmol/l

Given the most likely diagnosis, how should this lady be managed?

A. Watch and wait/symptomatic control with beta blockade
B. Radioactive iodine therapy
C. Subtotal thyroidectomy
D. Propylthiouracil
E. Block and replace carbimazole + thyroxine

D. Propylthiouracil

This lady has symptoms and biochemical evidence of hyperthyroidism. This lady's symptoms predate her pregnancy, therefore it is not pregnancy induced thyrotoxicosis and will not self-limit - she will need treatment to prevent complications to her and the foetus. Radioactive iodine is contraindicated. Subtotal thyroidectomy is a little risky and extreme during pregnancy. Carbimazole, whilst normally first line, has been associated with neonatal aplasia cutis before 12 weeks gestation and is therefore usually avoided. This leaves propylthiouracil as the current safest option. During the second trimester, propylthiouracil should be changed to carbimazole due to the potential risk of hepatotoxicity with propylthiouracil. The lowest dose that controls the hyperthyroid state should be used as both medications can cross the placenta.

Question 134 of 249

A 75-year-old man with a history of high blood pressure, type 2 diabetes and hypercholesterolaemia was admitted to the emergency department with confusion. His daughter states that this has come on slowly over the last week and prior to this he had no memory problems. He currently takes metformin, ramipril, amlodipine and atorvastatin.

On examination, he smells strongly of urine and his mucous membranes appear dry. His abbreviated mental test score is 7 out of 10 and he is oriented in person but not in place or time. His heart rate is 95 per minute and his blood pressure is 105/62 mmHg. His chest is clear and has a soft ejection systolic murmur which does not radiate. His jugular venous pressure is not visible and he has mild ankle oedema. He has diffuse tenderness in the lower abdomen with no peritonism and normal bowel sounds. He has no focal neurology.

Investigation results are as follows: Chest x-ray: Clear lung fields. Urine dip:

Glucose	+++
Blood	+
Protein	+
Leucocytes	+
Nitrites	+
Ketones	+

Venous blood gas:

pH	7.43
BE	- 1.5 mmol/l
HCO3	23 mmol/l
Glucose	34 mmol/l
Lactate	2.5 mmol/l

Full blood count:

Hb	120 g/l
Platelets	445 * 10 /l9
WBC	13 * 10 /l9

Renal function:

Na +	151 mmol/l
K +	5 mmol/l
Urea	10 mmol/l
Creatinine	137 µmol/l
Glucose	32 mmol/l
Ketones	2 mmol/l

Which would be the most appropriate initial resuscitation measure?

A. 0.45% saline
B. 0.9% saline
C. Fixed rate insulin and 0.9% saline
D. Hartmann's
E. Sliding scale insulin and 0.9% saline

B. 0.9% saline

This gentleman has hyperosmolar hyperglycaemic state (HHS), likely precipitated by urinary tract infection and his pre-existing diabetes.

According to the Joint British Diabetes Society Guidelines for HHS, 0.9% saline is the recommended initial resuscitation fluid, aiming for 3-6 litres positive at 12 hours. This should only be switched to 0.45% saline if osmolality is not declining despite positive fluid balance. Fixed rate insulin should only be added if glucose fails to fall with fluid.

Question 135 of 249

A 53 year old gentleman attends his General Practitioner for a 'Well Man Check'. He has a past medical history of hypertension which has been treated with ramipril for 4 years. As part of the screening the GP notes that the patient has been suffering from low back pain for the last couple of months. He has been taking paracetamol and ibuprofen that he has bought over the counter and this has eased his pain. His blood pressure today is 134/76 mmHg. His GP takes blood tests as part of the check and the results are shown below.

Hb	13.2 g/dl
Platelets	312 * 10 /l9
WBC	8.2 * 10 /l9

Na +	138 mmol/l
K +	6.6 mmol/l
Urea	6.2 mmol/l
Creatinine	114 µmol/l

His GP notifies the patient immediately on seeing these results and refers him to the local Medical Assessment Unit. Where more tests are carried out.

Arterial Blood Gases:

pH 7.34

PaCO2 5.1kPa PaO2 12kPa HCO3- 20 mmol/l

Serum Chloride 120mmol/l

Urinalysis:

pH 4.8

Protein negative Blood negative

Leukocytes negative Glucose negative

What is the most likely diagnosis?

A. Renal Tubular Acidosis type 1
B. Renal Tubular Acidosis type 2
C. Renal Tubular Acidosis type 3
D. Renal Tubular Acidosis type 4
E. NSAID induced nephropathy

D. Renal Tubular Acidosis type 4

This patient has RTA type 4. This often occurs asymptomatically as in this case. In this case it's aetiology is likely secondary to ibuprofen use which has caused an aldesterone resistance in the proximal tubule of the nephron. It is associated with a mild metabolic acidosis and classically presents in patients with a high serum potassium. Urinary pH is commonly normal, but given that the nephron has not lost its hydrogen ion buffering capacity there is scope for the urinary pH to become more acidic to buffer the metabolic acidosis. The serum chloride level is high in this patient, with hyperchloraemia being a cardinal feature of all subtypes of RTA. When calculated, the anion gap in RTA type 4 is normal.

In NSAID induced nephropathy the urinalysis would show white cells (leukocytes) and may even show proteinuria.

Question 1 of 114

A 55 year-old female presents to the outpatients department having been referred by her GP. She complains of fatigue, increased sweating and weight loss over the past four months. She also reports a loss of sex drive.

Examination reveals that she is pale and has a pulse rate of 121 per minute with a bounding pulse character. Her blood pressure is 118/79 mmHg and she has heart sounds 1 and 2 presents with no added sounds. On auscultation, her chest is clear and her abdomen is soft and non-tender with no organomegaly. She has a smooth goitre but has no signs of thyroid eye disease. Examination of her cranial nerves are normal.

The results of recent blood tests are as follows:

Hb	11.3 g/dl
Platelets	190 * 10 /l9
WBC	10.9 * 10 /l9
Na +	129 mmol/l
K +	4.3 mmol/l
Urea	7.9 mmol/l
Creatinine	94 µmol/l
ALP	155 u/l
Calcium	2.40 mmol/l
Albumin	40 g/L
TSH	11 mU/L
Free T4	41 pmol/L
Free T3	11 pmol/L

Which of the following is the most likely diagnosis?

A. Grave's disease
B. Thyroid cancer
C. Surreptitious thyroxine ingestion
D. De Quervain's thyroiditis
E. TSH secreting pituitary tumour

E. TSH secreting pituitary tumour

Biochemistry reveals elevated thyroid-stimulating hormone (TSH) with concurrent elevated thyroxine (T4) and tri-iodothyronine (T3). An elevated alkaline phosphatase (ALP) is consistent with thyrotoxicosis. Hyponatraemia suggests hypoadrenalism.

Taken with the symptoms, this patient has a likely diagnosis of a thyrotropinoma, which is a rare type of pituitary tumour accounting for approximately less than 1% of cases of pituitary tumours. 90% are macroadenomas.

Presentation is typically with features of thyrotoxicosis and include weight loss, sweating, fatigue and tachycardia. There may also be signs of hypopituitarism.

Question 2 of 114

A 24-year-old nurse presents after collapsing on a night shift. His blood glucose is measured at being 1.4 mmol/l. His blood pressure at the time was noted to be 115/82 mmHg. He has no palpitations and had not bitten his tongue or become incontinent during the episodes. He was shaken afterwards, although did not have memory loss and stated he had not tripped over anything. He also said he has had five of these episodes over the last two weeks.

Blood tests are sent off and unremarkable except for a low-normal Cpeptide level and markedly raised insulin level.

Which of the following is the most likely diagnosis of his multiple episodes of collapse?

A. Sulphonylurea misuse
B. Insulin misuse
C. Alcohol misuse
D. Retroperitoneal sarcoma
E. Insulinoma

B. Insulin misuse

Hyperinsulinaemia in the absence of raised C-peptide points towards the diagnosis of insulin abuse. Elevation of C-peptide, when combined with hyperinsulinaemia suggests sulphonylurea abuse. To rule this out it may be appropriate to assay levels of commonly used sulphonylureas in urine. Insulinomas are a more rare cause of repeated hypoglycaemic episodes.

Question 3 of 114

A 42-year-old man is referred from his GP to the outpatient department with a loss of libido and impotence. He has a stressful job as a pharmaceutical representative and has a five-year pack year history. He consumes four pints of beer per night.

Blood tests reveal a prolactin of 3340 mU/l (<360), normal thyroid function tests, MCV 100 fl, testosterone low.

Which of the following would be the most appropriate investigation?

A. Visual field testing
B. Urine cortisol
C. Pituitary dynamic function tests
D. MRI pituitary
E. Anterior pituitary function testing

D. MRI pituitary

This patient has a prolactin result of over 3340 mU/l suggesting the most likely diagnosis is a prolactinoma. As the result is below 6000 mU/l would be suggestive of microprolactinoma as opposed to a macroprolactinoma. The most appropriate investigation would therefore be an MRI of the pituitary. On a side note, his raised MCV reinforces his history that he consumes too much alcohol.

Question 4 of 114

A 58-year-old patient who has a history of hypertension is operated on by the neurosurgeons for an intracranial haemorrhage.

Over the next few days his serum sodium level progressively declines and by the third day has fallen to 118 mmol/l despite fluid restriction to 1L per day. Urine osmolarity is 700 mOsmo/l and urinary sodium is raised at 80 mmol/l.

What is the most likely diagnosis?

A. Addisonian crisis
B. Secretion of inappropriate antidiuretic hormone
C. Cranial diabetes insipidus
D. Hypovolaemia
E. Fluid overload

B. Secretion of inappropriate antidiuretic hormone

The hyponatraemia and hypotonic blood plasma, coupled with the raised urine osmolality and raised urinary sodium excretion indicates a diagnosis of syndrome of inappropriate ADH secretion. This is a condition that can occur after head trauma, a central nervous system infection and intracranial haemorrhage.

Question 5 of 114

A 32 year-old man is referred by his GP after collapsing while at work. He does not remember the episode but witnesses say that there was no incontinence or fitting and the patient does not have a sore mouth or tongue. This is the first time this has happened and the patient does not have any other past medical history of note and takes no regular medication.

Examination reveals a blood pressure of 162/95 mmHg, a pulse of 74 beats per minute, a respiratory rate of 16 and a temperature of 37.4ºC. Heart sounds 1 and 2 are present with no added sounds, the lung fields are clear and his abdomen is soft and non-tender.

Blood tests performed and reveal:

Na +	143 mmol/l
K +	3.0 mmol/l
Urea	5.6 mmol/l
Creatinine	76 µmol/l
Bicarbonate	31 mmol/l
Renin	low
Aldosterone	low

Which of the following is the best treatment?

A. Amiloride
B. Bumetanide
C. Spironolactone
D. ACE inhibitor
E. Angiotensin II receptor blocker

A. Amiloride

This man has Liddle's syndrome, an autosomal dominant disorder characterised by hypertension associated with hypokalaemic metabolic alkalosis, low plasma renin activity, and suppressed aldosterone secretion. Amiloride is the best treatment for the hypertension and hypokalaemia as it acts on the sodium channels directly, as opposed to spironolactone, which acts on mineralocorticoid receptors.

Question 6 of 114

A 29-year-old woman is referred by her GP to the outpatient department with increasing symptoms of heat intolerance, diarrhoea and anxiety over the past couple of weeks. The patient is 34 weeks pregnant with her first baby and has a past medical history of hyperthyroidism, currently being treated with 10mg carbimazole. She has no other past medical history of note and her mother also had hyperthyroidism. She does not smoke or drink alcohol and does not take any recreational drugs.

On examination, her pulse is 98 beats per minute, blood pressure is 124/82 mmHg and her respiratory rate is 14/min. Her oxygen saturation is 98% and temperature is 37.5ºC.

Blood tests are performed and reveal:

TSH	0.04 mu/l
Free T4	21 pmol/l
Total thyroxine (T4)	152 nmol/l

What is the most appropriate management?

A. Refer patient for immediate caesarean section
B. Increase carbimazole dose to 20mg once daily
C. Commence radioiodine treatment
D. Switch carbimazole to propylthiouracil
E. Refer for a thyroidectomy

B. Increase carbimazole dose to 20mg once daily

The carbimazole can be increased to up to 20mg once daily during pregnancy. Propylthiouracil can be started instead of the carbimazole if the increased carbimazole dose does not adequately control the patients hyperthyroidism.

Question 7 of 114

A 43-year-old man is referred by his GP with a 4 week history of a lump appearing on the right side of his neck. The lump is roughly 7 mm and is located on the right side of the thyroid gland, in the anterior triangle. It does not move when the patient sticks out his tongue, but it does move on swallowing. There is no history of weight loss of night sweats.

Blood tests are performed and reveal:

Hb	12.9 g/l
Platelets	210 * 10 /l9
WBC	6.0 * 10 /l9
Na +	141 mmol/l
K +	3.9 mmol/l
Urea	4.1 mmol/l
Creatinine	33 µmol/l

What is the most appropriate first-line investigation?

- A. Radioisotope scan of thyroid
- B. Magnetic resonance scan of head and neck
- C. Excision biopsy
- D. Fine needle aspiration biopsy
- E. Ultrasound scan of thyroid

E. Ultrasound scan of thyroid

High-resolution ultrasound scanning is an ideal first-line initial imaging investigation for most neck lumps. Because most lesions in the neck are site-specific, once a lesion has been located, specific ultrasound features can be used to establish the diagnosis.

Question 8 of 114

A 19-year-old woman presents to her GP with a 7 month history of weight loss, diarrhoea and palpitations. The diarrhoea is normal colour and over the last three months she has had roughly 2-3 bowel motions per day. The heart palpitations occur randomly throughout the day and night. She has also noticed that she has recently been getting episodes of feeling very hot and sweaty. She has no other past medical history and her only family history is a mother who has Hashimotos thyroiditis.

On examination, the patient is sweaty and her blood pressure is 130/80 mmHg, pulse is 102 bpm and regular, respiratory rate is 16/min and her oxygen SATs are 98% on air.

Blood tests are performed and reveal:

Hb	135 g/l
Platelets	220 * 10 /l9
WBC	7.1 * 10 /l9
Na +	139 mmol/l
K +	3.9 mmol/l
Urea	5.1 mmol/l
Creatinine	60 µmol/l
Free T4	28 pmol/l
TSH	0.08 mu/l

A thyroid radioisotope scan is performed and reveals a globally reduced uptake.

What is the most likely diagnosis?

- A. Graves disease
- B. Thyrotoxicosis factitia
- C. Hashimotos disease
- D. De Quervains thyroiditis
- E. Atrophic thyroiditis

B. Thyrotoxicosis factitia

The most like likely diagnosis in this case is thyrotoxicosis factitia. This is evidenced by the reduced thyroid uptake on radioisotope scanning, along with the fact she may have easy access to thyroxine he mother would be taking it for her Hashimotos thyroiditis. De Quervains thyroiditis can present with symptoms similar to factitious thyroiditis and a decreased uptake on radioisotope scan, however, the fact the symptom shave lasted for 7 months makes this diagnosis unlikely.

Question 9 of 114

A 39-year-old woman is referred to the outpatient department by her GP having been unsuccessfully treated with trimethoprim and nitrofurantoin for recurrent urinary tract infections. These have been occurring over the past six months. Over the last 2 days, the patient reports that urinating has become very painful and that the patient is now having difficulty urinating. The patient has a past medical history of type 2 diabetes mellitus. After completing a course of amoxicillin, the patient still complains of pain on urinating and pain in the lower abdomen. There is a family history of type 2 diabetes mellitus and the patient has a smoking history of 5 pack years, while drinking on average 15 units per week. An HbA1c test is performed and reveals a result of 82 mmol/mol.

What is the most likely organism causing the recurrent urinary tract infections?

- A. Candida
- B. Neisseria gonorrhoea
- C. Chlamydia trachomatis
- D. Trichomonas vaginalis
- E. Mycoplasma genitalium

A. Candida

The most likely organism in this scenario is candida - in those individuals who do have symptomatic Candida infections, the symptoms are indistinguishable from those caused by bacterial infections. Cystitis is associated with dysuria, urgency, suprapubic discomfort. This patients recent symptoms of oliguria, difficultly passing urine and painful urination suggest a complication such as the presence of a fungus ball.

Question 10 of 114

A 24-year-old female presents to the clinic with worsening of fatigue. She states that she has been feeling tired for months. On systematic enquiry she complains of constipation, cold intolerance and dry skin. She has no past medical history of note and is not on any regular medicines. On examination you note that her lying blood pressure is 110/75 mmHg which falls to 95/60 mmHg on standing.

Investigation results are as follows:

Hb	110 g/l	Na +	131 mmol/l	Fasting glucose	7.5 mmol/l
Platelets	425 * 10 /l 9	K +	5.1 mmol/l	Bicarbonate	19 mmol/l
WBC	10.1 * 10 /l 9	Urea	10.5 mmol/l	Creatinine	110 µmol/l

What is the most likely diagnosis?

- A. Autoimmune polyendocrinopathy syndrome (APS) - type 1
- B. Autoimmune polyendocrinopathy syndrome (APS) - type 2
- C. Addison's disease
- D. Type 1 diabetes mellitus
- E. Type 2 diabetes mellitus

B. Autoimmune polyendocrinopathy syndrome (APS) - type 2

APS type 2 patients have Addison's disease plus either T1DM or autoimmune thyroid disease

A fasting glucose of 7.5 mmol/l suggests a diagnosis of diabetes. In the absence of diabetic symptoms (e.g. polyuria, and polydipsia), this would need to be repeated to confirm the diagnosis. Alternatively, other tests such as HbA1c, or an oral glucose tolerance test could be used.

The clinical features of constipation, cold intolerance and dry skin are suggestive of hypothyroidism. Thyroid function tests should be performed.

The biochemistry (hyponatraemia, hyperkalemia, and acidosis) and postural hypotension are suggestive of hypoaldosteronism.

The unifying diagnosis is autoimmune polyendocrinopathy syndrome (APS) type 2. APS type 2 patients have Addison's disease plus either T1DM and/or autoimmune thyroid disease.

Question 11 of 114

A 55-year-old woman attends the emergency department with abdominal pain. She complains of right-sided loin pain which radiates to her groin. She is otherwise well. She has a past medical history of Sjogren's syndrome. On examination, you note right-sided renal angle tenderness.

Blood results are as follows:

Hb	126 g/L	Male: (135-180)Female: (115 - 160)
Platelets	245 * 10 /L 9	(150 - 400)
WBC	6.2 * 10 /L 9	(4.0 - 11.0)
Na +	136 mmol/L	(135 - 145)
K +	2.5 mmol/L	(3.5 - 5.0)
Cl -	120 mmol/L	(96 - 106)
Urea	6.9 mmol/L	(2.0 - 7.0)
Creatinine	56 µmol/L	(55 - 120)
Bicarbonate	14 mmol/L	(23 - 29)
Glucose	22.5 mmol/L	(4 - 7)
CRP	2 mg/L	(< 5)

What is the most likely diagnosis?

- A. Addison's disease
- B. Diabetic ketoacidosis
- C. Type 1 renal tubular acidosis
- D. Type 2 renal tubular acidosis
- E. Type 4 renal tubular acidosis

C. Type 1 renal tubular acidosis

Type 1 (**distal**) renal tubular acidosis may be caused by Sjogren's syndrome

The patient has a metabolic acidosis as indicated by the low serum bicarbonate. The anion gap (AG) is a derived variable primarily used for the evaluation of metabolic acidosis to determine the presence of unmeasured anions. The normal anion gap varies with different assays but is typically 4 to 12 mmol/L.

The anion gap = (Na + K) - (Cl + HCO3) = (136 + 2.5) - (120 + 14) = 4.5. Thus the patient has a normal anion gap metabolic acidosis (NAGMA).

Causes of NAGMA include ('ABCD'):

- Addison's
- Bicarbonate loss: GI (e.g. diarrhoea) or renal (e.g. renal tubular acidosis)
- Chloride excess
- Diuretics (e.g. acetazolamide)

Causes of high anion gap metabolic acidosis (HAGMA) include:

- Lactate
- Toxins (e.g. methanol, paracetamol, propylene glycol) Ketones
- Renal failure

Type 1 renal tubular acidosis is correct. The presence of kidney stones, hypokalemia, NAGMA, and a past medical history of Sjogren's favour the diagnosis of type 1 RTA.

Addison's disease is incorrect. Although this can also cause a NAGMA, hyperkalemia would be expected as a consequence of hypoaldosteronism. Furthermore, patients with Addison's will often be hypoglycaemic.

Diabetic ketoacidosis (DKA) is incorrect. Although the patient is significantly hyperglycaemia and likely has undiagnosed diabetes mellitus, the presence of ketones would result in a HAGMA.

Type 2 renal tubular acidosis is incorrect. This can also present with

hypokalemia and NAGMA, however, there is not an association with Sjogren's making this a less likely diagnosis.

Type 4 renal tubular acidosis is incorrect. Although diabetes is a cause of type 4 RTA, this condition would be associated with hyperkalemia in contrast to the hypokalemia demonstrated in this case.

Question 12 of 114

A 28-year-old woman is referred to the hospital with dysphagia. She complains of fatigue and that she often struggles to swallow certain foods such as bread. Drinking has not been a problem.

In addition, her eyes often feel gritty and uncomfortable. She has mild myalgia which interferes with her job at the local bakery.

What other finding would be consistent with the most likely diagnosis?

A. C-ANCA positive
B. P-ANCA positive
C. Type 1 renal tubular acidosis
D. Type 2 renal tubular acidosis
E. Type 4 renal tubular acidosis

C. Type 1 renal tubular acidosis

Type 1 (**distal**) renal tubular acidosis may be caused by Sjogren's syndrome

This patient has features consistent with Sjogren's syndrome. The xerostomia (dry mouth) can lead to difficulty swallowing, particularly foods needing more saliva. The other characteristic feature is xerophthalmia (dry eyes) which can make the eyes feel gritty and uncomfortable. Sjogren's syndrome is associated with **type 1 renal tubular acidosis**.

Type 1 (distal) renal tubular acidosis occurs due to an inability to excrete H+ in the distal tubule. The resulting acidosis is usually accompanied by hypokalaemia. Complications include nephrocalcinosis and renal stones. Causes include idiopathic, rheumatoid arthritis, SLE, Sjogren's, amphotericin B toxicity, and analgesic nephropathy.

A sometimes helpful reminder is that type 1 renal tubular acidosis is largely associated with structural/rheumatological problems, whereas, **type 2 renal tubular acidosis** is associated with metabolic disorders such as Fanconi syndrome, Wilson's disease, and cystinosis.

Type 4 renal tubular acidosis is associated with hypoaldosteronism and diabetes, rather than Sjogren's. Type 4 is associated with hyperkalaemia, whilst types 1 and 2 are associated with hypokalaemia.

Sjogren's is typically associated with elevated levels of anti-Ro and antiLa. Neither **cANCA** nor **pANCA** are typically associated. ANCA antibodies are commonly associated with vasculitides. cANCA (cytoplasmic-ANCA) is associated with granulomatosis with polyangiitis. pANCA is found in microscopic polyangiitis and eosinophilic granulomatosis with polyangiitis.

Question 13 of 114

You review a 68-year-old patient in the diabetic clinic. He was diagnosed 28 years ago with type 2 diabetes and over this time has been through a number of antiglycemic agents including biguanides, sulfonylureas, thiazolidinediones and insulin. He is generally well but reports painless macroscopic haematuria and would like to be referred to a urologist as he has read about bladder cancer associated with one of his medications.

Which of the following antiglycemic agents can cause bladder cancer?

A. Gliclazide
B. Tolbutamide
C. Pioglitazone
D. Insulin detemir
E. Sitagliptin

C. Pioglitazone

Thiazolidinediones are associated with an increased risk of bladder cancer

Answer: Pioglitazone

Pioglitazone has been associated with an increased risk of bladder cancer. The greatest risk was shown in those patients who have used pioglitazone long term.

Risk of Bladder Cancer Among Diabetic Patients Treated With Pioglitazone Interim report of a longitudinal cohort study http://care.diabetesjournals.org/content/34/4/916

Question 14 of 114

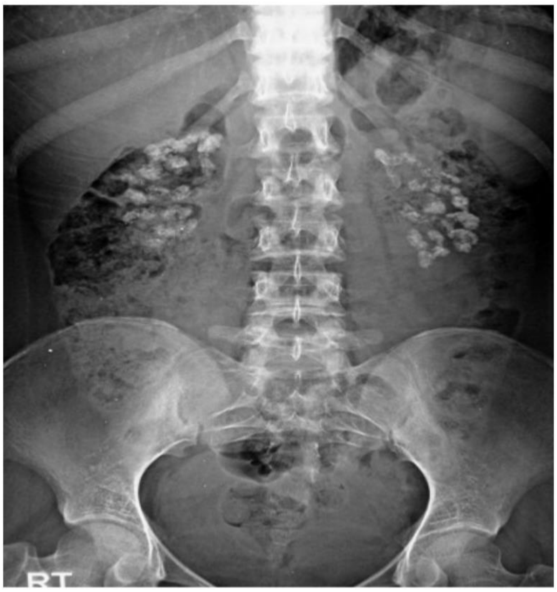
A 45-year-old woman presents to the Emergency Department with abdominal pain. Her GP is currently investigating her for lethargy, weakness and abdominal pain. Her symptoms have been getting progressively worse over the past few months. There is no past medical history of note. She smokes 5-10 cigarettes/day and drinks around 20 units of alcohol per week.

A urine dipstick has already been performed: protein trace, blood +, pH 5.5-6.0

Bloods show the following:

Hb	13.6 g/dl	Na$^+$	143 mmol/l
Platelets	225 * 10^9/l	K$^+$	2.3 mmol/l
WBC	8.4 * 10^9/l	Urea	6.1 mmol/l
Neuts	6.0 * 10^9/l	Creatinine	81 µmol/l
Lymphs	1.9 * 10^9/l	Bicarbonate	7 mmol/l
Eosin	0.3 * 10^9/l	Chloride	124 mmol/l

An abdominal film is requested due to her recurrent abdominal pains:



What is the most likely diagnosis?

A. Renal tubular acidosis type 1
B. Renal tubular acidosis type 2

C. Renal tubular acidosis type 4
D. Conn's syndrome
E. Bulimia

A. Renal tubular acidosis type 1

Hypokalaemia, nephrocalcinosis - type 1 renal tubular acidosis

This is probably a useful 'spot' diagnosis to learn for the exam: nephrocalcinosis on AXR → renal tubular acidosis (RTA) type 1. As well as nephrocalcinosis, the other pointers to RTA type 1 include hypokalaemia and a normal anion gap or hyperchloraemic metabolic acidosis.

If you weren't aware of the link between RTA type 1 and nephrocalcinosis another approach would be to work through the causes of 1. metabolic acidosis and 2. hypokalaemia.

1. Metabolic acidosis

It is useful to first calculate the anion gap:

Anion gap = (sodium + potassium) - (bicarbonate + chloride) = (143 + 2.3) - (7 + 124) = 12.3 mmol/l

A normal anion gap is 8-14 mmol/l

Causes of a normal anion gap or hyperchloraemic metabolic acidosis	Causes of a raised anion gap metabolic acidosis
gastrointestinal bicarbonate loss: diarrhoea, ureterosigmoidostomy, fistularenal tubular acidosisdrugs: e.g. acetazolamide ammonium chloride injection Addison's disease	lactate: shock, hypoxiaketones: diabetic ketoacidosis, alcoholurate: renal failure acid poisoning: salicylates, methanol

2. Hypokalaemia

Potassium and hydrogen can be thought of as competitors. Hyperkalaemia tends to be associated with acidosis because as potassium levels rise fewer hydrogen ions can enter the cells

Hypokalaemia with alkalosis	Hypokalaemia with acidosis
vomiting diuretics	diarrhoea
Cushing's syndrome Conn's syndrome (primary hyperaldosteronism)	renal tubular acidosis (types 1 and 2)acetazolamidepartially treated diabetic ketoacidosis

Question 15 of 114

A 23-year-old man is diagnosed as having type 1 diabetes mellitus after presenting with diabetic ketoacidosis. His blood sugars are now stable and he is well.

What is the first-line insulin regime he should be offered?

A. Twice-daily mixed insulin
B. Once-daily mixed insulin
C. Basal-bolus insulin regimen with twice-daily insulin detemir
D. Basal-bolus insulin regimen with once-daily insulin glargine
E. Rapid-acting insulin analogue before each meal with no longeracting insulin

C. Basal-bolus insulin regimen with twice-daily insulin detemir

In newly diagnosed adults with type 1 diabetes, the first-line insulin regime should be a basal-bolus using twice-daily insulin detemir

Question 16 of 114

A 62-year-old taxi driver is reviewed in the diabetes clinic some 6 weeks after suffering from an inferior myocardial infarction, for which he was stented. Current medication for control of blood sugar is metformin 1g twice a day. Other medication of note includes ramipril and indapamide. His blood pressure is 139/85 mmHg, his pulse is 84 beats per minute and regular. There are bilateral basal crackles on auscultation of the chest consistent with cardiac failure.

Na +	140 mmol/l
K +	4.5 mmol/l
Urea	7.2 mmol/l
Creatinine	112 µmol/l
HbA1c	64 mmol/mol

Which of the following is the most appropriate intervention with respect to his glycaemic control?

A. Add empagliflozin
B. Add gliclazide
C. Add insulin glargine
D. Add pioglitazone
E. Add saxagliptin

A. Add empagliflozin

SGLT-2 inhibition is the best option for glucose control in a patient with IHD and heart failure failing glycaemic control on metformin because there is good evidence for outcome benefit across the class

This patient has a history of diabetes and cardiac failure after a myocardial infarction. Evidence in this patient group is strong for a putative benefit of SGLT-2 inhibitors. In particular both empagliflozin and canagliflozin have reported outcomes data which demonstrates a reduction in mortality of around 1/3rd. Their outcome trials also demonstrate a reduction in episodes of symptomatic cardiac failure and reduced progression of microvascular complications of diabetes.

The other options are all inappropriate. Adding gliclazide is associated with increased risk of hypoglycaemia, weight gain, and is not associated with positive cardiovascular outcomes. In particular, given his occupation as a taxi driver, hypoglycaemia should be avoided. Insulin glargine may promote significant weight gain, can worsen cardiac failure and does also cause hypoglycaemia. Pioglitazone promotes weight gain and fluid retention and is therefore contraindicated in cardiac failure. Saxagliptin was associated with an increase in cardiac failure in the SAVOR-TIMI cardiac outcome trial.

Question 17 of 114

A 40-year-old female presented to Endocrinology Clinic with a 3-month history of weight gain, fatigue and headaches. Over the last 3 weeks, she has also experienced galactorrhoea and reduced libido. She was diagnosed with type 2 diabetes and hypertension 1 month ago and is on diet control for both. She is not currently on any regular medications. On examination, there was evidence of hirsutism and acne, a cervical fat pad, striae on her abdomen and proximal myopathy. Areas of hyperpigmentation were noted on her mucous membrane and palmar creases.

Which of the following investigations will reveal the diagnosis?

A. Low dose dexamethasone suppression test
B. Prolactin levels
C. Urinary cortisol
D. CT brain
E. MRI pituitary

E. MRI pituitary

The history suggests Cushing's syndrome, but the occurrence of galactorrhoea and reduced libido brings to attention the possibility of hyperprolactinaemia, and headaches may indicate an intracranial pathology. The diagnosis of a secreting pituitary tumour causing a raised level of prolactin and ACTH (causing hyperpigmentation) and hence, cortisol, should be suspected.

Low dose dexamethasone suppression test and 24-hour urinary cortisol will aid confirming the presence of Cushing's syndrome. Raised prolactin levels will confirm hyperprolactinaemia, however, it is the MRI pituitary that will lead to the diagnosis of a pituitary tumour.

Question 18 of 114

An 82-year-old female presents to clinic with her daughter complaining of a four month of history of urinary incontinence. She explains that she has not previously had problems with continence. Her only past medical history include hypertension and angina. Now, she is incontinent of urine only when she laughs or coughs. At times, she reports sudden urges to urinate at all times during the day, resulting in a leak when she is unable to reach the toilet in time. This is significantly impacted on her sleep as well as it is increasingly frequent at night. The patient has reduced her caffeine intake already and has commenced 'bladder training' recommended by her GP.

What additional management would you commence?

A. Pelvic floor exercises only
B. Pelvic floor exercises and tolterodine
C. Pelvic floor exercises and duloxetine
D. Pelvic floor exercises and desmopressin
E. Long term urinary catheter

B. Pelvic floor exercises and tolterodine

The question concentrates on the management of an elderly patient with mixed stress incontinence and overactive bladder syndrome. In the context of mixed urinary incontinence, pharmacological therapies should be offered in addition to conservative therapies such as pelvic floor exercises. A long-term catheter in whom persistent incontinence causes skin wounds, the patient requires such significant care that continuous changing of bed linen and clothes would be required, chronic retention with a risk of renal impairment (and inability to self-catheterise) or if the patient opts for the catheterisation. The 2013 NICE guidelines recommends first line pharmacological treatment for mixed incontinence or overactive bladder syndrome alone is either tolterodine, oxybutynin or darifenacin1. Duloxetine should only be used if the patient does not tolerate first line therapy and is not a candidate for surgery. Desmopressin can reduce nocturia. However, its effects in platelet activation contradicts its use in ischaemic heart disease.

Question 19 of 114

A 43-year-old woman presents to the emergency department with abdominal pain radiating from her right loin to right groin. Her past medical history includes Sjogren's syndrome. She takes regular lubricating eye drops and mouth spray. She does not smoke or drink alcohol.

Her observations are heart rate 94 beats per minute, blood pressure 135/88mmHg, respiratory rate 18/minute, oxygen saturations 97% on air and temperature 36.7ºC.

On examination, there is left costovertebral angle tenderness. There is no evidence of peritonism and abdominal sounds are present. The cardiorespiratory examination is unremarkable.

Urinalysis:

Leucocytes	negative
Nitrites	negative
Blood	++
Glucose	-ve
pH	5.7 (<5.3)

Venous blood gas:

pH	7.27	(7.35-7.45)
Lactate	0.8 mmol/L	(0.0-2.0)
HCO3-	15 mmol/L	(22-26)
Cl-	110 mEq/L	(98-106)
Na	135 mmol/L	(135-145)
K+	3.1 mmol/L	(3.5-5.0)

Blood tests:

Hb	137 g/L	Male: (135-180)Female: (115 - 160)
Platelets	189 * 10 /L $_9$	(150 - 400)
WBC	8.2 * 10 /L $_9$	(4.0 - 11.0)
Na $_+$	135 mmol/L	(135 - 145)
K $_+$	3.0 mmol/L	(3.5 - 5.0)
Urea	4.2 mmol/L	(2.0 - 7.0)
Creatinine	89 µmol/L	(55 - 120)
CRP	4 mg/L	(< 5)
Calcium	2.05mmol/L	(2.20-2.60)
Parathyroid hormone	7.8 pmol/L	(1.6-6.9)

A CT KUB demonstrates a left-sided ureteric stone but also bilateral nephrocalcinosis.

What is the most likely explanation for the collected findings?

A. Primary hyperparathyroidism
B. Tertiary hyperparathyroidism
C. Type 1 renal tubular acidosis
D. Type 2 renal tubular acidosis
E. Type 4 renal tubular acidosis

C. Type 1 renal tubular acidosis

Hypokalaemia, nephrocalcinosis - type 1 renal tubular acidosis

Type 1 renal tubular acidosis is the correct answer. This is characterized by failure of hydrogen ion secretion into the lumen of the nephron. It leads to an inability to acidify the pH of the urine to < 5.3. This leads to a normal anion gap metabolic acidosis, hypokalaemia, hyperchloraemia, hypocalcaemia and in some cases, the development of nephrocalcinosis and renal stones. The development of this condition is associated with Sjogren's syndrome. The development of nephrocalcinosis is not seen in other types of renal tubular acidosis.

Type 2 renal tubular acidosis is incorrect. This can cause a normal anion gap metabolic acidosis (NAGMA) and hypokalemia. However, this is more associated with the development of osteomalacia. Nephrocalcinosis does not develop in this condition.

Type 4 renal tubular acidosis is incorrect. This is characterized by a NAGMA and hyperkalemia rather than hypokalaemia. This is associated with drugs such as NSAIDs and diabetic nephropathy.

Primary hyperparathyroidism is incorrect. The PTH level is appropriately raised given the low calcium.

Tertiary hyperparathyroidism is incorrect. This would cause a raised PTH in the setting of raised calcium, typically as a consequence of advanced chronic kidney disease. This is not the biochemical profile seen in this case.

Question 20 of 114

A 42-year-old woman was seen in clinic with a history of palpitation, tremor and weight loss. There is no other past medical history and she takes no regular medication.

On examination, she had a palpable goitre, exophthalmos, and a tremor of the out-stretched hands.

Thyroid function tests showed:

Free T4	36 pmol/L (10-25)
Free triiodothyronine (T3)	15 pmol/L (5-10)
Thyroid-stimulating hormone	0.1mU/L (0.4-5.0)

Which of the following treatments should be prescribed initially to improve symptoms?

A. Thyroidectomy
B. Propanolol
C. Radioiodine ablation
D. Carbimazole
E. Propylthiouracil

B. Propanolol

Propranolol should be used in new cases of Graves' disease to help control symptoms

There is a high suspicion of Grave's disease in this patient, and further investigation with a thyroid autoantibody profile is warranted.

Beta blockers are used to treat the symptoms of increased betaadrenergic tone that are seen in Grave's disease. Other such symptoms include anxiety and heat intolerance.

Thioamides are used to treat Grave's hyperthyroidism, but not specifically the symptoms of increased beta-adrenergic tone. While the anti-thyroid effect of these drugs has a rapid onset, their clinical effect is more delayed because the pre-formed store of hormone in the thyroid gland and bound to thyroid-binding globulin must first be exhausted.

Question 21 of 114

A 56-year-old man with type 1 diabetes arrives to hospital with one day of diarrhoea and vomiting. He felt well this morning but suffered indigestion and took some antacids without relief. The indigestion which he describes as a burning pain in the chest spreading into his throat lasted 4 hours however his lunch time blood glucose continued to rise despite several insulin boluses. He has been otherwise well over the past week and denies skipping meals, changes in exercise or any missed doses of his insulin. He smokes twenty cigarettes a day and maintains his diabetes on a basal bolus regime with correction doses based on carbohydrate counting.

On examination, he is sweaty with dry mucosa. His heart rate is 125/min, respiratory rate is 28/min with prolonged expiration phases, blood pressure is 110/90mmHg and temperature is 37ºC.

The nurse does his blood glucose which is 27mmol/l and his blood ketones are 4mmol/l.

Hb	130 g/l	Na +	133 mmol/l
Platelets	356 * 10 /l 9	K +	4.5 mmol/l
WBC	9.8 * 10 /l 9	Urea	6.2 mmol/l
Neuts	7.5 * 10 /l 9	Creatinine	98 µmol/l
Lymphs	1.0 * 10 /l 9	CRP	34 mg/l
Eosin	0.1 * 10 /l9		
HbA1c 48mmol/mol (normal range <42)			

ECG	deep T-wave inversion in V1-V4
Chest x-ray	nil acute seen

What is the likeliest cause that precipitated this presentation?

A. Missed insulin dose
B. Faulty blood glucose monitor
C. Lower respiratory tract infection
D. Myocardial infarction
E. Gastritis

D. Myocardial infarction

Myocardial infarction can precipitate diabetic ketoacidosis

This patient is a well controlled type 1 diabetic presenting with diabetic ketoacidosis (DKA) with no clear precipitant. Given a normal HbA1c and denial of any lifestyle changes, we should assume that he has not just missed his insulin. The blood glucose monitors are very reliable these days and rarely are wrong. Gastritis would not cause DKA. Myocardial infarction (MI) and lower respiratory tract infections can both precipitate DKA and given the burning chest pain and ECG changes, it would be warranted to do a troponin immediately as an MI this is the likeliest cause of DKA. To remember the causes of DKA, think 3 'I's which are insulin (missed), infection or infarction (i.e. MI).

Question 22 of 114

A 65-year-old retired nurse was referred to the endocrine clinic by her primary care physician in view of incidentally-detected hypercalcaemia. She had no significant past medical history and was not taking any regular medications. Her general physical and systemic examination was unremarkable.

Urea	7.5 mg/dl
Calcium	2.8 mmol/l
Phosphate	0.74mmol/l
Creatinine	98µmol/l
Alkaline phosphatase	450IU/l

Which one of the following investigations is most likely to help establish the diagnosis?

A. 1,25-OH vitamin D levels
B. PTH level
C. 25-OH vitamin D levels
D. Myeloma screen
E. Sestamibi scan

B. PTH level

Measuring PTH level is the first step in investigating hypercalcaemia

The most likely cause of hypercalcaemia in this case is primary hyperparathyroidism. The annual incidence is approximately 1 in 1000 and is commonly seen in post-menopausal women. The underlying aetiology of primary hyperparathyroidism is parathyroid adenoma (80- 85%) followed by parathyroid hyperplasia (~10-14%). Measuring PTH level is the first step in investigating hypercalcaemia.

Question 23 of 114

A 60-year-old woman had a thyroid function test requested by her General Practitioner after reporting some symptoms of mild lethargy. This had unexpectedly demonstrated a suppressed Thyroid Stimulating Hormone level (0.25 microU / L) but normal free T4 level (14.1 pmol / L). She denied any heat intolerance, weight loss, diarrhoea, hair or skin changes, palpitations or eye symptoms.

The patient had a hysterectomy without oophorectomy at age 45 as a treatment for menorrhagia secondary to fibroids. She remembers reaching menarche at around the age of 13 or 14 years. There is no significant family history of coronary artery disease. The patient reported that her mother had suffered a fractured neck of femur at the age of 75 years following a fall. The patient was a retired school teacher with an active lifestyle. She had never smoked and drank very little alcohol.

Examination showed no evidence of a goitre, no fine tremor and no lid lag. External examination of the eyes was unremarkable. The cardiovascular and respiratory examination was unremarkable.

The GP requested some further basic investigations and then repeated blood tests 2 months after the original test. At this time, the patient reported her previous symptoms of lethargy had improved; with hindsight, she attributed this to grief due to the recent death of a close friend.

Ambulatory blood pressure monitoring: average blood pressure 125 / 75 mmHg

ECG: sinus rhythm at 75 bpm; normal axis; no abnormality of QRS, ST interval or T waves.

Haemoglobin	12.8 g / dL
White cell count	6.5 x 10 /l9
Platelets	206 x 10 /l9
Urea	6.2 mmol / L
Creatinine	95 micromol / L

Sodium	137 mmol / L
Potassium	4.0 mmol / L
C-reactive protein	< 1
Parathyroid hormone	3.7 pmol / L (reference 1.2-5.8)
Thyroid-stimulating hormone	0.21 microU / L (reference 0.4-5.0)
T4 free serum	13.8 pmol / L (reference 8.5-15.2)
T3 free serum	5.6 pmol / L (reference 3.5-6.5)
HbA1C	5.6 % (reference 4-6)
Total cholesterol	4.0 mmol / L
LDL cholesterol	1.8 mmol / L
HDL cholesterol	1.9 mmol / L

What is the most appropriate management of the deranged thyroid function tests?

A. DEXA scan
B. Thyroid ultrasound
C. Start treatment with simvastatin
D. Radioiodine therapy
E. Treat with propylthiouracil

A. DEXA scan

The patient has subclinical hyperthyroidism with persistently suppressed TSH levels but normal serum thyroid hormone levels and with no clinical evidence of thyrotoxicosis. This usually occurs in the setting of thyroid overactivity due to Graves' disease or autonomously functioning thyroid nodules sufficient to suppress pituitary TSH secretion but insufficient to cause an elevation of circulating hormones. Progression to overt hyperthyroidism occurs in 1-3 % of elderly patients per year.

The main risk of subclinical hyperthyroidism is the increased risk of atrial fibrillation and hip fractures. The American Association of Clinical Endocrinologists recommends that treatment is considered in patients with a persistently low TSH level if they are older than 65 years or are at risk of osteoporosis or heart disease.

This patient has a low level of cardiac risk factors with a low-risk lipid profile. Assessment of her osteoporosis risk is complicated by her hysterectomy preventing knowledge of her age at menopause. Therefore, a DEXA scan is appropriate next line management to quantify her osteoporosis risk and inform the decision as to whether or not to treat the sub-clinical hyperthyroidism.

Thyroid ultrasound would not influence the decision to treat at this stage and so is not required.

Question 24 of 114

A 19-year-old woman presents to the emergency department drowsy and vomiting. She is accompanied by a friend who tells you she has been out drinking all day and has been vomiting for the last few hours.

Her speech is slurred and confused, she opens her eyes in response to her name and pushes you away in response to a painful stimulus. Heart rate is 100 beats per minute and regular, blood pressure is 100/60 mmHg, capillary glucose is 18 mmol/L, and a urine dip shows pH: 4, blood: trace, ketones: +++, protein: trace, nitrites: negative and leukocytes: negative.

Chest x-ray: Normal Venous blood gas:

pH	7.27 (7.35-7.45)
Bicarbonate	10mmol/L (22-26)
Base excess	-10 (-2 to +2)
Sodium	135 mmol/L (137-144)
Potassium	2.9 mmol/L (3.5-4.9)
Chloride	99 mmol/L (95-107)

Serum Glucose: 21 mmol/L

What is the most important initial intervention?

A. Fixed rate intravenous insulin infusion (FRIII)
B. Intravenous calcium gluconate
C. Intravenous fluids
D. Sliding scale insulin
E. Urgent CT head

C. Intravenous fluids

This woman fits the diagnostic criteria for diabetic ketoacidosis (DKA):

- Ketonaemia > 3.0mmol/L or significant ketonuria (more than 2+ on standard urine sticks)
- Blood glucose > 11.0mmol/L or known diabetes mellitus
- Bicarbonate (HCO3-) < 15.0mmol/L and/or venous pH < 7.3

Diabetes UK guidance states that the most important initial therapeutic intervention in DKA is appropriate fluid replacement followed by insulin administration.

The key benefits of fluid resuscitation in this context include:

- Recovery of circulatory volume
- Clearance of ketones and therefore improvement of acidosis
- Correction of electrolyte imbalance

Weight based fixed rate intravenous insulin infusion (FRIII) is now the recommended mode of insulin administration in DKA, over sliding scale.

DKA is a complication of type i diabetes (but can rarely complicate type ii diabetes). It can be the first presentation of type i diabetes, result from poor diabetic control or be precipitated by another factor such as infection.

Question 25 of 114

A 75-year-old woman presents to her general practitioner (GP) due to increased tiredness and weight gain. Her past medical history includes hypothyroidism and hypertension, for which she takes levothyroxine and amlodipine.

She last saw the GP two weeks previously when she was started on ferrous sulphate due to mild anaemia.

What is the most likely cause of her symptoms?

A. Bowel malignancy
B. Addison's disease
C. Amlodipine toxicity
D. Undercorrected hypothyroidism
E. Worsening anaemia

D. Undercorrected hypothyroidism

The most likely explanation here is undercorrected hypothyroidism. Ferrous sulphate is well known to reduce the absorption of other medications, particularly levothyroxine. Patients should be advised to take these tablets separately from their regular medications.

Although anaemia in a post-menopausal woman should always be investigated, there are no symptoms to suggest malignancy here. Addison's disease would be unlikely to cause weight gain.

Question 26 of 114

A 65-year-old man attends with a hot swollen right toe. On examination there is swelling over the MTP joint of the right toe. There is reduced range of movement and it is hot, swollen and painful to touch. He has a past medical history of alcohol excess. He takes no regular medications.

An aspirate of the joint fluid is as follows:

Microscopy	Negatively birefringent crystals - needle shaped

The patient is intolerant of NSAIDs and colchicine. You therefore start a trial of prednisolone. Unfortunately the patient develops steroid induced psychosis.

What agent will you consider prescribing?

A. Canakinumab
B. Ibuprofen
C. Naproxen
D. Paracetamol
E. Allopurinol

A. Canakinumab

Canakinumab is a human monoclonal antibody that selectively inhibits interleukin-1 beta receptor binding. It can be used for treatment of acute gout has not responded adequately to treatment with NSAIDs or colchicine, or who are intolerant of them

Canakinumab is a human monoclonal antibody that selectively inhibits interleukin-1 beta receptor binding. It can be used for treatment of acute gout has not responded adequately to treatment with NSAIDs or colchicine, or who are intolerant of them.

The patient is intolerant of NSAIDs therefore ibuprofen and naproxen would be inappropriate.

Paracetamol is a simple analgesic and unlikely to control the patients pain. In addition, the inflammatory nature of gout warrants the use of a drug which reduces inflammation.

Allopurinol is indicated for recurrent gout. It should not be initiated in the acute setting as doing so can prolong the duration and severity of the gout.

Question 27 of 114

A 20-year-old female presented to the accident and emergency department with severe abdominal pain, vomiting and lethargy. On further questioning she stated that she had been generally unwell for the last four months during which time she lost 10 Kg in weight and had been tired all the time.

Last month she has been diagnosed with hypothyroidism and was prescribed levothyroxine 50 mcg daily.

Her mother and sister have hypothyroidism and take thyroxine. On examination, she looks unwell and dehydrated.

Her pulse is 105 beats per minute and blood pressure is 70/40 mmHg

Her temperature is 37.6ºC and BMI is 19 kg/m². Cardiovascular, respiratory and abdominal examination were normal. Investigations done last month showed:

Hb	9.5 g/dl
MCV	105 fl
Platelets	190 * 10 /l9
WBC	4.5 * 10 /l9

Serum free T4	8.5 pmol/l
Serum TSH	5.5 mU/l

While awaiting new investigations, what is the most appropriate immediate treatment for this patient?

A. Intravenous glucose 10%
B. Intravenous normal saline
C. Intravenous normal saline and antibiotics
D. Intravenous normal saline and hydrocortisone
E. Intravenous thyroxine

D. Intravenous normal saline and hydrocortisone

This patient presented with Addisonian crisis (abdominal pain, vomiting, dehydration and hypotension). She has been complaining of tiredness and weight loss (which are features of Addisons disease) for four months but what precipitated the crisis is the thyroxine given for the presumed hypothyroidism.

Actually, a slightly raised TSH and a decreased T4 are features of primary hypoadrenalism and do not necessarily indicate frank hypothyroidism.

This is a medical emergency and should be treated immediately with intravenous normal saline and hydrocortisone. Thyroxine should not be given as it will exacerbate the condition.

Her low haemoglobin and high MCV may point towards pernicious anaemia which is an autoimmune disease seen sometimes in association with Addisons disease.

Question 28 of 114

A 47-year-old builder presented with paraesthesia in both hands which was worse at night. His hands felt swollen, although they were not painful, and he had needed to buy a larger pair of work gloves. When at work he found that his hands felt weak. Over the past six months, he had been experiencing urinary frequency, fatigue and increased thirst.

He had a past medical history of obesity and hypertension and his brother had type II diabetes mellitus. His only medication was ramipril. He was a heavy smoker with a 20 pack year history.

On examination of the arms, there was weakness of thumb abduction bilaterally and diminished sensation over the radial three and a half digits. Percussion over the palmar aspect of the wrist reproduced the paraesthesia he described on presentation. On examination of the chest and abdomen, there were areas of pigmentation in both axillae and striae over the abdomen. He had a protuberant abdomen and an elevated body mass index (BMI).

Which of the following investigations is most likely to be diagnostic?

A. Magnetic Resonance Imaging of the Pituitary and visual field testing
B. Fasting glucose on three occasions, glycosylated haemoglobin(HbA1c) and a 9am cortisol measurement
C. Growth hormone measurement and dexamethasone suppressiontest
D. Nerve conduction studies and electromyogram (EMG)
E. Oral glucose tolerance test with serum glucose, IGF-1 andgrowth hormone measurements

E. Oral glucose tolerance test with serum glucose, IGF-1 andgrowth hormone measurements

This gentleman has bilateral carpal tunnel syndrome with an underlying diagnosis of acromegaly.

The features in the question which point to the diagnosis of acromegaly include the presence of a recent increase in hand size and known complications of acromegaly such as diabetes mellitus (polyuria and polydipsia), acanthosis nigricans (pigmentation in the axillae) and carpal tunnel syndrome.

The increased BMI and striae could be indicative of underlying Cushing's disease, but in this case, this were red herrings.

The diagnosis of acromegaly is made when there is a failure to suppress the release of growth hormone during an oral glucose tolerance test. This test also allows the diagnosis of diabetes mellitus to be made. Due to the diurnal variation in growth hormone levels, a random measurement is not helpful in making a diagnosis.

Although an MRI scan of the pituitary and visual field testing are very important investigations and can confirm the presence of a pituitary adenoma they will not confirm or exclude a diagnosis of acromegaly.

A fasting glucose and HbA1c measurement may aid the diagnosis of diabetes mellitus but not acromegaly.

A 9 am cortisol and dexamethasone suppression test could be used to investigate Cushing's syndrome and therefore are not relevant here.

Nerve conduction studies and electromyography would confirm the diagnosis of carpal tunnel syndrome but not the underlying cause, i.e. acromegaly.

Question 29 of 114

A 55 year-old male presents to endocrine outpatient clinic for investigation of gynaecomastia. On examination he has bilateral growth of breast tissue with palpable glandular tissue around the areolae. His past medical history includes hypertension, hypothyroidism, and congestive cardiac failure. He drinks 30 units of alcohol per week. His regular medications include: levothyroxine, amlodipine, bisoprolol, lisinopril and spironolactone.

On examination he is of normal stature, there are no peripheral stigmata of chronic liver disease or testicular masses.

What is the most likely explanation of his gynaecomastia?

A. Cirrhosis
B. Hypopituitarism
C. Iatrogenic
D. Klinefelter's syndrome
E. Idiopathic

C. Iatrogenic

The most likely explanation for this gentlemans gynaecomastia given the negative examination findings is a side effect of his current regular medications. There are many drugs that can cause gynaecomastia; in this cause the culprit could be either amlodipine or spironolactone.

Question 30 of 114

You are seeing a 50-year-old lady with type 2 diabetes mellitus in the outpatient clinic. She has a past medical history of gastritis, moderate left ventricular dysfunction and chronic obstructive pulmonary disease. She is currently on metformin and gliclazide. Since last review she has gained 5kg in weight and her HbA1c has deteriorated to 70 mmol/mol from 62 mmol/mol. Body mass index today in clinic is 33 kg/m².

Recent blood tests are as follows:

Na +	141 mmol/l
K +	3.9 mmol/l
Urea	6 mmol/l
Creatinine	140 µmol/l

She was unable to previously tolerate liraglutide due to nausea and vomiting.

What would be the best alteration to her therapy?

A. Empagliflozin (SGLT-2 inhibitor)
B. Add insulin
C. Add pioglitazone
D. Increase dose of metformin
E. Increase dose of gliclazide

A. Empagliflozin (SGLT-2 inhibitor)

SGLT inhibitors have the advantage of improving glycaemic control/HbA1c and having beneficial effects on weight. This is because their mode of action is independent of insulin release. They act upon the SGLT-2 receptors in the kidney and lead to increased loss of glucose in the urine.

Question 31 of 114

A 32-year-old alcoholic presents with abdominal pain and vomiting. His amylase is 1200 U/l and he is being treated for acute pancreatitis. You are called to see him as the nursing staff report the patient is becoming restless. He complains of numbness around his mouth and appears to be in some discomfort.

Your foundation year 2 colleague notes this morning's blood results:

Adjusted calcium	1.8mmol/l
Na +	136 mmol/l
K +	3.7 mmol/l
Urea	6.9 mmol/l
Creatinine	81 µmol/l

What's the next step in management?

A. Parathyroid hormone
B. Oral calcium supplementation
C. Calcitonin assay
D. Intravenous 10% calcium gluconate
E. Serum magnesium

D. Intravenous 10% calcium gluconate

Intravenous calcium gluconate is used for the acute management of hypocalcaemia

In the acute setting of a patient with symptomatic hypocalcaemia intravenous replacement with 10% calcium gluconate would be the most sensible next step. Typically this is given over 10-30 minutes ideally with cardiac monitoring.

Checking the serum magnesium would be very useful as hypomagnesaemia would require replacement to also correct hypocalcaemia.

Oral calcium replacement would be an option in asymptomatic hypocalcaemic patients.

Question 32 of 114

A middle age woman is being treated for symptomatic hypercalcaemia associated with a squamous cell lung cancer (serum calcium 3.60 mmol/L). She is slow to respond to initial measures of saline hydration and intravenous pamidronate. Whilst awaiting surgical resection for her underlying cancer what may be the next best step in her management?

Na +	142 mmol/l
K +	4.3 mmol/l
Urea	7.0 mmol/l
Creatinine	89 µmol/l
Glucose	4.8 mmol/l

What is the most appropriate management?

A. High dose loop diuretics
B. Calcitonin 4 units/kg
C. Insulin actrapid 50 units in 50% dextrose
D. IV colloid administration instead of crystalloid
E. Plasma exchange

B. Calcitonin 4 units/kg

General symptoms of hypercalcaemia may include malaise, lethargy, depression, dehydration and can lead to depressed consciousness. Bone pain and abdominal pain may feature and can be summarised by the classic 'bones, stones, moans and abdominal groans'.

Alongside searching for the underlying cause, management initially involves aggressive rehydration, typically 4-6 L saline on the first day. Bisphosphonates act by interfering with osteoclastic bone resorption and typically IV pamidronate is used at a dose of 60-90mg over 2-4 hours. Calcitonin (extracted from salmon) also interferes with osteoclast activity as well as increasing renal calcium excretion.

Diuretics may lead to further dehydration. Dialysis may be a last line treatment for life threatening hypercalacemia, but not plasma exchange.

Question 33 of 114

A 65-year-old Muslim man with type II diabetes on metformin (500mg three times a day) comes to your endocrine clinic. He is about to start fasting between sunrise and sunset for Ramadan. He will typically eat a light meal before sunrise (Suhoor) and a large meal at sunset (Iftar).

How would you advise this gentleman in respect to his metformin prior to the large meal at sunset?

A. Change to a sulphonylurea
B. Stop metformin
C. Take 1.5g metformin before the large meal at sunset
D. Take 500mg metformin before the large meal at sunset
E. Take 1g metformin before the large meal at sunset

E. Take 1g metformin before the large meal at sunset

This patient will still require metformin and therefore it is not appropriate to stop it.

The total daily dose of metformin should be split:

- One-third before sunrise (Suhoor) (500mg)
- Two-thirds after sunset (Iftar) (1g)

There is no need to change to a sulphonylurea if his blood glucose levels are controlled on metformin alone.

Question 34 of 114

An elderly male presents with a 2 week history of breathlessness. His past medical history includes diet-controlled type 2 diabetes, ischaemic heart disease, hypothyroidism and depression. His medication list includes levothyroxine, aspirin, simvastatin, ramipril, bisoprolol and citalopram. Observations on presentation to Emergency Department are as follows: respiratory rate 26/min, saturations 94% (on 4 litres oxygen via Venturi), heart rate 80 beats per minute, blood pressure 156/82 mmHg. Auscultation demonstrates crackles at the left base with no wheeze. The abdomen is soft and non-tender. There is no oedema peripherally.

Blood results on admission are provided below:

Hb	134 g/l
Platelets	172 * 10 /l9
WBC	13.3 * 10 /l9
Na +	128 mmol/l
K +	5.1 mmol/l
Urea	13 mmol/l
Creatinine	178 µmol/l
Serum osmolality	220 mosm/kg
Urinary sodium	50 mEq//l

What is the most likely cause of hyponatraemia?

A. Hypothyroidism
B. Chronic kidney disease
C. Addison's disease
D. Salt-losing nephropathy
E. Syndrome of inappropriate antidiuretic hormone (SIADH)

E. Syndrome of inappropriate antidiuretic hormone (SIADH)

This question demonstrates a common scenario in clinical practice. Management of hyponatraemia first requires clarification of fluid status (clinical hypovolaemia, euvolaemia or hypervolaemia), as differentials are influenced by this. This patient's history, examination findings and haemodynamic parameters are consistent with clinical euvolaemia.

Differentials for euvolaemic hyponatraemia would include hypothyroidism and SiADH. There are no clinical features suggestive of the former. Findings are consistent with community-acquired pneumonia with associated SiADH. This is confirmed by the presence of reduced serum osmolality and high urinary sodium. Measurement of urinary sodium concentration is an useful adjunct in helping to differentiate between hyponatraemia secondary to hypovolaemia and SiADH. With SiADH (and salt-wasting syndrome), the urinary sodium is high. With hypovolaemia, the urinary sodium is typically low.

Question 35 of 114

A 19-year-old man is brought by ambulance to the emergency department. The patient himself was too unwell to provide a coherent history but his mother reports that he had been very unwell with loss of appetite and abdominal pain for the past two days. She had also noted a significant weight loss in her son over the past six months and that he had been unusually fatigued over the same time period. The patient had no previous family history although his older sister had recently been diagnosed with pernicious anaemia.

Examination showed the patient to be tachypnoeic and tachycardic with a central capillary refill time of six seconds. Respiratory and cardiovascular examination was otherwise unremarkable. Abdominal examination revealed some inconsistent tenderness without obvious signs of localised peritonism.

Please see below for results of initial investigations.

Urea	10.6 mmol / L
Creatinine	134 micromol / L
Sodium	149 mmol / L
Potassium	5.8 mmol / L
Fingerpick blood glucose	42 mmol / L
Fingerpick blood ketones	7.5 mmol / L

Venous blood gas (room air)

pH	7.09
PCO2	16 mmHg (reference 32-43)
PO2	52 mmHg (reference 70-100)
Bicarbonate	10.9 mmol / L (reference 20.0-26.0)
Chloride	107 mmol / L (reference 99-108)
Lactate	4.9 mmol / L

Portable chest x-ray: poor quality film secondary overlying artefact; no free air under diaphragm, lung fields clear, no pneumothorax.

Electrocardiogram: sinus tachycardia at 129; normal intervals and axis; QRS morphology normal; T wave morphology normal.

What is the correct immediate management for this patient?

A. 1000 mg calcium gluconate
B. 10 mL 8.4 % sodium bicarbonate
C. 1000 mL 0.9 % saline
D. 1000 ml 0.45 % saline
E. Intravenous insulin bolus

C. 1000 mL 0.9 % saline

The patient has clear biochemical evidence of diabetic keto-acidosis, most likely representing a first presentation of type 1 diabetes mellitus.

Correct immediate management is to give a litre of 0.9 % saline to restore intravascular volume while preventing a rapid change in extracellular osmolality. For subsequent IV fluids, The Joint British Diabetes Society recommends ongoing use of 0.9 % saline, in contrast to the American Diabetes Association that recommends 0.45 % saline if sodium level normal or high.

An initial bolus dose of intravenous insulin is not recommended after a randomisation controlled trial evidence found no benefit. A weightbased insulin infusion of 0.1 units/kg/hour is recommended.

Bicarbonate infusion are not routinely recommended in the treatment of DKA, although may be considered in patients with severe acidosis (pH < 7) and associated life-threatening effects of acidaemia.

Given the absence of ECG changes of hyperkalaemia in this patient, an infusion of calcium gluconate is not required. In fact, the patient is likely to be more at risk of hypokalaemia as the treatment for DKA progresses.

Question 36 of 114

A 50-year-old obese gentleman presented to the emergency department with sweating, pallor, shortness of breath and central chest pain. He was diagnosed with a STEMI and underwent primary PCI. An ECHO after the event showed an EF of 35%. Following work up for his coronary artery disease he was diagnosed with type 2 diabetes. He was commenced on iv insulin which controlled his blood glucose in the interim.

Investigations

Creatinine	122umol/L
Urea	8.2mmol/l
Na+	140 mmol/l
K+	3.6 mmol/l
eGFR	62 ml/min
HbA1C	9.4%
HCO3	22
Aspartate transaminase	52 U/L
Alkaline phosphatase	110 U/L
Gamma-glutamyl transferase	39 U/L

In the long term treatment of his diabetes which hypoglycaemic agent is best avoided?

A. Metformin
B. Pioglitazone
C. Gliclazide
D. Acarbose
E. Insulin glargine

B. Pioglitazone

Pioglitazone - contraindicated by: heart failure

Answer: Pioglitazone

One of the side effects of pioglitazone is fluid retention, therefore it is best avoided in a patient with a risk of heart failure like the above patient as it can precipitate or worsen heart failure due to fluid overload.

Question 37 of 114

A 80-year-old patient was referred to Accident and Emergency after being found unresponsive in his home. He had just completed a course of antibiotics for a chest infection. He had not been seen for the preceding 36 hours. He had a past medical history of hypertension and type two diabetes.

His medication included Metformin, Gliclazide, Humulin M3 insulin twice a day, Ramipril and Bendroflumethiazide.

His initial examination revealed. Blood pressure 104/53, heart rate 103 beats per minute, respiratory rate 24 and oxygen saturations 90% on air. He had inspiratory crackles on his left lower lung zone. He had sunken eyes, capillary refill time of four seconds and no lower limb swelling. GCS 13 out of 15.

Initial blood tests;

Hb	11.0 g/dL
WCC	21.4 *10^9/l
Platelets	189 *10^9/l
CRP	340 mg/L
Na+	149 mmol/l
K+	4.4mmol/l
Ur	28 mmol/l
Cr	180 µmol/l
Glucose	54mmol/l

ABG on air

pH	7.32
pCO2	3.7kPa
pO2	9kPa
HCO3	18 mmol/l
Lactate	2.4mmol/l

Urine dipstick analysis - ++ glucose, - WCC, - leucocytes, + ketones

The patient was treated with oxygen, intravenous antibiotics for a chest infection and prophylactic low molecular weight heparin. They were treated with the local diabetic ketoacidosis protocol with IV inulin sliding scale and IV fluids 5500ml in 24 hours.

His repeat bloods 12 hours later were;

Na+	132 mmol/l
K+	3.9 mmol/l
Ur	12 mmol/l
Cr	110 µmmol/l
Glucose	5 mmol/l
HCO3	24 mmol/l
Lactate	1.7 mmol/l
CRP	270mg/l

The patient developed a grand mal seizure. His Glasgow coma scale remained 10 an hour after the seizure.

What is the most likely cause of his neurological deterioration?

A. Hypoglycaemia
B. Intracranial venous sinus thrombosis
C. Cerebral oedema
D. Sepsis
E. Renal failure

C. Cerebral oedema

All the answers are potentially associated with hyperosmolar hyperglycaemic coma. The current guidance advises treatment initially with normal saline intravenous rehydration. The target reduction in osmolality is 3-8 mosmol/kg an hour. Only if this target is not being met and the glucose level not reducing sufficiently is insulin to be started.

This patient developed cerebral oedema secondary to rapid reduction in serum osmolality.

Question 38 of 114

A 55-year-old man presents to the endocrine clinic. He was diagnosed five years ago with type 2 diabetes and is struggling to control his sugars. He is currently taking:

Metformin 1g BD Glicazide 160mg BD Sitagliptin 100mg OD

He is a bus driver and struggles to control his weight with his hectic shifts. Current BMI is 34 kg/m².

Investigations:

Serum creatinine	120 µmol/L (60-110)
Haemoglobin A1c	66 mmol/mol (8.2%)

What would be the most appropriate next step?

A. Canagliflozin
B. Glibenclamide
C. Increase metformin
D. Stop sitagliptin and add insulin
E. Stop sitagliptin and add exenatide

E. Stop sitagliptin and add exenatide

Given the NICE guidance the most appropriate step would be to start this patient on exenatide. This patient is already of metformin, glicazide and sitagliptin and the blood sugar levels are not under control. His BMI is under 35 but insulin would make 'it much more difficult for you to do your job'.

Question 39 of 114

A 72-year-old male was admitted drowsy and confused. His family describe a 4-day history of shortness of breath and a productive cough. His past medical history includes type 2 diabetes mellitus, hypertension and hypercholesterolaemia. He usually takes metformin 500 mg three times daily, gliclazide 80 mg twice daily, amlodipine 5 mg daily and simvastatin 40 mg nightly. On examination he is confused with dry mucous membranes, blood pressure of 100/50 mmHg, a pulse of 110/min, a temperature of 37.6 C and a respiratory rate of 20/min. Course crepitations were found at the right base and his pulse was thready with a capillary refill of 3 seconds; jugular venous pressure was not visible. Capillary blood glucose was found to be HI.

A venous blood sample is taken:

Hb	129 g/l	Na +	161 mmol/l
Platelets	204 * 10 /l 9	K +	4.9 mmol/l
WBC	13.1 * 10 /l 9	Urea	15.2 mmol/l
Neuts	11.9 * 10 /l 9	Creatinine	97 µmol/l
Glucose	56 mmol/l	eGFR	62 mg/l
Ketones	1.9 mmol/l	HbA1c	75 mmol/mol
pH	7.35	HCO 3	20 mmol/mol

What treatment would you initiate first?

A. 0.9% normal saline
B. 0.45 % normal saline
C. Hartmann's solution
D. Intravenous insulin
E. 5% dextrose

A. 0.9% normal saline

The underlying diagnosis is hyperosmolar hyperglycaemic state (HHS) precipitated by a lower respiratory tract infection. The diagnosis is made when a patient shows marked hyperglycaemia and hypovolaemia with a serum osmolality > 320 mosm/kg in the absence of marked ketonaemia or acidosis. This patient is clinically dry with a glucose of 56 and a calculated osmolality of 2(161+4.9) + 56 + 15 = 403 mosm/kg with mild ketonaemia.

Despite this gentlemen's hypernatraemia, the first line fluid therapy is 0.9% normal saline as patients in HHS are sodium deplete. One litre should be administered rapidly and electrolytes then repeated to gauge potassium requirements in further fluid bags. A small initial rise in sodium is expected and this should not discourage further use of normal saline. Insulin is not routinely started immediately in the absence of significant ketonaemia as glucose will fall with fluid therapy alone. When glucose stops falling insulin may be started if glucose still remains high. Too rapid correction of osmolality i.e. with aggressive insulin and fluids in combination can precipitate cerebral oedema.

Question 40 of 114

A 78-year-old man with type 2 diabetes mellitus is reviewed in the diabetes clinic. He is currently taking metformin 1g bd. He also has a history of hypertension and hypothyroidism. His HbA1c one year ago was 44 mmol/mol (6.2%). The most recent test is reported as 46 mmol/mol (6.4%).

What is the most appropriate next step in management?

A. Increase dose of metformin
B. Add glimepiride
C. Add sitagliptin
D. Add pioglitazone
E. Make no changes

E. Make no changes

The correct answer is to **make no changes** to the patient's current management. This is appropriate because the patient's glycaemic control is well within target range for his age and comorbidities. According to UK guidelines (NICE), the target HbA1c for older adults with type 2 diabetes should be individualised, with less stringent targets (up to 64 mmol/mol or 8.0%) being appropriate for those who are frail or elderly. This patient's HbA1c of 46 mmol/mol (6.4%) represents excellent control, and the small increase from 44 mmol/mol (6.2%) over the past year is clinically insignificant. Intensifying treatment would increase the risk of adverse effects without providing meaningful clinical benefit.

Increasing the dose of metformin would be inappropriate as the patient already has good glycaemic control. Increasing the dose would only increase the risk of gastrointestinal side effects and potentially vitamin B12 deficiency without providing significant clinical benefit.

Adding glimepiride, a sulfonylurea, would be inappropriate as it would increase the risk of hypoglycaemia in this elderly patient. Sulfonylureas are particularly risky in older adults due to their potential to cause severe hypoglycaemia, which can lead to falls and cognitive impairment.

Adding sitagliptin, a DPP-4 inhibitor, would be unnecessary given the current level of glycaemic control. While DPP-4 inhibitors have a good safety profile, adding another medication would increase cost and pill burden without clear benefit.

Adding pioglitazone would be inappropriate as thiazolidinediones can cause fluid retention and increase the risk of heart failure. Given this patient's age and existing cardiovascular risk factors (hypertension), pioglitazone would be a poor choice, particularly when current glycaemic control is already adequate.

Question 41 of 114

A 19-year-old with type 1 diabetes presents to the Emergency Department feeling unwell. She states she has had vomiting and diarrhoea for 2 days and has not been taking her full insulin doses as she has been off her food. Her capillary glucose is 37 mmol/l and there are 4+ ketones on urinalysis.

An arterial blood gas is performed and the results are as follows:

pH	7.12
pO2	13 kPa
pCO2	3.5 kPa
HCO3	13
Na	129 mmol/l
K	6.1 mmol/l

Which of the following is the most appropriate initial management?

A. IV 0.9% NaCl bolus
B. IV 10 units actrapid + 50ml 50% dextrose
C. IV 8.4% sodium bicarbonate
D. Empirical IV antibiotics
E. Insulin sliding scale

A. IV 0.9% NaCl bolus

This is a classical presentation of diabetic ketoacidosis. While precise protocols vary, the key principals are initial fluid resuscitation with normal saline prior to starting an IV insulin infusion, and careful potassium replacement.

Low sodium is often seen and is a pseudohyponatraemia secondary to the high serum glucose.

Serum potassium derangements are common and need careful management. Potassium is driven into cells by insulin. Serum potassium levels are therefore often high on presentation while blood insulin levels are depleted. Despite this, total body potassium is low due to fluid losses and requires careful monitoring and replacement during treatment.

Question 42 of 114

A 62-year-old woman with a history of type 2 diabetes comes to the clinic for review. She has a history of mild cardiac failure managed with ramipril and bisoprolol. Her current medication for diabetes is metformin 1g BD. On examination her blood pressure is 122/82 mmHg, pulse is 80 beats per minute and regular. There are bilateral basal crackles on auscultation of the chest, and pitting oedema of both ankles. Her body mass index is elevated at 33 kg/m².

HbA1c	73 mmol/mol
Creatinine	82 µmol/l

Which of the following is the most appropriate next step for managing glucose control?

A. Empagliflozin
B. Glipizide
C. Liraglutide
D. Pioglitazone
E. Saxagliptin

A. Empagliflozin

This patient has moderate obesity with a BMI of 33, cardiac failure, and a poorly controlled HbA1c at 33. Out of the available options empagliflozin, an SGLT-2 inhibitor is the most appropriate. As well as lowering blood glucose, SGLT-2 inhibition also has a diuretic like effect, lowering blood pressure and reducing fluid retention. Empagliflozin has also been shown to reduce cardiovascular mortality, and this patient is at high risk of an event.

Glipizide is inappropriate as it may drive further weight gain in a patient who is already obese, saxagliptin is recognised to cause heart failure, and pioglitazone has also been shown in clinical trials to cause fluid retention. This leaves liraglutide as the other potential option, which

doesn't significantly impact on fluid balance and according to NICE is only indicated at BMI of 35 and above.

Question 43 of 114

A 19-year-old woman with a history of type 1 diabetes is brought to the Emergency department with nausea and vomiting. there is no history of diarrhoea. She also has coeliac disease. She follows a gluten free diet and takes a basal bolus insulin regime with a usual HbA1c of 53 mmol/mol. On examination her blood pressure is 100/80 mmHg with a postural drop of 20 mmHg. Pulse is 88 beats per minute and regular. She looks dehydrated and tanned, she puts her tan down to weeks in the garden after her exams.

Investigations

Hb	102 g/l	Na +	129 mmol/l
Platelets	189 * 10 /l 9	K +	5.0 mmol/l
WBC	10.9 * 10 /l 9	Urea	9.9 mmol/l
Neuts	6.2 * 10 /l 9	Creatinine	113 µmol/l
Lymphs	1.1 * 10 /l 9	CRP	42 mg/l
Eosin	1.5 * 10 /l9		

Which of the following is the most important intervention with respect to her management?

A. Fluid restriction
B. IV anti-emetic
C. IV hydrocortisone
D. IV normal saline
E. NG feeding

C. IV hydrocortisone

In a patient with type 1 diabetes, such tight glycaemic control with hypoglycaemia would be considered very unusual. Coupled with the easy tanning, nausea, vomiting, and postural drop in blood pressure, this is highly suggestive of possible Addison's disease. The slight rise in eosinophil count, anaemia, hyponatraemia and potassium at the upper end of the normal range, all support the diagnosis.

In this situation corticosteroid replacement is crucial, without it normal saline replacement alone won't correct any hypotension, nor will it improve hyponatraemia. Although anti-emetics and NG feeding may be useful in restoring this patient to health, they won't correct underlying adrenal insufficiency.

Question 44 of 114

A 30-year-old woman with a history of type 1 diabetes presents to the Emergency department with nausea and vomiting coupled with increased urinary frequency over the past 3 days. She has been progressively losing weight and reducing her insulin dose after starting empagliflozin prescribed to help her lose weight and reduce glucose fluctuations. She also admits to taking a Chinese herbal remedy for weight control. Blood pressure is 100/70 mmHg, pulse is 88 beats per minute. pH is 7.25, glucose is 8.1 mmol/l, urine testing reveals ketones +++

Which of the following is the most likely diagnosis?

A. Empagliflozin related nephrotoxicity
B. Hyperosmolar non-ketotic state
C. Normoglycaemic ketoacidosis
D. Starvation ketoacidosis
E. Urinary sepsis

C. Normoglycaemic ketoacidosis

Hyperosmolar non-ketotic state is associated with marked hyperglycaemia and no ketosis. Starvation ketosis results in the formation of urinary ketones, but pH remains normal.

Question 45 of 114

A 50-year-old man who drives a heavy goods vehicle comes to the diabetes clinic for review. Current medication for diabetes includes metformin 1g BD and gliclazide 160mg BD. His blood sugars have steadily increased over the past few months and his most recent HbA1c is 72 mmol/mol. He has increased in weight by 5kg over the past 12 weeks which he puts down to work, (driving his lorry for excessive periods). On examination his blood pressure is 155/88 mmHg, pulse is 75 beats per minute and regular. Abdomen is soft and non-tender, body mass index is 37 kg/m².

Investigations:

Na +	140 mmol/l
K +	5.0 mmol/l
Urea	7.1 mmol/l
Creatinine	110 µmol/l

Which of the following is the most appropriate next step?

A. Add basal insulin
B. Add mixed insulin
C. Add pioglitazone
D. Switch gliclazide to linagliptin
E. Switch gliclazide to liraglutide

E. Switch gliclazide to liraglutide

A number of factors come into play with respect to management of this patient's blood glucose. His occupation as a lorry driver precludes medication which may significantly increase his risk of hypoglycaemia. This effectively rules out insulin initiation unless he is prepared to accept any impact on his job. Insulin would also promote further weight gain and salt and water retention, and is therefore less desirable as an option. Pioglitazone promotes weight gain and fluid retention, so again is not really an option.

This leaves us with the two switching scenarios. Switching to liraglutide is preferred, because in trials GLP-1 receptor agonists have been shown to reduce HbA1c by similar levels to basal insulin, without increasing the risk of hypoglycaemia, and promote approximately 3% weight loss over a 6 month period. DPPIV inhibitors such as linagliptin are potentially less effective in reducing HbA1c than sulphonylureas and linagliptin is therefore not an option here.

Question 46 of 114

A 50-year-old woman with a history of Grave's disease is reviewed on the surgical ward some 12hrs after parathyroidectomy. She has begun suffering from episodes of carpopedal spasm and pins and needles affecting both hands and around her mouth. On examination on the ward, her blood pressure is 115/72 mmHg, and pulse is 88 beats per minute. Her serum calcium is measured at 1.85 mmol/l.

Which of the following is the most appropriate intervention?

A. Intravenous diazepam
B. Intravenous calcium
C. Intravenous magnesium
D. Oral calcium
E. Oral vitamin D

B. Intravenous calcium

Intravenous calcium gluconate is used for the acute management of hypocalcaemia

This patient has symptomatic hypocalcaemia, most likely due to an acute fall in parathyroid hormone after surgery. This is considered a medical emergency and calcium replacement IV is essential:

IV calcium gluconate is administered initially with 20 ml of 10% calcium gluconate in 50-100 ml of 5% dextrose IV, given over 10 minutes with ECG monitoring.This can be repeated until the patient is asymptomatic. It should be followed up with a calcium gluconate infusion where 100ml of 10% calcium gluconate is diluted in 1 litre of normal saline or 5% dextrose and infused at 50-100 ml/hr.

Not intervening with respect to the electrolyte disturbance risks significant sequelae including cardiac arrhythmia, diazepam is therefore

not appropriate. IV magnesium is most useful where hypocalcaemia is resistant to correction, and oral interventions would take too long to elevate serum calcium levels.

Question 47 of 114

A 23-year-old female presents with worsening acne and a marked increase in the development of body and facial hair which she finds very distressing. She is also overweight and is markedly stressed by her physical appearance and the development of stretch marks over her abdomen. She has tried multiple hair removal techniques with only mild success.

On examination, she has a body mass index of 28 kg/m², coarse hair over the anterior and posterior part of her chest and under her chin. Her Blood Pressure is 135/90mmHg.

Her lab results are as follows:

9:00 am Cortisol	345 nmol/l (170 700 nmol/l)
LH	17 iU/l (1 20 iU/l)
Basal FSH	7.1 iU/l (1.0 8.8 iU/l)
DHEAS	545 µg/dl (31 228 µg/dl)
Prolactin	160 mU/l (<360 mU/l)
17 OH Progesterone	1025 ng/dl (<80 ng/dl)
Testosterone	3.9 nmol/l (0.9 3.1 nmol/l)

Ultrasound abdomen and pelvis reveals two cysts in the right ovary.

Which of the following is the most appropriate treatment option for her condition?

A. Combined oral contraceptive pill
B. Finasteride
C. Surgical resection of the ovarian cysts
D. Reverse circadian rhythm steroids
E. Metformin in combination with spironolactone

D. Reverse circadian rhythm steroids

The diagnosis in this scenario is non-classical congenital adrenal hyperplasia which manifests in adolescence/adulthood. It is caused by a deficiency of the enzyme 21 hydroxylase in the steroid biosynthetic pathway. The result is a shift in the production of steroid hormones towards the androgenic pathway. Since cortisol secretion is reduced, feedback leads to increased ACTH production and resultant hyperplasia of the adrenals. The level of the compounds that are formed prior to the action of 21 hydroxylase is increased, therefore levels of 17 hydroxyprogesterone are elevated. Due to excessive androgen production, there is virilization and hirsutism.

Treatment involves steroids given in reverse circadian rhythm, i.e. a higher dosage at night and a lower dose in the morning.

The rationale behind this approach is due to the pathophysiology of CAH. The adrenal hyperplasia and the over-secretion of adrenal androgens are due to excessive ACTH production. When steroids are given in higher doses at night, ACTH is suppressed and the normal physiological steroid peak in the morning is also reduced.

Cysts in the ovaries are a common finding on routine ultrasound and do not necessarily represent polycystic ovarian syndrome.

Question 48 of 114

A 45-year-old man was commenced on 500mg metformin BD for type 2 diabetes mellitus as he was unable to meet his target HBA1c of 6.5% 6 months ago. He is now presenting for his regular follow up and feels well. He states that he has been compliant with his metformin, maintaining a healthy diet and exercising more regularly. His latest HBA1c is 7.3% and the rest of his investigations are normal.

What is the next best step to manage his diabetes?

A. Increase his dose of metformin
B. Add pioglitazone
C. Make no change to his current treatment
D. Add a sulfonylurea
E. Advise to increase frequency of exercise

A. Increase his dose of metformin

As per NICE guidelines on the management of type 2 diabetes in adults, at an HBA1c of 7.3% and a relatively low dose of metformin, the first step would be to increase his dose of metformin. His aim should still be an HBA1c of 6.5% on metformin.

Additional medications are only required if his HBA1c is 7.5% or higher, therefore the addition of pioglitazone or a sulfonylurea is incorrect. Whilst regular exercise is important, it is unlikely to be sufficient at this stage.

Click on the link below for more information and the algorithm for blood glucose lowering therapy in adults with type 2 diabetes:

Question 49 of 114

A 28-year-old woman has presented with a 5 month history of weight loss (despite an increase in appetite), tremor, loose bowels, and heat intolerance. She has otherwise been well and her only significant family history is that her brother has alopecia areata. She tells you that she had a positive pregnancy test last week and is awaiting her booking appointment. On examination, she appears anxious and her heart rate is 105 beats/minute. She has a tremor when her arms are outstretched and her eyes appear large. She also has a goitre. The rest of her examination is unremarkable. Her blood results find hyperthyroidism.

Which of the following medications are most suited to treat her hyperthyroidism?

A. Propylthiouracil
B. Carbimazole
C. Radioactive iodine
D. Carbimazole and Levothyroxine
E. Levothyroxine

A. Propylthiouracil

During early pregnancy, propylthiouracil should be used. The block and replace strategy is not advised as it can lead to problems in the fetus and radioactive iodine is contraindicated. Please see the link below for more information:

Question 50 of 114

A 55-year-old male presented to his general practitioner with a 4-month history of sweating, fatigue and daytime tiredness. He attributed his tight rings to 'fluid retention' and has been experiencing worsening headaches and deterioration in his vision.

He was diagnosed with acromegaly and underwent surgery for this condition 1 month ago. He has been feeling well since and has not experienced any new symptoms.

Which of the following investigations would be most useful for monitoring the effect of his therapy?

A. MRI pituitary
B. Echocardiography
C. Growth hormone levels
D. Insulin-like growth factor levels
E. Oral glucose tolerance test

D. Insulin-like growth factor levels

Insulin-like growth factors (IGF-1) have a long half-life and so is a useful measurement to assess growth hormone secretion and therefore screen for acromegaly and monitor the response to therapy. Serum IGF-1 is the most feasible parameter to assess clinical disease activity, in everyday practice in the outpatient clinic setting. It is monitored every 6 months, and growth hormone (GH) levels are done yearly.

Note: Oral glucose tolerance test (plus GH levels) is not helpful for patients receiving somatostatin analogues, and for patients receiving GH receptor antagonist therapy, only IGF-1 should be measured.

Question 51 of 114

A 66-year-old male was admitted with agitation and confusion, worsening over the past 1 week. His past medical history includes hypertension, ischaemic heart disease and chronic back pain. His daughter noticed that he had lost about 1 stone in weight (currently weighs 71 kg), has been more tired over the last month and that he has been drinking a lot more water. This was associated with the development of urinary incontinence.

On examination, his heart rate was 108 beats/min, blood pressure was 95/42 mmHg, saturations were 94% on air and respiratory rate was 20/min. He is confused, with a Glasgow Coma Scale of 14 and appeared dehydrated.

Blood results are as follows:

Na +	125 mmol/l
K +	5.0 mmol/l
Urea	18 mmol/l
Creatinine	180 µmol/l
Blood glucose	34 mmol/l

Venous blood gas was done and showed the following:

pH	7.32
pCO2	4.6 kPa
pO2	6.1 kPa
HCO3	17mmol/l
BE	-3.6 mmol/l

Which is the most important treatment?

A. Intravenous 0.9% sodium chloride
B. 10 units of human actrapid stat
C. Start insulin sliding scale at 6 units/hr
D. Calcium gluconate
E. Intravenous 1.8% sodium chloride

A. Intravenous 0.9% sodium chloride

Characteristic features of hyperosmolar hyperglycaemic state (HHS) includes:

- high osmolality, often 320 mosmol/kg or more
- high blood glucose, usually 30 mmol/L or more severely dehydrated and unwell.
- without significant hyperketonaemia or acidosis

HHS typically occurs in the elderly and is often the first presentation of Type 2 Diabetes Mellitus.

Using the blood results, osmolality can be calculated with the formula 2(Na+K) +glucose+urea

Goals of treatment include:

- Normalise the osmolality
- Replace fluid and electrolyte losses
- Normalise blood glucose
- Prevention of complications: Arterial or venous thrombosis/cerebral oedema

Fluid replacement must commence first; an initial insulin bolus of 0.15 U per kg may be given once infusions are underway. Fluid replacement alone with 0.9% sodium chloride solution will result in falling blood glucose. Insulin treatment prior to adequate fluid replacement may result in cardiovascular collapse as water moves out of the intravascular space, with a resulting decline in intravascular volume.

Patients with HHS are often exquisitely sensitive to insulin and require much lower doses than in diabetic ketoacidosis (DKA).The recommended insulin dose is a fixed rate intravenous insulin infusion (FRIII) given at 0.05 units per kg per hour (e.g. 4 units/hr in an 80 kg man) is used

Beware of rapid correction of hyponatraemia, may lead to cerebral pontine myelinolysis

Question 52 of 114

A 53-year-old woman comes for review in the general medical clinic. She was diagnosed with type 2 diabetes mellitus six months ago after having developed fatigue and polyuria. She also has hypothyroidism but no other comorbidities. She was started on metformin 500mg twice daily struggled to cope due to gastrointestinal side effects such as diarrhoea.

What is the most appropriate action?

A. Reduce to metformin 500mg once daily
B. Change to dipeptidyl peptidase-4 inhibitor
C. Trial of modified release metformin
D. Change to sulfonylurea
E. Change to pioglitazone

C. Trial of modified release metformin

The correct answer trial of modified release metformin. NICE guidelines advise to offer standard release metformin as the first-line treatment for type 2 diabetes and to gradually increase the dose to minimise the risk of gastrointestinal side effects. If gastrointestinal side effects are not tolerated, then a trial of modified release metformin would be appropriate. If metformin is not tolerated at all then a dipeptidyl peptidase-4 inhibitor, sulfonylurea or pioglitazone would be indicated.

Question 53 of 114

A 24-year-old female presents with one week of progressive and persistent double vision. She reports increasing tiredness at all times of day over the past 2 months and occasional chest tightness associated with palpitations. She has no past medical history. She was also adopted and unaware of any family history. On examination, you find a loss of left eye abduction, right eye upwards gaze, right eye adduction. Systemic examination also reveals bilateral clammy hands and a heart rate of 120 per minute, irregular.

Which test is most likely to be diagnostic?

A. Autoimmune screen
B. Thyroid function tests
C. CT thorax
D. Anti-acetylcholine receptor antibodies
E. 12 lead ECG

B. Thyroid function tests

This patient presents with systemic symptoms and a complex ophthalmoplegia, the diagnosis of thyroid eye disease, secondary to Graves disease, is most likely. The important test would be thyroid function tests and also MRI of her orbits, which would almost certainly demonstrate retro-orbital and extraocular muscle inflammation. The severity of the patient's eye disease needs to be assessed: the most frequently used criteria was developed by the American thyroid association, which spells out helpfully NO SPECS

- Class 0 No symptoms or signs
- Class I Only signs, no symptoms (lid retraction, stare, lid lag) Class II Soft tissue involvement
- Class III Proptosis
- Class IV Extraocular muscle involvement Class V Corneal involvement
- Class VI Sight loss (optic nerve involvement)

Any patient presenting with eye movement weaknesses that cannot be explained by isolated or multiple cranial nerve palsies is called complex ophthalmoplegia. The differentials include myasthenia gravis, mononeuritis multiplex, thyroid eye disease, Kearns-Sayre syndrome, complex progressive external ophthalmoplegia, Miller-Fisher syndrome and botulinum poisoning.

Question 54 of 114

You are asked to review a 55-year-old male surgical for the fourth time in seven days with persistent hyperkalaemia on his blood tests. He has been admitted for 5 weeks under the surgeons following AP resection of sigmoid carcinoma complicated by a superficial wound infection requiring a vacuum dressing. In all of the previous three medical reviews, the patient presented with a serum potassium of greater than 6.5 mmol/l and was treated with insulin-dextrose and calcium gluconate.

His past medical history includes type 2 diabetes mellitus, non-alcoholic steatohepatitis and neuromyelitis optica diagnosed 6 years ago and stable on the last review 2 months ago. His regular medications include gliclazide 80mg BD, Lantus (insulin glargine) 15 units OD, prednisolone 15 mg OD and baclofen 10mg QDS. During this review, he is alert and comfortable, blood pressure measures 135/82 mmHg, heart rate 90/min and sinus.

His blood tests are as follows:

Hb	121 g/l
Platelets	334 * 10 /l9
WBC	8.2 * 10 /l9
Na +	136 mmol/l
K +	6.9 mmol/l
Urea	7.5 mmol/l
Creatinine	110 µmol/l
CRP	4 mg/l
Renin	Raised
Aldosterone	Decreased

Blood gases show the following:

pH	7.24
PaO2 (air)	15.8 kPa
PaCO2	2.2 kPa
Bicarbonate	24 mmol/l

Urinary pH = 6.2

A repeat CT abdomen and pelvis demonstrates appropriate wounding healing with no local collections at the resection site. No other abdominal pathology is noted.

What is the most likely diagnosis?

A. Type 1 renal tubular acidosis
B. Type 2 renal tubular acidosis
C. Type 4 renal tubular acidosis
D. Waterhouse-Friderichsen syndrome
E. Addisonian crisis

C. Type 4 renal tubular acidosis

Refractory hyperkalaemia in a patient with a prolonged illness should raise suspicions for adrenal insufficiency. Note that mineralocorticoid deficiency can occur with hyperkalaemia alone without hyponatraemia. In this case, the serum demonstrates a metabolic acidosis with normal bicarbonate and urinary pH greater than 5.5, ruling out type 1 and 2 renal tubular acidosis (RTA). Waterhouse-Friedrichsen syndrome is caused by adrenal haemorrhage, classically secondary to tuberculosis or meningococcal infection, which if present, should be visualised on CT imaging. He does not demonstrate circulatory collapse, abdominal pain or nausea suggestive of Addisonian crisis. Type 4 RTA, causing a failure of the sodium-potassium antiporter is thus the most appropriate diagnosis, in the context of a patient with chronic steroid use and hence predisposition for adrenal insufficiency during acute severe illness.

Question 55 of 114

A 62-year-old male, recently emigrated from India, presents with 5 day history of feeling generally unwell. His niece, who has accompanied him to hospital, denies a history of recent productive cough, diarrhoea or vomiting or dysuria. Her uncle had been gradually increasingly malaised over the past 5 days and not eating and drinking well. He has no known past medical history. On examination, he has dry mucous membranes and cool peripheries, his JVP is +1cm above the angle of Louis. Heart sounds, chest and abdomen are unremarkable. Urine dip and chest radiograph are awaited. His blood tests are as follows:

WBC	16 * 10 /l9
Neutrophils	14.8 * 10 /l9

Na +	152 mmol/l
K +	3.7 mmol/l
Urea	22 mmol/l
Creatinine	208 µmol/l
CRP	38 mg/l
Glucose	38 mmol/l
Ketones	2.8 mmol/l

Arterial blood gases:

pH 7.31

PaO2 20.2 kPa PaCO2 3.0 kPa Bicarbonate 16 mmol/l Lactate 4 mmol/l

What is the unifying diagnosis?

A. Diabetic ketoacidosis (DKA)
B. Lactic acidosis
C. Hyperosmolar hyperglycaemic state (HHS)
D. Urinary tract sepsis
E. Chest sepsis

C. Hyperosmolar hyperglycaemic state (HHS)

This patient has presented with dehydration and non-specific symptoms and a diagnosis difficult to diagnose clinically. However, his biochemistry is diagnostic: calculation of his osmolality, (2[Na + K] + urea+ glucose) reveals an osmolality greater 371.4mosmol/kg. He is likely to present acutely with undiagnosed type 2 diabetes mellitus and a diagnosis of HHS, previously known as HONK.

There is no evidence to suggest uro or chest sepsis but an infectious underlying decompensating trigger should be considered with a prescription of broad spectrum antibiotics. Although lactate is mildly raised, this is likely secondary to intravascular dehydration and hypoperfusion of internal organs. Lactic acidosis alone does not account for the full biochemical picture. Ketones are present and the patient is mild acidotic. However, be aware that neither is sufficiently significant for a diagnosis of DKA.

Question 56 of 114

A frail 82-year-old gentleman was brought in by his daughter, who found him on the floor in his flat. He had tripped in a mechanical and had been unable to get back up, lying on the floor for the past 3 days. On examination, he appears extremely dehydrated but has no specific focal weakness, systemic examination is unremarkable. He has sustained no musculoskeletal injuries. His blood tests are as follows:

Na +	168 mmol/l
K +	6.0 mmol/l
Urea	24 mmol/l
Creatinine	260 µmol/l (baseline 107 three months ago)
Creatinine kinase	11,000 mmol/l

ECG shows normal sinus rhythm at 99/ minute.

You diagnose him with rhabdomyolysis and an acute kidney injury, likely of a pre-renal cause. Intravenous fluid rehydration is initiated with intravenous 5% dextrose. You ask your colleague to check the patient's blood tests in 12 hours.

What is the aim of correcting the patient's hypernatraemia?

A. Reduce blood sodium to under 145 mmol/l as quickly as possible
B. Reduce blood sodium by 0.5mmol/hr. The drop in 12 hoursshould be no greater than 6 mmol/l
C. Reduce blood sodium by 1mmol/hr. The drop in 12 hours shouldbe no greater than 12 mmol/l
D. Aim for blood sodium above 145 mmol/l
E. Blood sodium does not require monitoring if intravenous fluids is running, CK falling and renal function improving

B. Reduce blood sodium by 0.5mmol/hr. The drop in 12 hoursshould be no greater than 6 mmol/l

The correct answer is to **reduce blood sodium by 0.5mmol/hr. The drop in 12 hours should be no greater than 6 mmol/l**. In cases of hypernatraemia, it is important to correct the patient's sodium levels gradually to avoid complications such as cerebral oedema and central pontine myelinolysis. Aiming for a reduction rate of 0.5mmol/hr ensures that the correction is slow and controlled, allowing the body to adapt safely.

The first option, **reduce blood sodium to under 145 mmol/l as quickly as possible**, is incorrect because rapidly correcting hypernatraemia can lead to severe neurological complications such as osmotic demyelination syndrome (central pontine myelinolysis). It is crucial to correct sodium levels slowly and cautiously.

The second incorrect option, **reduce blood sodium by 1mmol/hr. The drop in 12 hours should be no greater than 12 mmol/l**, suggests a faster rate of correction than recommended. This approach also increases the risk of complications, particularly in elderly patients who may have reduced physiological reserve and increased vulnerability to rapid changes in serum electrolyte concentrations.

The fourth option, **aim for blood sodium above 145 mmol/l**, contradicts the goal of treating hypernatraemia. A normal serum sodium level ranges from approximately 135-145 mmol/l; thus, maintaining a level above this range would not address the patient's underlying issue.

Finally, the last option, **blood sodium does not require monitoring if intravenous fluids are running, CK falling and renal function improving**, is incorrect because regular monitoring of blood sodium levels during treatment for rhabdomyolysis and acute kidney injury is essential. Monitoring allows clinicians to assess response to therapy, adjust fluid management as needed, and detect potential complications early.

Question 57 of 114

A 17-year-old girl is brought into the emergency department by her mother. The patient appears terrified after she experienced an episode on waking earlier in the morning when she could not move at all for 2 hours. This was her second episode. She reports no loss of consciousness and was aware throughout the episode. She has no other past medical history documented. She is not aware of a previous episode of epilepsy. On examination, her heart sounds and breath sounds are unremarkable. Neurological examination demonstrated no abnormalities. She has normal dentition and her body mass index is 19.5 kg/m². A 12 lead ECG demonstrated a jerky baseline with flat T waves.

What is the most likely diagnosis?

A. Partial or absence seizures
B. Guillain-Barre syndrome
C. Botulinum toxicity
D. Myasthenia gravis
E. Hypokalaemia

E. Hypokalaemia

The patient describes episodes of periodic paralysis and the ECG characteristics are consistent with that of hypokalaemia. The underlying diagnosis is a rare familial condition of skeletal muscle ion channels called hypokalaemic periodic paralysis, which tends to develop in childhood and adolescence. Attacks last hours and the neurological examination is usually unremarkable in between attacks. The average potassium on diagnosis is 2.4 mmol/L . Diagnosis is often made clinically in association with low potassium but genetic testing can help if known mutations are present. 1

Question 58 of 114

You are the medical doctor on an acute medical unit. A 28 year old, adopted female with a history of recurrent kidney stones has been referred by her GP with hypokalaemia (K+ 2.6 mmol/l). She feels well in herself and denies any vomiting or diarrhoea. Her observations are normal and blood results are as below:

Na +	141 mmol/l
K +	2.6 mmol/l
Bicarbonate	18 mmol/l
Urea	5.0 mmol/l
Creatinine	67 µmol/l
Anion gap	13 mEq/L

What is the most likely cause of her presentation?

A. Liquorice poisoning
B. Renal tubular acidosis type 1
C. Renal tubular acidosis type 2
D. Renal tubular acidosis type 3
E. Renal tubular acidosis type 4

B. Renal tubular acidosis type 1

The patient has presented with a history of recurrent kidney stones, hypokalaemia, acidosis and a normal anion gap. This is typical for renal tubular acidosis type 1. Renal tubular acidosis type 2 would present with similar biochemical features but is more unlikely to have a history of kidney stones. Renal tubular acidosis type 3 is an obsolete concept. Renal tubular acidosis type 4 is associated with hyperkalaemia.

Liquorice poisoning would cause hypokalaemia and hypernatraemia but is not associated with the other biochemical abnormalities found here.

Question 59 of 114

A 40 year old man presents to the Emergency Department with tiredness and dizziness (worse on standing) which has been ongoing for the past few months. He had a past medical history of epilepsy and mentions that he has had 'brain surgery' in the past. He is on some medications but cannot remember the names. He has no allergies.

On assessment, he has no focal neurological deficit and cardiovascular/respiratory examination is normal. Observations show a blood pressure of 135/90 mmHg (dropping to 105/82 mmHg on standing), a heart rate of 67 beats per minute, a temperature of 36.2 degrees, oxygen saturations of 94% on air and a respiratory rate of 18/min. Given his medical history, you opt to keep this gentleman in the short stay unit for observation overnight.

Baseline blood tests are as follows:

Hb	125 g/l
WCC	9.2 x109/l
Plt	290 x109/l
CRP	10 mg/l
Gluc	3.9 mmol/l
Na+	138 mmol/l
K+	5.8 mmol/l
Ur	7.2 mmol/l
Cr	100 µmol/l
TSH	0.4 mU/l
T4	5.0 pmol/l

Given the above, what is the most likely underlying diagnosis?

A. Hypopituitarism
B. Hypothyroidism
C. Acromegaly
D. Pheochromocytoma
E. Medication side effects

A. Hypopituitarism

This gentleman has hypopituitarism following 'brain surgery'. Though the details of this are obscured in the question, it is likely that removal of a pituitary mass with trans-sphenoidal surgery. This is exhibited by fairly non-descript symptoms coupled with some underlying evidence of lack of anterior pituitary hormones: low BP/dizziness/postural hypotension, high/normal K+, low/normal Na+ and low/normal blood glucose all indicate lack of cortisol due to low ACTH; the low/normal temperature and heart rate and the tiredness steer you towards low thyroxine level due to lack of TSH.

This gentleman needs assessment of his pituitary function. This can be done in many ways. A baseline pituitary hormone profile can be quite useful; however the most definitive tests involve assessing dynamic pituitary function. The insulin stress test (coupled with TRH and GnRH tests) creates a hypoglycaemic effect in the body and the response of the pituitary (cortisol surge) is measured. However, inducing hypoglycaemia in epileptics, such as this gentleman, is contraindicated. Therefore the next best investigation is the glucagon stimulation test which mimics hypoglycaemia in the body and causes a fake stress on the pituitary, therefore being safe to use in epileptics.

Question 60 of 114

A 55-year-old man was seen in the Emergency Department after a fainting episode. He describes a history of fatigue and nausea. His past medical history includes type 2 diabetes mellitus and HIV infection and he admits that he has not been compliant with any medications, including his anti-retroviral therapy.

On examination, his pulse was 65 beats per minute and regular, blood pressure 90/62 mmHg and respiratory rate 26 breaths per minute.

Investigations:

Haemoglobin	14.0 g/dL (13.0-18.0)
White cell count	4 x 10^9/L (4-11)
Platelets	150 x 10^9/L (150-400)
Sodium	130 mmol/L (135-145)
Potassium	5.8 mmol/L (3.5-5.0)
Creatinine	80µmol/L (60-110)
Glucose	4.0 mmol/L (4.0-7.8)

What is the most appropriate next step management step?

A. Salbutamol nebuliser
B. Broad-spectrum antibiotics
C. Sliding scale insulin infusion
D. Intravenous hydrocortisone
E. Restart anti-retroviral medications

D. Intravenous hydrocortisone

Adrenal insufficiency affects approximately 10% of patients with HIV, commonly due to cytomegalovirus (CMV)-related necrotising adrenalitis. It is the failure of the adaptive immune system seen in HIV infection, and particularly AIDS, that increases susceptibility to CMV infection, and thus

risk of CMV-associated adrenal failure.

The immediate management of hypoadrenalism involves rapid steroid replacement; fluid resuscitation would also be a priority.

There is no firm evidence here to suggest bacterial infection, and the hyperkalaemia should resolve with appropriate management of the hypoadrenalism.

Question 61 of 114

An 18-year-old male with no prior medical history is admitted to the resuscitation room of Emergency Department where you are asked to assess him. He is noted to be hypotensive, tachycardic and febrile. Further assessment highlights a widespread, non-blanching, purple rash. He is diagnosed with meningococcal septicaemia and is treated appropriately. Nevertheless, his condition deteriorates further when he also develops Waterhouse-Friderichsen syndrome.

Which of the following sets of blood results would be most consistent with his condition at this point?

A. Na+ 147mmol/L, K+ 5.8mmol/L, Glucose 7.5mmol/L
B. Na+ 147mmol/L, K+ 3.0mmol/L, Glucose 2.0mmol/L
C. Na+ 129mmol/L, K+ 5.8mmol/L, Glucose 2.0mmol/L
D. Na+ 147mmol/L, K+ 5.8mmol/L, Glucose 11.5mmol/L
E. Na+ 129mmol/L, K+ 5.8mmol/L, Glucose 11.5mmol/L

C. Na+ 129mmol/L, K+ 5.8mmol/L, Glucose 2.0mmol/L

Waterhouse-Friderichsen syndrome is defined as adrenal failure due to bleeding into the adrenal glands (otherwise referred to as haemorrhagic adrenalitis) and is most commonly caused by meningococcal septicaemia.

The biochemical pattern which results is analogous to that seen in an Addisonian crisis, and so you would expect to see hyponatraemia, hyperkalaemia and hypoglycaemia

Question 62 of 114

A 34-year-old woman presents for the first time as being 12 weeks pregnant. She has a past medical history of Hashimoto's thyroiditis. Her current medication is levothyroxine 100 mcg and recent blood tests reveal a TSH level of 1.0 mU/l.

What is the most appropriate management with regards to her levothyroxine treatment, given her recent diagnosis of pregnancy?

A. Reduce her levothyroxine dose by an average of 25 mcg
B. Increase her levothyroxine dose by an average of 100 mcg
C. Increase her levothyroxine dose by an average of 25-50 mcg
D. Reduce her levothyroxine dose by an average of 50 mcg
E. Keep her levothyroxine dose unchanged

C. Increase her levothyroxine dose by an average of 25-50 mcg

In patients currently on levothyroxine who become pregnant, thyroid function tests should be assessed at 6-8 weeks gestation, 16-20 and at 28-32 weeks. During pregnancy, the average thyroxine requirements typically increase by 25-50 mcg. The patient normally returns to their original dose of levothyroxine straight after delivery.

Question 63 of 114

A 42 year-old man presents to his GP with a 3 month history of increasing anxiety. On further questioning, he has lost 6 kg of weight over the past 2 months and has been experiencing increased bowel movements and diarrhoea.

Blood tests are performed and reveal:

Hb	14.2 g/dL	
Platelets	210 * 10 /l9	
WBC	6.9 * 10 /l9	
TSH	0.08 mu/l	
Free T4	17.4 pmol/l	
Total triiodothyronine (T3)	13.4 nmol/l	Normal range (4.0-8.3 nmol/l)

What is the most appropriate treatment?

A. Reassurance
B. Carbimazole
C. Radio-iodine
D. Surgery
E. Propranolol

B. Carbimazole

The diagnosis in this scenario is triiodothyronine thyrotoxicosis. A small subset of those patients experiencing thyrotoxicosis (roughly 5%) have isolated triiodothyronine thyrotoxicosis. As with other types of thyrotoxicosis, carbimazole is the main initial treatment for the condition.

Question 64 of 114

A 19-year-old pharmacy student is admitted to hospital after collapsing while at work. She denies biting her tongue or becoming incontinent during the collapse and was groggy but alert on coming around. At the time, a first aider measured her blood glucose to be 1.5 mmol/l. The patients mother reports that the patient has had 2 other episodes of collapse.

The students observations include a blood pressure of 127/77 mmHg, pulse of 81 bpm, and oxygen sats of 97%.

What is the best first-line investigation?

A. Glucose, c-peptide and insulin
B. Morning c-peptide
C. Evening c-peptide
D. Computed tomography (CT) scan of the abdomen
E. Oral glucose tolerance test

A. Glucose, c-peptide and insulin

Measuring blood glucose, insulin and c-peptide allows the differentiation between the causes of hypoglycaemic attacks, including insulinoma, insulin or sulphonylurea misuse.

Question 65 of 114

A 76 year old woman was admitted to hospital after presenting to the Emergency Department with shortness of breath, productive cough and palpitations. A chest x-ray demonstrated a left lower lobe pneumonia and ECG showed atrial fibrillation with a fast ventricular response. Initial management included intravenous antibiotics, intravenous fluids and oral digoxin loading.

Two days after admission, the patient's condition had significantly improved and she was able to start mobilising on the ward. The palpitations that she had been experiencing at presentation had also ceased. Following review by the Senior House Officer on the ward round, a repeat ECG was requested when demonstrated that the patient had cardioverted back to sinus rhythm. Digoxin therapy was subsequently held.

To investigate for an underlying cause of atrial fibrillation, thyroid function tests were added to blood tests from admission, with results as listed below.

Haemoglobin	125 g / dL
White cell count	13.7* 10 /l9
Neutrophils	11.9* 10 /l9
Platelets	351 * 10 /l9
Urea	4.6 mmol / L
Creatinine	130 micromol / L
Sodium	139 mmol / L
Potassium	3.6 mmol / L
C-reactive protein	105 mg / L
Thyroid stimulating hormone	0.25 microU / L

T4 free serum	14.1 pmol / L
T3 free serum	7.4 pmol / L

What is the most appropriate next investigation to assess deranged thyroid function tests?

A. Thyroid ultrasound
B. Thyroid peroxidase antibody levels
C. Repeat TFT in 6 weeks
D. Thyroid scintiscanning
E. Thyroglobulin antibody levels

C. Repeat TFT in 6 weeks

Sick euthyroid is common in unwell, elderly patients and often needs no treatment

Any acute and severe illness may alter thyroid hormone deiodination through the effects of cytokines and result in various changes in levels of TSH, fT3 and fT4. Low TSH levels in hospitalised patients are three times more likely to be due to this effect than to hyperthyroidism.

It is therefore best to avoid thyroid function testing around the time of an acute illness unless there is good clinical evidence of a primary thyroid illness. If TFT remain deranged following recovery from acute illness then further investigation to assess for thyroid disease can be considered.

Question 66 of 114

A 45-year-old lady is admitted to hospital with abdominal pain and malaise. She has no past medical history and takes no regular medications or supplements. Bloods tests show:

Ca 2+ 2.70 mmol/l+
PO4 1.2 mmol/l+
Creatinine 60 µmol/l

Chest X-ray - normal appearances

She denies taking any medications or supplements. Her chest X-ray is normal in appearance,and renal function normal. You ring the GP and find out her calcium was also slightly raised 8 years ago.

What is the most likely diagnosis?

A. Secondary hyperparathyroidism
B. Malignancy with bony metastasis
C. Primary hyperparathyroidism
D. Familial hypocalciuric hypercalcaemia
E. Sarcoidosis

D. Familial hypocalciuric hypercalcaemia

PO4 would normally be low in primary hyperparathyroidism. Her renal function is normal excluding secondary hyperparathyroidism. Sarcoidosis is unlikely with a normal CXR. This leaves malignancy or familial hypocalciuric hypercalcaemia. Although malignancy is possible her raised Ca2+ 8 years makes familial hypocalciuric hypercalcaemia more likely.

Question 67 of 114

A 68-year-old man was admitted with fever and difficulty passing urine. He had a positive urine dip and was commenced on intravenous coamoxiclav for a lower urinary tract infection. He also complains of 'burning pain' and ascending numbness from his feet.

His past medical history includes poorly controlled diabetes with retinopathy, hypertension, ischaemic heart disease and hypercholesterolaemia. His medications include metformin, gliclazide, linagliptin, ramipril, aspirin and simvastatin.

Examination of the cardio-respiratory system is normal. He has mild suprapubic tenderness on abdominal palpation. On examination of his feet, you notice decreased sensation and loss of proprioception.

What would be the first-line treatment for the pain the patient is suffering with?

A. Codeine
B. Paracetamol
C. Tramadol
D. Ibuprofen
E. Duloxetine

E. Duloxetine

First line treatment in diabetic neuropathy is with amitriptyline, duloxetine, gabapentin or pregabalin

The patient is suffering from diabetic neuropathy. This is evident from the pain/numbness experienced in the glove-stocking distribution and the abnormal neurology elicited from his feet. First-line treatment for diabetic neuropathic pain includes duloxetine.

Codeine and paracetamol are both analgesics, but would not be of benefit in neuropathic pain.

Tramadol is used as a rescue treatment in acute flares of pain associated with diabetic neuropathy and is not first-line management.

Ibuprofen is also an analgesic that will offer limited benefit in neuropathic pain. The patient is also taking aspirin and dual NSAID use is not advisable due to the risks of gastric ulcers and bleeding.

Question 68 of 114

A 65-year-old man who is known to have type 2 diabetes mellitus presents for advice. He is a Muslim and is considering fasting for Ramadan. His diabetes is currently controlled with a combination of diet and metformin 500mg tds. Looking at his records the last HbA1c was 6.4% (46 mmol/mol).

If he decides to fast during Ramadan, what is the most appropriate advice to give regarding his metformin?

A. Metformin should be stopped
B. Metformin 1.5g after sunset
C. Metformin 500mg before sunrise, 1g after sunset
D. Metformin 500mg after sunset
E. Metformin 1g before sunrise, 500mg after sunset

C. Metformin 500mg before sunrise, 1g after sunset

During Ramadan, one-third of the normal metformin dose should be taken before sunrise and two-thirds should be taken after sunset

The correct answer is **Metformin 500mg before sunrise, 1g after sunset**. This is because during Ramadan, individuals who fast are required to refrain from consuming food and drink from sunrise to sunset. In order to maintain adequate glycaemic control while minimizing the risk of hypoglycaemia, it is recommended that metformin be taken in a divided dose - a smaller dose before sunrise (pre-dawn meal or Suhoor) and a larger dose after sunset (evening meal or Iftar). This allows for better distribution of the medication throughout the day while still adhering to the fasting requirements.

Metformin should be stopped is incorrect because stopping metformin abruptly may result in poor glycaemic control and increased risk of hyperglycaemia. It is important for patients with diabetes to continue their medications during Ramadan, with appropriate adjustments made under medical supervision.

Metformin 1.5g after sunset is not the best option because taking the entire daily dose at once can increase the risk of gastrointestinal side effects and may not provide optimal glycaemic control throughout the day. Dividing the dose as mentioned earlier helps maintain better blood glucose levels during fasting hours.

Metformin 500mg after sunset is also incorrect because this dosing regimen significantly reduces the total daily dose of metformin compared to his current regimen, which may lead to inadequate glycaemic control. Additionally, it does not account for any pre-dawn medication intake.

Finally, **Metformin 1g before sunrise, 500mg after sunset** is not ideal as it provides a higher dose before sunrise than necessary. The recommended approach is to take a smaller dose before sunrise and a larger one after sunset in order to minimize potential hypoglycaemia during fasting hours while maintaining adequate glycaemic control.

Question 69 of 114

A 28-year-old male with a history of epilepsy, for which he is taking carbamazepine and has not had any seizures for the last two years, presents with irritability and nausea for the last 2 weeks. His girlfriend says that he is often confused and seems to be lost most of the time. He takes alcohol occasionally and smokes ten to twelve cigarettes per day.

On examination, he is irritable but conscious and alert. Clinical examination revealed eczema over the face, shins and extensor surfaces of the forearms and a tattoo on the right shoulder. There was no evidence of any peripheral oedema.

Lab reports were as follows:

Hb	150 g/l
MCV	81 fl
MCH	31 pg
WBC	9 * 10 /l9
Plt	250 * 10 /l9
Urea	3.2 mmol/l
Creatinine	75 µmol/l
9:00 am Cortisol	345 nmol/l (170 700 nmol/l)
TSH	2.4 mU/l
Total T4	102 nmol/l (68 174 nmol/l)
Na+	119 mmol/l
K+	4.2 mmol/l

Which of the following would be the most appropriate initial management option?

A. Fluid restriction to 500 - 1000 ml daily
B. Demeclocycline 600 - 1200 mg daily
C. IV hypertonic saline
D. Intranasal desmopressin twice daily
E. Hydrochlorothiazide 12.5 mg daily

A. Fluid restriction to 500 - 1000 ml daily

The diagnosis in the scenario described above is SIADH secondary to carbamazepine treatment. The findings of eczema are coincidental and have no bearing on the diagnosis. However, in the management the presence of skin lesions such as eczema may pose difficulty in opting for the use of demeclocycline which may cause photosensitive rashes.

The initial treatment option for SIADH is by fluid restriction which, under most circumstances, will correct the biochemical abnormalities in almost every case.

Demeclocycline is indicated if fluid restriction is poorly tolerated or ineffective.

Hypertonic saline is indicated only when the syndrome is very severe, acute and symptomatic.

Intranasal desmopressin is used for the management of diabetes insipidus, and thiazide diuretics are only rarely used as alternative agents in diabetes insipidus.

The causes of SIADH are as follows

1. Tumours
    - Small cell CA of the lung
    - Prostate
    - Thymus
    - Pancreas
    - Lymphoma
2. Pulmonary Lesions
    - Pneumonia
    - Tuberculosis
    - Lung abscess
3. CNS Causes
    - Meningitis
    - Tumours
    - Head injury
    - Subdural haematoma
    - Cerebral abscess
    - SLE
    - Vasculitis
4. Metabolic Causes
    - Alcohol withdrawal
    - Porphyrias
5. Drugs
    - Carbamazepine
    - Chlorpropamide

- Cyclophosphamide
- Vincristine
- Phenothiazines

Question 70 of 114

A 52 year old lady presents complaining of polydipsia and polyuria. She has a background of hypertension, hypercholesterolaemia and bipolar affective disorder and a strong family history of diabetes - she is unsure which type.

Results show the following:

Na+	131mmol/l
urine osmolality	287mOsmol/kg (300 - 900mOsmol/kg)
plasma osmolality	287mOsmol/kg (285 - 295mOsmol/kg)

Which of the following is the most likely explanation for this lady's symptoms?

A. Psychogenic polydipsia
B. Syndrome of inappropriate anti-diuretic hormone (SIADH)
C. Diabetes insipidus
D. Diabetes mellitus type 1
E. Hyponatraemia

A. Psychogenic polydipsia

Although this lady is biochemically hyponatraemic, this is unlikely to be the cause of her symptoms.

With a past medical history of bipolar affective disorder, although not stated in the question, there is a good chance she may be on lithium which predisposes her to developing nephrogenic diabetes insipidus. However with this diagnosis, we would expect a much lower urine osmolality and a higher plasma osmolality. She would also have a normal to high serum sodium. The opposite would indicate a diagnosis of SIADH (serum hypo-osmolality and high urine osmolality).

The osmolality results here reflect a diagnosis of psychogenic polydipsia with a low urine osmolality and a low end of normal plasma osmolality.

Question 71 of 114

You are reviewing a 57 year-old gentleman in the diabetes outpatient clinic. He has type 2 diabetes mellitus and is currently taking metformin 850mg three times a day and gliclazide 80mg once daily.

On further questioning he admits having frequent hypoglycaemic episodes at night that distress him as he lives alone. His BMI is calculated at 30.3 kg/m², HbA1c 7.8% (62 mmol/mol) and his co-morbidities include congestive cardiac failure.

How would you change his diabetic treatment?

A. Stop gliclazide, start insulin
B. Add exenatide
C. Add sitagliptin to current regimen
D. Stop gliclazide, start pioglitazone
E. Stop gliclazide, start sitagliptin

E. Stop gliclazide, start sitagliptin

The NICE guidance on the management of type 2 diabetes mellitus:

- This gentleman has been started on metformin and a sulphonyurea as first line therapy.
- He is having frequent hypoglycaemic episodes secondary to his sulphonylurea and yet control remains poor, HbA1c 7.8% (62 mmol/mol)
- Pioglitazone is contraindicated due to his congestive cardiac failure.
- A DPP-4 inhibitor such as sitagliptin would be a sensible option, the sulphonylurea should be stopped to prevent hypoglycaemia.

Question 72 of 114

A 56-year-old lady presents with a 3 month history of abdominal pains, low mood and constipation. Past medical history includes hypertension and depression following the death of her husband 2 years ago. Routine blood tests are performed by the GP and upon review the patient is referred into hospital.

Blood tests are as below:

Hb	100 g/l	Na +	135 mmol/l
Platelets	230 * 10 /l $_9$	K +	4.7 mmol/l
WBC	10 * 10 /l $_9$	Urea	6 mmol/l
Calcium (adjusted)	2.96 mmol/l	Creatinine	110 µmol/l
Phosphate	1.35 mmol/l	CRP	30 mg/l
Albumin	35 g/L		

Which diagnostic test should be performed first?

A. Parathyroid hormone level
B. Myeloma screen
C. CT chest, abdomen and pelvis
D. Urinary calcium levels
E. Skeletal X-ray

A. Parathyroid hormone level

The two main causes of hypercalcaemia are primary hyperparathyroidism and malignancy. Parathyroid hormone level will help to differentiate between these two main differentials and help guide further investigations.

Question 73 of 114

You are seeing a 58-year-old man in clinic who has been referred by his GP with resistant hypertension despite treatment with ramipril, amlodipine, indapamide and bisoprolol. He looks comfortable at rest.

Observations are as follows: temperature 36.5ºC, blood pressure 182/125 mmHg, heart rate 88/min, respiratory rate 16/min, saturations 97% on air

Investigations are as follows:

Na +	148 mmol/l
K +	2.9 mmol/l

Urea	6.5 mmol/l
Creatinine	92 µmol/l
Renin	Low
Aldosterone (supine)	High
Aldosterone (prolonged standing)	Increase from supine levels

Blood gases:

PaO2	11.2kPa
PaCO2	5.2kPa
pH	7.49
HC03-	32 mmol/l
BE	+4

What is the single most likely diagnosis?

A. Adrenal hyperplasia
B. Conn's adenoma
C. Malignant hypertension
D. Phaeochromocytoma
E. Renal artery stenosis

A. Adrenal hyperplasia

All of the above diagnoses could cause hypertension. This patient has a hypokalaemic alkalosis and low renin. The differentials are therefore Conn's adenoma and adrenal hyperplasia. To differentiate between the two look at the aldosterone on standing. In this case, the aldosterone increases on prolonged standing, therefore, the diagnosis is adrenal hyperplasia. In Conn's adenoma, the aldosterone would stay the same or drop on standing.

In malignant hypertension and renal artery stenosis, the renin would be high.

Question 74 of 114

A 46-year-old woman presents with polyuria. She has also noticed a swelling over the front of her neck and has been having headaches, sweating, and palpitations. Observations are as follows: heart rate 115 beats per minute, blood pressure 125/85 mmHg, respiratory rate 14 breaths per minute, SpO2 99% (on air), temperature 37.1ºC.

Blood results are as follows:

Calcium	2.9 mmol/L	(2.1-2.6)
Phosphate	0.6 mmol/L	(0.8-1.4)
PTH	4.8 pmol/L	(1.6 - 6.9)
TSH	4.2 mU/L	(0.5-5.5)
Free T4	14.2 pmol/L	(9.0 - 18)

What investigation is required?

A. Radioactive iodine uptake scan
B. Serum calcitonin
C. Serum thyroglobulin
D. Thyroid peroxidase antibodies
E. Thyroid stimulating hormone receptor antibodies

B. Serum calcitonin

MEN type II: obtain a thyroid ultrasound and serum calcitonin to exclude medullary thyroid cancer

Serum calcitonin is correct. The patient most likely has multiple endocrine neoplasia (MEN) type 2. The presence of hypercalcemia with

an 'inappropriately normal' PTH is suggestive of primary hyperparathyroidism. The patient also likely has a phaeochromocytoma due to the classic triad of episodic headache, sweating, and tachycardia. The neck swelling is concerning for medullary thyroid cancer which should be investigated with a thyroid ultrasound and serum calcitonin.

Radioactive iodine uptake scan is incorrect. Medullary thyroid cancer cells do not absorb iodine, and therefore radioiodine scans are not used to diagnose this type of cancer.

Serum thyroglobulin is incorrect. Papillary and follicular thyroid cancer release thyroglobulin which can therefore be used as a tumour marker for these types of cancer. However medullary cell cancer does not produce thyroglobulin. Medullary cell cancer cells produce calcitonin which can therefore be used as a tumour marker.

Thyroid peroxidase antibodies is incorrect. The patient is euthyroid and thus investigation for hypothyroidism with thyroid autoantibodies is not required.

Thyroid stimulating hormone receptor antibodies is incorrect. The patient is euthyroid and thus investigation for hypothyroidism with thyroid autoantibodies is not required.

Question 75 of 114

You are asked to review a 43-year-old man in theatre recovery who has developed a fever and tachycardia post-operatively. He is previously fit and well, does not smoke and drinks alcohol only occasionally. He had fallen the previous night and suffered a distal radius fracture and has just undergone a open reduction and internal fixation under general anaesthetic. During anaesthesia he received 4mg ondansetron and 8mg dexamethasone for post-operative nausea and 10mg morphine for pain. He denies feeling unwell and has no symptoms suggestive of intercurrent infection.

On examination his heart rate is 130 beats/min and irregular, his blood pressure is 135/74 mmHg and his temperature is 39.4ºC. His chest is clear to auscultation, his abdomen soft and non-tender and there is no rash or meningism. His right forearm is in plaster, but is not particularly painful and his fingers are warm and have normal sensation.

Hb	130 g/l
Platelets	460 * 10 /l9
WBC	10.5 * 10 /l9
Na +	138 mmol/l
K +	4.1 mmol/l
Urea	5.1 mmol/l
Creatinine	95 µmol/l
C-reactive protein	1 mg/L
Thyroid stimulating hormone	<0.02 mIU/L
Cortisol	45 µg/dL

What is the most appropriate initial treatment?

A. Carbimazole
B. Hydrocortisone
C. Propranolol
D. Broad spectrum antibiotics

E. Crystalloid infusion

C. Propranolol

The diagnosis here is thyrotoxicosis as a presenting feature of hyperthyroidism. Infection is unlikely given the normal clinical examination and normal CRP. During initial treatment of thyrotoxicosis it is important to treat hypoadrenalism first - if present - in order to not precipitate a addisonian crisis. However, this patient has no features in the history to suggest pre-existing Addisons disease, he has normal electrolytes and the suppressed cortisol can be explained by the perioperative use of dexamethasone. Initial treatment of thyrotoxicosis should focus on sympathetic storm suppression using beta blockade. Anti-thyroid medications - i.e. carbimazole - take up to six weeks to take full effect and are not useful in the acute scenario.

Question 76 of 114

A 54-year-old woman attends with severe headaches. The headaches have been recurrent and are precipitated by exercise or drinking coffee. She has also felt intolerant to heat and is generally nauseated.

She has a past medical history of anxiety, constipation, and recurrent renal stones.

On examination, she is diaphoretic and tachycardic. There is no focal neurology. She has palpable cervical lymphadenopathy.

Heart rate 120 beats per minute, blood pressure 180/102, SpO2 98% room air, respiratory rate 18 breaths per minute, temperature 37.3ºC.

Hb	120 g/L	Male: (135-180)Female: (115 - 160)
Platelets	222 * 10 /L 9	(150 - 400)
WBC	6.5 * 10 /L 9	(4.0 - 11.0)
Calcium	3.3 mmol/L	(2.1-2.6)
Thyroid-stimulating hormone (TSH)	4.5 mU/L	(0.5-5.5)
Free T4	14 pmol/L	(9.0 - 18)
LH	10 IU/L	(5-25)
FSH	0.6 IU/L	(0.3-10)
Capillary blood glucose	5.4 mmol/L	(3.9-7.8)

Given the likely diagnosis, which of the following options should form part of the workup for this patient?

A. MRI head
B. MRI pancreas
C. Serum calcitonin
D. Skeletal survey
E. Ultrasound adrenals

C. Serum calcitonin

MEN type II: obtain a thyroid ultrasound and serum calcitonin to exclude medullary thyroid cancer

Here we have a patient presenting with the classic triad of pheochromocytoma - hypertension, headaches, and sweating alongside cervical lymphadenopathy and hypercalcemia. The most common cause of hypercalcemia is primary hyperparathyroidism. The unifying diagnosis here is MEN type 2A. Up to 100% of patients with MEN type 2A have medullary thyroid cancer at some time in their life and so testing for this is essential.

Serum calcitonin is correct. Alongside a thyroid ultrasound scan, serum calcitonin is required as part of the workup for MEN type II to look for medullary thyroid cancer. Unlike other thyroid cancers, thyroid function tests are usually normal, and presenting features are usually cervical lymphadenopathy or thyroid lumps.

MRI head is incorrect. MEN type 1 is associated with the '3 Ps' - pituitary tumours (most commonly prolactinomas), pancreatic tumours, and parathyroid tumours. In this case, a prolactinoma is unlikely given the normal LH and FSH (prolactin usually suppresses LH and FSH levels) and there are no signs or symptoms of pancreatic tumours and blood glucose levels are normal. MEN type II is more likely. MRI head imaging to look for pituitary tumours is not indicated.

MRI pancreas is incorrect. In this case, there are no signs or symptoms of pancreatic tumour, and blood glucose is normal. This is, therefore, not the most appropriate option.

Skeletal survey is incorrect. This would form part of the workup for multiple myeloma. Although there is hypercalcemia here, there are no other signs or symptoms of multiple myeloma. A diagnosis of multiple myeloma would not explain the headaches precipitated by activity and caffeine. This is, therefore, not the correct answer.

Ultrasound adrenals is incorrect. Adrenal imaging is important in order to assess for pheochromocytoma. Ultrasound, however, is not the appropriate imaging modality - CT scanning is used.

Question 77 of 114

A 52-year-old lorry driver was referred to the secondary care diabetes mellitus by his GP with poorly controlled diabetes. He was diagnosed with type 2 diabetes mellitus six years ago. Unfortunately, he has not engaged with lifestyle interventions and his diabetes has been poorly controlled since diagnosis. He has since developed diabetic nephropathy and proliferative retinopathy. Other than diabetes he has a past medical history comprising of ischaemic heart disease, hypertension, hypercholesterolaemia, osteoarthritis and gout. At the point of referral he was prescribed aspirin 75mg OD, ramipril 10mg OD, simvastatin 40mg ON, naproxen 250mg BD, co-codamol 8/500 2tabs QDS, lansoprazole 30mg OD, metformin 500mg TDS, gliclazide 80mg BD and pioglitazone 30mg OD. He smoked 20 cigarettes per day and consumed 15 units of alcohol per week.

Examination revealed an obese gentleman with a BMI of 38 kg/m. His blood pressure was 148/88 mmHg. Cardiovascular examination revealed the presence of normal heart sounds and a JVP of 3cm. Examination of the respiratory, abdominal and neurological systems was unremarkable. Investigations reveal the following results:

Na +	136 mmol/l
K +	5.1 mmol/l
Urea	14.1 mmol/l
Creatinine	148 µmol/l
Total cholesterol	5.1 mmol/l
HDL cholesterol	1.3 mmol/l
HbA1c	68 mmol/mol (8.4%)

What is the next best step management step?

A. Commence exenatide
B. Commence sitagliptin
C. Commence insulin glargine
D. Increase dose of pioglitazone
E. Commence orlistat

A. Commence exenatide

This gentleman fulfills the NICE guidelines criteria for exenatide therapy. He is obese with poorly controlled diabetes despite multiple oral hypoglycaemic agents and is suffering from other morbidities relating to his weight. Exenatide may help facilitate weight loss and does not cause hypoglycaemia. Insulin is the other option here, but it may lead to weight gain. Furthermore, owing to his occupation as a lorry driver he would require a period of close observation of his blood sugar levels prior to being allowed to resume HGV driving. Exenatide is therefore the best option.

Question 78 of 114

A 68-year-old gentleman was admitted to the medical admissions unit with increasing drowsiness. He had been diagnosed with primary small cell carcinoma of the lung six months ago and had declined curative chemotherapy. At the time of diagnosis there was no evidence of metastasis, and his past medical history comprised of chronic obstructive pulmonary disease, ischaemic heart disease, hypertension, hypercholesterolaemia and depression.

His wife had taken him to the Emergency Department having noted that he had been increasingly drowsy over the last few hours, as well as developing new onset confusion. He had otherwise been relatively well prior to the admission. He had consulted his GP about new onset generalised aches and pains within the last four weeks for which his GP had commenced Oramorph solution PRN. Since then he had developed abdominal pain which the GP had diagnosed as secondary to opiateinduced constipation and he was accordingly prescribed lactulose 15ml BD with partial relief of his symptoms. There had been no evidence of weakness or numbness and no evidence of speech impairment; as far as his wife was aware he had taken the prescribed dose of oramorph. His drug history comprised of oramorph solution 10mg BD, paracetamol 1g QDS, dihydrocodeine 60mg QDS, lactulose 15ml BD, aspirin 75mg OD, atorvastatin 20mg ON, bisoprolol 2.5mg OD, Ramipril 2.5mg OD and furosemide 40mg OD.

Examination revealed a drowsy gentleman with a GCS of 12 (E 3 M5 V4). His blood pressure was 102/68, heart rate 58bpm, respiratory rate 10/min, oxygen saturations of 95% on air and temperature 36.6ºC. Examination of his cardiovascular and respiratory systems were unremarkable. Examination of his central nervous system revealed the presence of normal sized pupils; he was not compliant with formal neurological examination but no other focal neurological signs were found. There was no evidence of neck stiffness and Kernig's sign was negative. He was not cooperative with an abbreviated mental state examination.

Which investigation is most likely to be diagnostic of the underlying cause?

A. Urgent CT head scan
B. Urgent serum liver function and calcium profile
C. Urgent isotope bone scan
D. Urgent septic screen
E. Urgent PET scan

B. Urgent serum liver function and calcium profile

This gentleman has developed signs of life-threatening hypercalcaemia, having manifested previous potential symptoms including new onset abdominal pain and constipation. This question is asking what the single next most important investigation should be, and whilst most of the above options are relevant, candidates are asked to discriminate from what is deemed to be the next essential management should be. It is likely that he has developed metastasis to his spine thus whilst an isotope bone scan may be an important part of his overall management, it is not as relevant in the acute setting. Likewise, it is likely that a septic screen and CT head would be performed, but clinically speaking there is little definite evidence of acute raised intracranial pressure or sepsis. There is no indication at present that he is suffering from opiate toxicity and therefore IV naloxone should not be administered at this stage.

Question 79 of 114

A 33-year-old female is brought into the emergency department as a stand-by. She has felt unwell for the past 2 weeks. She describes lethargy, light-headedness and occasional shortness of breath. More recently she developed urinary frequency and dysuria. She received a domiciliary visit from her general practitioner 2 days ago and was prescribed trimethoprim for a possible urinary tract infection.

On arrival, she appears pale and clammy. Her peripheries are cold. Her observations reveal oxygen saturations of 94% on air, respiratory rate 28/min, heart rate 117/min, blood pressure 65/30 mmHg.

She has a past medical history of type 1 diabetes, uterine fibroids and hypothyroidism.

Initial investigations reveal:

White cell count	17.8 *109/l
Haemoglobin	97 g/l
Mean cell volume (MCV)	103.7 fL
Sodium	134 mmol/l
Potassium	4.9 mmol/l
Urea	7.0 mmol/l
Creatinine	120 µmol/l
Bilirubin	45 µmol/l
Alanine transaminase (ALT)	1051 U/l
Albumin	16 g/l
C-reactive protein (CRP)	71 mg/dL
Glucose	9.1 mmol/l

Urinalysis: ++protein, ++blood, +++leukocytes, ++nitrites, trace ketones

She is given intravenous fluids. Her blood pressure is 82/45 mmHg after a total of 3 litres of fluids. She is started on intravenous amoxicillin and gentamicin.

What is the next step in her management?

A. 1000mls intravenous colloid fluid
B. Intravenous hydrocortisone
C. Intravenous noradrenaline
D. Intravenous albumin
E. Intravenous meropenem

B. Intravenous hydrocortisone

This patient has an autoimmune diathesis: she has type 1 diabetes and hypothyroidism. Her macrocytic anaemia also raises the possibility of undiagnosed pernicious anaemia. She is therefore at very high risk of primary adrenal insufficiency (Addison's Disease). IV hydrocortisone is a potentially life-saving treatment and should be given immediately. It is not contra-indicated in the presence of sepsis. Her biochemistry is not classical for Addison's, however, this does not exclude the diagnosis. Her abnormal LFTs are in keeping with a shocked liver. Intensive care review should be sought and she may well require inotropes but treatment of potential adrenal insufficiency is the immediate priority.

Question 80 of 114

A 67-old-lady presents a week after a recent episode of *Campylobacter* gastroenteritis with increased thirst and passing urine. She estimates that she drinks 9-10 glasses of water or tea a day which is twice what she would normally have. In addition, her urine is clear in colour. She feels low in energy with easy fatigue but is able to continue her voluntary work at a local shop. She reports occasional cramps in her legs after standing for some time. She takes indapamide for hypertension but is otherwise well.

On examination, she appears euvolaemic with moist mucosa and no oedema. Her abdomen is soft with bowel sounds . She appears slow, overweight and lethargic. with a There are no neck masses or eye changes. Blood pressure is 105/90mmHg and heart rate is 67/min.

Na +	138mmol/l
K +	2.6 mmol/l
Urea	4.2 mmol/l
Creatinine	53 µmol/l
Calcium (corrected)	2.34 mmol/l (2.2-2.6)
TSH	3.2 mU/L (normal range 0.4-4.0)
Cortisol	400 nmol/l (normal range 129-450)
Water deprivation test	pending

What is the likeliest cause of these symptoms?

A. Addison's disease
B. Hypothyroidism
C. Syndrome of inappropriate ADH secretion
D. Psychogenic polydipsia
E. Hypokalaemia

E. Hypokalaemia

Hypokalaemia is a rare cause of polyuria and polydipsia

This lady has polydipsia and polyuria. Of the above options, only hypokalaemia and psychogenic polydipsia are recognised causes of these symptoms. Hyperthyroidism can rarely cause polydipsia but not hypothyroidism. Addison's disease can cause hypercalcaemia which can cause these symptoms but her calcium is normal. Psychogenic polydipsia can occur but this is usually a chronic problem. This lady has presented very soon after her gastroenteritis and has evidence of hypokalaemia making this a rare but the likeliest cause of her symptoms. Her indapamide is probably contributing to the persistent hypokalaemia and should be stopped.

Question 81 of 114

A 58 year-old man with type 2 diabetes mellitus presents to his GP for his annual review. His diabetes is currently being treated with metformin 500mg twice daily and gliclazide 40mg once daily. He complains that recently he has had several episodes of hypoglycaemia and this has been affecting him at work where his job is as a construction worker. His most recent HbA1c was 66 mmol/mol. Apart from the diabetes, he also has a past medical history of stage 2 heart failure, hypertension and mild chronic obstructive pulmonary disease.

What is the most appropriate treatment change?

A. Stop gliclazide and start exenatide
B. Stop gliclazide and start sitagliptin
C. Commence daily insulin
D. Stop gliclazide and start glibenclamide
E. Stop gliclazide and start pioglitazone

B. Stop gliclazide and start sitagliptin

This patient's hypoglycaemic attacks is most likely due to the sulfonylurea he is currently on. NICE guidance recommends that if the patient is suffering hypoglycaemic side effects from the sulfonylurea they can be commenced on a combination of metformin and a glitazone or metformin and a DPP-4 inhibitor. However, due to this patient's heart failure, pioglitazone is not suitable, therefore a combination of metformin and sitagliptin is the most appropriate option. Glibenclamide has long-acting hypoglycaemic effects so would not be appropriate in this scenario.

Question 82 of 114

You receive a phone call from a general practitioner regarding a 50 yearold man who has had thyroid function tests performed for a history of weight loss. There is no history of illicitly taking levothyroxine. His results show : TSH 0.01 mIU/L, T4 8.5 ug/dL.

You should advise which of the following:

A. Admit for urgent MRI head
B. Repeat the bloods and include parathyroid hormone (PTH)
C. Add on T3 as this may represent T3 toxicosis
D. Start radio-iodine treatment immediately
E. Start thyroxine replacement

C. Add on T3 as this may represent T3 toxicosis

In approximately 5% of patients with clinical and biochemical hyperthyroidism T3 may be elevated prior to T4. This is known as T3 toxicosis.

When both free hormones are normal but TSH is low, the term subclinical thyrotoxicosis can be applied.

Radio-iodine treatment should never be started without discussion with the patient and is only used prior to carbimazole in certain circumstances. An MRI head would be part of the work up for secondary hyperthyroidism. PTH would not be useful in this circumstance and starting thyroxine would not be a good idea! T3 toxicosis is treated in the same manner as T4 hyperthyroidism.

Question 83 of 114

A 19-year-old girl was seen in clinic with lethargy, weakness worsening over the past 4 weeks. She also complains of recurrent muscle cramps in her legs, causing her to have trouble sleeping. On further questioning she admits to urinary frequency, passing urine up to ten times a day, and feels dehydrated all the time. She also mentions that her periods which were usually irregular, have stopped 4 months ago.

On examination, she is thin, with a body mass index of 17kg/m². Her heart rate is 88 bpm and blood pressure is 108/86 mmHg.

C Reactive protein	2mg/l
Haemoglobin	158 g/l
White cell count	7.6 x 10^9/L
Na+	136 mmol/l
K+	2.9 mmol/l
Urea	7.2 mmol/l
Creatinine	108 µmol/l
Corrected calcium	2.42 mmol/l

Venous blood gas result

pH	7.532
Bicarbonate	37mmol/l

What would be the next most useful investigation?

A. Transvaginal ultrasound (TVUS) of the ovaries
B. Urine diuretic assay
C. Early morning cortisol
D. Serum renin and aldosterone levels
E. Fasting blood glucose levels

B. Urine diuretic assay

Patients with hypokalaemia, metabolic alkalosis and a normal - low blood pressure the following differentials should be considered - diuretic abuse, Bartter's syndrome, Gitelman's syndrome. Of the three, the most common cause is diuretic abuse, especially in young women, and can be ruled out with a urine diuretic assay.

Bartter's syndrome presents early in life, with classical features of triangular facies, polyuria, polydipsia and renal failure. Serum renin and aldosterone levels are high despite a low or normal blood pressure. Urine calcium may be raised, and renal stones are a common feature. In Gitelman's syndrome patients may present later on in adulthood, but have a milder disease course or may be asymptomatic compared to patients with Bartter's syndrome. Hypomagnesaemia and hypocalciuria differentiates Gitelman's syndrome from Bartter's syndrome.

This patient may need other further investigations such as a TVUS, early morning cortisol and fasting blood glucose tests to rule out other conditions, but in view of her biochemistry profile, a urine diuretic assay would be the most useful next investigation to perform.

Question 84 of 114

A 50-year-old woman is reviewed in your follow-up endocrinology clinic. She was referred one year ago by her GP, having noticed a nodule in the front of her neck. Before noticing this nodule, she had suffered from a 6- month history of worsening diarrhoea, and occasional flushing. She was found to have a mutation of the RET proto-oncogene and underwent a thyroidectomy, followed by external beam radiotherapy. Her only current medication is levothyroxine.

Which of the following is the most useful test in monitoring for recurrence of this woman's disease?

A. Alfa fetoprotein
B. Calcitonin
C. Thyroglobulin
D. Thyroid stimulating hormone
E. Thyroxine

B. Calcitonin

Medullary thyroid cancer - calcitonin is used for screening, prognosis and monitoring

Medullary thyroid carcinoma originates from the C cells, or parafollicular cells, which secrete calcitonin. It makes up approximately 5% of thyroid cancers. It may be sporadic, but in 25% of cases, it is associated with RET oncogene, as part of multiple endocrine neoplasia type 2 (MEN2). Despite testing positive for RET oncogene it is possible that other features of MEN2, such as hyperparathyroidism or phaeochromocytoma, do not manifest. Such is the case with this woman.

Also, note that radioiodine was not used in the treatment of her disease. Unlike other thyroid cancers, it is of little benefit in medullary thyroid cancer. Her symptoms of flushing and diarrhoea, are typical of medullary

cancer and are caused by calcitonin and calcitonin gene-related peptide. Papillary and follicular cancers are more commonly asymptomatic.

Calcitonin is used for screening, prognosis and monitoring in medullary thyroid cancer. CEA is often used in conjunction with calcitonin but is not an option here.

Thyroglobulin is used in monitoring follicular thyroid cancer.

Thyroxine (T4) and thyroid-stimulating hormone (TSH) are both typically normal in thyroid cancer. This woman has had her thyroid gland removed, and is taking levothyroxine. As such, she would likely be having her TSH monitored. Unlike in other thyroid cancers, TSH suppression is not appropriate for the treatment of medullary thyroid cancer and so thyroxine replacement is monitored similarly to that of hypothyroidism, with a target TSH within the normal range. Her TSH may be monitored, but not to detect recurrence of medullary thyroid cancer.

Alfa fetoprotein is not of use in monitoring thyroid cancer.

Question 85 of 114

A 51-year-old woman is reviewed in clinic. Two months ago she underwent an operation to remove a medullary thyroid cancer after presenting with diarrhoea and a neck lump. Genetic testing showed she has a mutation of the RET oncogene. The patient reports being well and there are no signs of local recurrence on examination.

What is the most appropriate test to monitor for recurrence?

A. Thyroglobulin
B. Thyroid transcription factor-1
C. Chromogranin
D. Calcitonin
E. S100 protein

D. Calcitonin

Medullary thyroid cancer - calcitonin is used for screening, prognosis and monitoring

S100 protein is used in patients with melanoma. Thyroglobulin is used in other types of thyroid malignancy but not medullary thyroid cancer.

Question 86 of 114

A 45-year-old female presents with a 2-year history of headache and visual blurring. When initially presenting to her GP 2 years ago her blood pressure was found to be 235/160mmHg. Subsequently, despite maximal doses of four anti-hypertensives, including 50mg spironolactone, her blood pressure remains poorly controlled. Her latest blood tests demonstrate the following:

Na +	140 mmol/l
K +	2.9 mmol/l
Urea	5.8 mmol/l
Creatinine	78 µmol/l
CRP	2 mg/l
Serum ambulatory renin activity	0.34 pmol/L @ 3-4 hours (normal range 0.8-3.5 pmol/ml/hr)
Serum ambulatory aldosterone	2052 pmol/L@ 3-4 hours (normal range 100-800)

A CT adrenal reveals a right adrenal mass of 2.5cm diameter. The patient is keen to take away the underlying problem.

What is the most appropriate next management step?

A. Add amiloride
B. Add eplerenone
C. Increase spironolactone to 100mg OD
D. Adrenal vein sampling
E. Right adrenalectomy

D. Adrenal vein sampling

Adrenal venous sampling (AVS) can be used to distinguish between unilateral adenoma and bilateral hyperplasia in primary hyperaldosteronism

The patient presents with poorly controlled hypertension, is hypokalaemic, hypertensive, metabolically alkalotic, has increased aldosterone and reduced renin. The underlying diagnosis is Conn's disease. The treatment involves removal of the aldosterone secreting tumour. However, those pursuing adrenalectomy must undergo adrenal vein sampling first, CT imaging alone is not diagnostic of the symptomatic side responsible for aldosterone secretion.

Question 87 of 114

A 47-year-old female presents with recurrent palpitations. She also complains of associated sweating, blurring of vision and generalized weakness. She denies any chest pain, shortness of breath or loss of consciousness. She had several similar episodes over last few months. The episodes usually occur in the morning or just before a meal. She has noticed that her symptoms improve after she eats something. She has gained weight of about 9 kg over last few months. She has a past history of an anxiety disorder but is not currently taking any regular medications. She does not smoke and only occasionally drinks alcohol. Her blood glucose at present is 4.9 mmol. You suspect insulinoma based on the history.

What is the next step in the evaluation of this patient?

A. Check insulin level
B. Check pro-insulin level
C. CT abdomen
D. Screening for sulphonylurea drugs
E. Check blood glucose during or after the episode to confirmhypoglycemia

E. Check blood glucose during or after the episode to confirmhypoglycemia

Completion of Whipple's triad is required before further investigations for insulinoma

European Neuroendocrine Tumor Society (ENETS) guidelines published in 2016 outline the diagnostic criteria for insulinomas. As symptoms of hypoglycemia can be non-specific and attributable to several different conditions, it is important to confirm hypoglycemia by checking blood glucose. Therefore, completion of Whipple's triad is required before embarking on further investigations. Whipple's triad is defined as the presence of hypoglycemic symptoms, accompanying low blood glucose,

and resolution of symptoms after correcting the blood glucose levels.

Therefore, the best answer here is 5 i.e confirming the hypoglycemia will be the next step in the evaluation of this patient.

All the other options are relevant to the diagnosis but not the best next step here.

Question 88 of 114

A 35-year-old woman is known to have a family history of multiple endocrine neoplasia type 2a. She has tested positive for the RET oncogene and is concerned as her mother had suffered from thyroid cancer. However, she is reluctant to undergo a prophylactic thyroidectomy and asked if there is another option available.

Which of the following management options would be appropriate for her case?

A. Annual monitoring of thyroid peroxidase (TPO)
B. Annual monitoring of thyroglobulin
C. Annual 5 hydroxyindoleacetic acid levels (5-HIAA)
D. Annual monitoring of calcitonin
E. Annual monitoring of chromogranin A

D. Annual monitoring of calcitonin

Calcitonin can be used as a marker to monitor for Medullary thyroid carcinoma in MEN type II in those unwilling to undergo prophylactic thyroidectomy

Calcitonin is a marker for medullary thyroid carcinoma which is associated with MEN type II.

Chromogranin A and 5-HIAA can be used as markers for neuroendocrine tumours and carcinoid syndrome respectively.

Thyroid peroxidase antibody titre can be used as a marker in autoimmune thyroid conditions such as Hashimoto's thyroiditis and Graves disease.

Thyroglobulin can be used as a marker for papillary or follicular thyroid carcinoma. For example, a rise in this marker following a thyroidectomy can indicate the recurrence of these particular cancers.

Question 89 of 114

A 28-year-old woman presents to the clinic with amenorrhea. She had a regular menstrual cycle up until 4 months ago. On examination, you note a male pattern of her growth.

Blood results are as follows:

Testosterone	2.2 nmol/L	(< 1.8)
Oestradiol	34 pg/ml	(25-75)
FSH	12 IU/L	(1.8 - 22.5)
LH	84 IU/L	(1.2-103)

What is the most likely diagnosis?

A. Androgen-secreting tumour
B. Kallmann's syndrome
C. Partial androgen insensitivity syndrome (PAIS)
D. Polycystic ovary syndrome (PCOS)
E. Turner's syndrome

D. Polycystic ovary syndrome (PCOS)

Stereotypical PCOS results:

- raised LH:FSH ratio
- testosterone may be normal or mildly elevated
- SHBG is normal to low

Polycystic ovary syndrome (PCOS) is correct. The patient has several clinical features of PCOS including secondary amenorrhea and hirsutism. The mildly elevated testosterone and raised LH:FSH ratio further support the diagnosis.

Androgen-secreting tumour is incorrect. An androgen-secreting tumour would result in a more profound increase in testosterone levels resulting in virilisation.

Kallmann's syndrome is incorrect. Kallmann's syndrome is characterised by primary amenorrhea secondary to hypogonadotropic hypogonadism. However, in this case, the biochemical profile is not in keeping with Kallmann's syndrome - which would present with low oestradiol and inappropriately low/normal LH, and FSH levels.

Partial androgen insensitivity syndrome (PAIS) is incorrect. PAIS results in a spectrum of disorders that ranges from a female phenotype with clitoromegaly and/or minimal posterior labial fusion (Lub syndrome) to genital ambiguity to an unequivocally male phenotype with a minor defect such as isolated hypospadias or azoospermia. There are no clinical features in this case to support this diagnosis.

Turner's syndrome is incorrect. Turner's syndrome is a cause of primary ovarian failure. It would present with primary amenorrhoea, rather than secondary amenorrhea seen in this case. Furthermore, the biochemical profile, in this case, is not in keeping with Turner's syndrome - which would present with low sex hormone levels and markedly raised LH and FSH levels.

Question 90 of 114

A 37-year-old woman is referred by her GP after complaining of a swelling on the anterior aspect of her neck. On examination she is found to have a 5 cm nodule on the thyroid gland that moves on swallowing.

She has a past medical history of anxiety/depression and currently takes sertaline 100mg od. Her mother was diagnosed as having hypothyroidism in her 60's.

Thyroid functions tests are shown below:

Free T4	14 pmol/l
TSH	2.6 mu/l

A fine needle aspiration of the mass is consistent with papillary thyroid cancer. There is no current evidence of metastases.

What is the most appropriate treatment?

A. Total thyroidectomy followed by radioiodine-131
B. Localised radiotherapy
C. Total thyroidectomy
D. Total thyroidectomy followed by localised radiotherapy
E. Radioiodine-131

A. Total thyroidectomy followed by radioiodine-131

The correct answer is **Total thyroidectomy followed by radioiodine- 131**. In the case of a patient with papillary thyroid cancer, the recommended treatment is total thyroidectomy followed by radioiodine- 131 therapy. Total thyroidectomy removes the entire gland, which eliminates the primary tumor and any remaining microscopic disease. Radioiodine-131 is then used to ablate any residual thyroid tissue and treat potential microscopic metastases.

Localised radiotherapy is not the most appropriate treatment in this

case because it may not effectively remove all cancerous cells. Additionally, papillary thyroid cancer is typically less sensitive to radiation compared to other types of cancers. Therefore, radiotherapy alone would be insufficient in treating the patient's condition.

Total thyroidectomy without adjuvant therapy may not be enough to treat papillary thyroid cancer as there could still be residual thyroid tissue or microscopic metastases after surgery. Radioiodine-131 helps to eliminate these remaining tissues and reduce the risk of recurrence.

Total thyroidectomy followed by localised radiotherapy is also not considered the best option for this patient because papillary thyroid cancer usually responds better to radioiodine-131 therapy than external beam radiation. Localised radiotherapy might be considered in cases where there are contraindications for using radioactive iodine or if there are gross extrathyroidal extensions that cannot be completely resected surgically.

Finally, **Radioiodine-131** alone would not be an effective treatment for a large nodule like this one since it relies on uptake by functional thyroid tissue. Without prior surgical removal of the bulk of the tumor through total thyroidectomy, it would likely have limited effectiveness in eradicating all cancerous cells.

Question 91 of 114

A 42-year-old lady comes to see you in outpatients. Incidentally, you notice that her TSH was < 0.1 mU/l on a recent blood test requested by the GP. Her only past medical history is thyroid cancer which has been resected and her only medication levothyroxine 100mcg per day.

She is otherwise asymptomatic.

What is the best course of action?

A. Stop levothyroxine
B. Continue at 100mcg per day
C. Repeat thyroid function in 6 weeks
D. Change to liothyronine equivalent dose
E. Reduce the levothyroxine as she is over treated

B. Continue at 100mcg per day

As TSH is a growth factor for many thyroid cancers it is routinely suppressed with levothyroxine. TSH and thyroglobulin should be monitored in patients with a history of thyroid cancer and decisions on the level required are usually specialist led.

High and intermediate risk patients should have their TSH suppressed bellow 0.1 mU/l and low-risk patients TSH should be 0.1-0.5 mU/l.

As she is asymptomatic and her TSH is adequately suppressed this lady should be maintained on her current dose pending specialist review.

British Thyroid Association, guidelines for the management of thyroid cancer (2014) and The American Thyroid Associations Guidelines (2009).

Question 92 of 114

A 29-year-old woman presents to the ENT clinic with hoarseness. She has found herself to be hoarse for three weeks and despite drinking plenty of fluids finds herself unable to improve. She was referred to the ENT clinic after a phone consultation with her GP. On examination she has a goitre which is not painful. On further questioning she has no symptoms consistent with an overactive or underactive thyroid. She also feels well in herself. She has no problem speaking apart from the hoarseness, and is not struggling to catch her breath.

What is the most appropriate course of action?

A. Immediate hospital admission under endocrinology
B. Two-week wait appointment with an endocrinologist
C. Routine appointment with an endocrinologist
D. Reassure and discharge back to GP
E. Arrange for bronchoscopy

B. Two-week wait appointment with an endocrinologist

Goitre with unexplained hoarseness should be referred urgently

The combination of thyroid swelling with hoarseness or change in voice which is unexplained needs urgent referral to endocrinology for likely thyroid cancer. Thyroid nodules in a child, thyroid mass with cervical lymphadenopathy or a rapidly enlarging and painless thyroid mass also need a two-week wait referral for suspect thyroid cancer. A patient with a thyroid mass and stridor may need immediate admission. Patients with a thyroid swelling and abnormal thyroid function tests, or sudden pain in a thyroid lump, can be referred non-urgently.

Question 93 of 114

A 22-year-old lady presents over a year with mild-moderate, intermittent abdominal pain. She has felt low in mood over this period and also her periods have stopped. Her history includes two previous attacks of renal calculi formation managed conservatively. She had a car crash recently, where she says that the car just 'came out of nowhere'. She is intermittently getting global headaches that can be very severe in nature but are otherwise featureless. On prompting, she tells you that she has sometimes noticed a white nipple discharge on her clothing. She has had low blood pressure and several faints over this last year and after her GP discovered a low serum cortisol level he has started her on oral hydrocortisone and referred her to your clinic. On examination today her blood pressure 130/80. She has a blistering, red rash across her lower abdomen and back. Her abdomen is largely non-tender with no palpable organomegally or peritonism. Visual fields are reduced bitemporally. Her urine dipstick shows glycosuria. The remainder of the examination is unremarkable.

Which of the following is likely to treat the underlying condition most effectively?

A. Bisphosphonates
B. Surgery
C. Cabergoline
D. Octreotide
E. Insulin

B. Surgery

The unifying diagnosis here is is multiple endocrine neoplasia (MEN) type 1. MEN1 consists of tumours of the parathyroid, pituitary, and pancreas.

This patient has symptoms of hypercalcaemia (abdominal groans, renal stones, and psychic moans of the 'bones, stones, groans, psychic moans'). This can be caused by a parathyroid tumour. She has symptoms suggestive of a non-functioning pituitary macro-adenoma causing hyperprolactinemia (amenorrhea and galactorrhoea) and hypopituitarism

presenting as Addison's from suppressed ACTH. Dopamine is the inhibitor of prolactin in the pituitary, and comes from the hypothalamus. A nonfunctioning pituitary macro-adenoma compresses the pituitary stalk, interrupting dopamine flow to the pituitary, and therefore abolishing dopaminergic inhibition of prolactin, resulting in hyperprolactinaemia. At the same time, the macro-adenoma causes hypopituitarism through local pressure effects on the pituitary itself. 76% of pituitary tumours in MEN1 are prolactinomas, with the remainder being nonfunctioning adenomas. Prolactinomas are extremely sensitive to medical management with e.g. cabergoline or bromocriptine and even shrink in size subsequently. First line treatment for nonfunctioning adenomas however is surgical removal. The gylcosuria is suggestive of hyperglycaemia, which in the context of this MEN picture suggests a glucagonoma.

The answers try and trick you into looking to manage just one of the abnormalities here in an isolated fashion, e.g. targeting the pituitary with cabergoline, the pancreas with insulin, or the parathyroid with bisphosphonates. This may may happen if you do not recognise that this is MEN. However, as the question asks what treatment is likely to treat the underlying condition, clearly insulin alone, cabergoline alone, or bisphosphonate alone will not suffice. Of the options available, only surgery can tackle all of the problems, and indeed is what most patients with MEN end up needing.

MEN is an autosomal dominant condition. MEN1 consists of tumours of the parathyroid, anterior pituitary, and pancreas:

- The parathyroid tumours cause hypercalcaemia and its symptoms (which don't forget include polyuria and polydypsia). The management of hypercalcaemia is fluid resuscitation and bisphosphonates.
- Pituitary tumours can be a prolactinoma, somatotroph adenoma (causing acromegally by secreting growth hormone), or an ACTHsecreting tumour causing Cushing's disease. There can be any combination of these, although the question will usually point you toward one particular pituitary abnormality. Remember that you are unlikely to elicit a galactorrhoea history unless you specifically ask and are sensitive about it.
- The pancreas tumours can be an insulinoma (persistent hypoglycaemia -check C-peptide which will be high in endogenous insulin secretion as opposed to exogenously given insulin e.g. in self harm/non-accidental injury, where c-peptide levels remain low.), gastrinoma (Zollinger-Ellison, presenting as refractory gastric ulcers), glucagonoma causing persistent hyperglycaemia and also a necrolytic migratory erythema (as in this case), or a VIPoma causing profuse watery diarrhoea (VIP is the antagonist to gastrin and therefore tries to get you to flush the gut out rather than hold and digest contents.

MEN II has two forms. MEN2a presents with medullary thyroid carcinoma (neck lump), parathyroid tumour (hypercalcemia) and pheochromocytoma (hypertension, flushing, tachycardia intermittently). MEN2b presents with medullary thyroid carcinoma and pheochromocytoma.

The key message for the exam is that if you are presented with a case where you suspect there is an endocrine abnormality (even just a high calcium), look again at the stem to make sure you are not missing other endocrine abnormalities being present (e.g. amenorrhoea) that might alert you to there being an underlying diagnosis of MEN. If you know a few key features of each of the possible abnormalities within each type of MEN, you should be able to pinpoint which MEN type is present.

Question 94 of 114

A 55-year-old woman attends the clinic with refractory hypertension. A systematic enquiry was otherwise unremarkable. Her 7-day ambulatory mean blood pressure is 182/94 mmHg. She takes ramipril, amlodipine, and indapamide.

Blood results are as follows:

Na +	142 mmol/L	(135 - 145)
K +	2.5 mmol/L	(3.5 - 5.0)
Bicarbonate	36 mmol/L	(22 - 29)
Urea	4.2 mmol/L	(2.0 - 7.0)
Creatinine	64 µmol/L	(55 - 120)
Renin	3.2 mU/	(5.4 - 30)
Aldosterone	820 pmol/L	(100 - 450)

A CT scan is performed:

CT abdomen with contrast	Bilateral nodular adrenal hyperplasia

What treatment is indicated?

A. Amiloride
B. Laparoscopic adrenalectomy
C. Metyrapone
D. Pituitary adenectomy
E. Spironolactone

E. Spironolactone

Primary hyperaldosteronism: manage with spironolactone

Spironolactone is correct. The patient has primary hyperaldosteronism secondary to bilateral adrenal hyperplasia. The first-line treatment for this condition is spironolactone (an aldosterone antagonist).

Amiloride is incorrect. Although this is a potassium-sparing diuretic like spironolactone, it has a very different mechanism of action. Its primary site of action is the distal tubule of the nephron where it selectively blocks sodium transport, thereby inhibiting sodium-potassium exchange. The mechanism of action of amiloride is independent of aldosterone.

Laparoscopic adrenalectomy is incorrect. Indications for this would be Conn's Syndrome (primary hyperaldosteronism secondary to a benign adrenal adenoma). The CT in this case has confirmed bilateral adrenal hyperplasia rather than an excisable adenoma.

Metyrapone is incorrect. Metyrapone is a competitive inhibitor of 11β- hydroxylation in the adrenal cortex; the resulting inhibition of cortisol (and to a lesser extent aldosterone) production leads to an increase in ACTH production which, in turn, leads to increased synthesis and release of cortisol precursors. Metyrapone can be used

to treat resistant oedema due to increased aldosterone secretion in cirrhosis, nephrotic syndrome, and congestive heart failure, however, there is insufficient evidence to recommend its use in the management of primary hyperaldosteronism.

Pituitary adenectomy is incorrect. Cushing's disease occurs due to an ACTH-secreting pituitary adenoma resulting in high levels of cortisol and aldosterone. Although this remains within the differential diagnosis, there are no other clinical features (e.g. proximal myopathy, abdominal striae, weight gain) to support a diagnosis of Cushing's.

Question 95 of 114

A 54-year-old man attends with persistent hypertension. His blood pressure is 175/101mmHg.

His past medical history includes type 2 diabetes mellitus for which he takes metformin.

Tests are arranged and the results are detailed below:

Na +	136 mmol/L	(135 - 145)
K +	3.3 mmol/L	(3.5 - 5.0)
Bicarbonate	23 mmol/L	(22 - 29)
Urea	4.5 mmol/L	(2.0 - 7.0)
Creatinine	65 µmol/L	(55 - 120)
Aldosterone	680 pmol/L	(100-450)
Renin	0.3 mU/L	(5.4-30)

A saline infusion test is completed and the results post-infusion are as follows:

Aldosterone	550 pmol/L	(100-450)

The patient is keen to undergo surgical management of his condition if possible.

What investigation would be the most reliable in confirming the location of the lesion in this case?

A. Adrenal venous sampling
B. Delayed contrast-enhanced CT adrenals
C. Fine-needle aspiration adrenal biopsy
D. MRI adrenals
E. MRI brain

A. Adrenal venous sampling

Adrenal venous sampling (AVS) can be used to distinguish between unilateral adenoma and bilateral hyperplasia in primary hyperaldosteronism

The most likely diagnosis here is hyperaldosteronism. Adrenal adenomas and adrenal hyperplasia are the most common causes. Secondary hyperaldosteronism can occur due to renin-angiotensin-aldosterone system overactivity as seen in cardiac failure, ascites, and fibromuscular dysplasia. In this case, renin is suppressed, so primary hyperaldosteronism is the most likely answer.

Adrenal venous sampling is the correct answer. This involves sampling blood from the adrenal veins and assessing the levels of aldosterone. This is the gold-standard test to distinguish between unilateral adrenal adenoma and bilateral hyperplasia and should be completed to confirm the location of the abnormality if surgery is to be considered. CT scanning can help identify lesions, however, many adrenal adenomas are non-functioning and not clinically relevant. Hormonal studies via AVS are therefore advised to confirm the location to avoid the removal of an adrenal gland unnecessarily.

Delayed contrast-enhanced CT adrenals is incorrect. Although CT scanning is a good modality to look for adrenal pathology many adrenal adenomas can be too small to pick up on CT. CT can also pick up incidental adenomas that are non-functioning and of no clinical consequence. CT is therefore not the most reliable modality to use to confirm the location of the lesion.

Fine-needle aspiration adrenal biopsy is incorrect. Adrenal adenomas are usually extremely small and not amenable to biopsy.

MRI adrenals is incorrect. Although MRI can be used, there are similar pitfalls to CT scanning as detailed above - MRI may pick up nonfunctioning adrenal adenomas or miss small adenomas.

MRI brain is incorrect. Pituitary lesions are not implicated in primary hyperaldosteronism.

Question 96 of 114

A 24-year-old nurse is admitted after collapsing at the end of a night shift. She is found to have a capillary blood glucose of 1.2mmol/L. Following administration of IV glucose, she recovers quickly.

She undergoes a CT scan of the abdomen and pelvis which shows a hypervascular lesion within the pancreas which enhances with contrast.

What further finding would be most supportive of the likely diagnosis?

A. Elevated C-peptide following administration of IV insulin
B. Low serum C-peptide level
C. Normal serum proinsulin level
D. Positive anti-GAD antibodies
E. Sulphonylurea detectable in urine

A. Elevated C-peptide following administration of IV insulin

An elevated C-peptide level following administration of IV insulin can confirm insulinoma

The correct answer is **elevated C-peptide following administration of IV insulin**. IV insulin administration should generally suppress endogenous insulin secretion and therefore C-peptide levels should fall. An inappropriate elevation in C-peptide levels should raise the suspicion of an endogenous source of insulin secretion. The CT findings are suggestive of an insulinoma.

Low serum C-peptide level is incorrect. This would be expected in cases of factitious hypoglycaemia related to insulin administration. While this patient's employment would give her access to insulin and may raise suspicion of this, the CT findings suggest an insulinoma is more likely.

Normal serum proinsulin level is incorrect. This would also be expected in a patient with factitious hypoglycaemia, whether related to insulin or sulphonylurea administration. In an insulinoma, we would expect elevated proinsulin levels, especially after a prolonged fast.

Positive anti-GAD antibodies is incorrect. These would be expected in a patient with type 1 diabetes mellitus. In the absence of treatment, this would present with hyper- rather than hypoglycaemia.

Sulphonylurea detectable in urine is incorrect. In this context, this would be suggestive of factitious hypoglycaemia related to sulphonylurea use. Again, this patient would have access through her employment but the CT findings suggest insulinoma is more likely.

Question 97 of 114

A 28-year-old woman is referred by her GP with refractory hypertension. Despite combination therapy with ramipril, amlodipine, bendroflumethiazide and atenolol, her blood pressure in clinic today is 181/105 mmHg. Some of her bloods are shown below. On direct questioning she also admits passing urine more than 10 times per day. What is the most likely diagnosis?

Na +	145 mmol/l
K +	3.0 mmol/l
Urea	6.0 mmol/l
Creatinine	71 µmol/l

What is the most likely diagnosis?

A. Phaeochromocytoma
B. Coarctation of the aorta
C. Renal artery stenosis
D. 21-hydroxylase deficiency
E. Conn's syndrome

E. Conn's syndrome

All of the answers above are causes of secondary hypertension, except for 21-hydroxylase deficiency which accounts for over 90% of congenital adrenal hyperplasia.

The high sodium and low potassium in the bloods reflects the increased levels of aldosterone produced in Conn's syndrome. Aldosterone stimulates the Na+/K+-ATPase membrane transporter in the distal convoluted tubule resulting in increased sodium reabsorption and potassium excretion. Polyuria and polydipsia can result from the kidneys inability to concentrate urine.

The diagnosis is usually confirmed using plasma aldosterone:renin ratio

Question 98 of 114

A 40-year-old woman found a neck lump that was palpable in the left lobe of her thyroid gland. She was clinically euthyroid.

TSH	3.6 (NR 0.4-5.0)
free T4	15.1 (NR 10-25)
corrected calcium	2.41 (NR 2.2-2.6)

USS: 1.7 x 1.6cm solid lesion in left lobe of thyroid with microcalcification.

What is the next step in management?

A. Fine needles aspiration cytology
B. Radio-iodine uptake scan
C. Left lobectomy
D. Calcitonin level
E. Thyroglobulin level

A. Fine needles aspiration cytology

This nodule Is suspicious for malignancy because: microcalcifications, solid lesion, and no evidence of a hot lesion (as euthyroid). The next step is therefore FNA of the nodule.

Work up for thyroid nodules is as follows:

1. Check TSH
2. TSH suppressed → Thyroid uptake scan → Cold/iso nodule → FNA cytology
3. TSH suppressed → Thyroid uptake scan →Hot nodule → No FNA required
4. 2. TSH normal/elevated → Thyroid USS → Suspicious features → FNA cytology
5. FNA cytology is then graded using the Royal College of Pathologist classification on a spectrum from benign → malignant.

Question 99 of 114

A 22-year-old Asian woman with a body mass index of 24 kg/m² presents with new onset acne, hirsutism, and weight gain. Upon further questioning, it is found that she has had irregular periods for the last two years. On examination, there is mild acne and thick hair growth on her chin and areola region. Abdominal exam is unremarkable.

What are the most likely biochemical results given the clinical findings?

A. Raised testosterone, low LH/FSH ratio, insulin resistance
B. Low testosterone, low LH/FSH ratio, insulin resistance
C. Low testosterone, raised LH/FSH ratio, insulin resistance
D. Raised testosterone, raised LH/FSH ratio, increased insulinsensitivity
E. Raised testosterone, raised LH/FSH ratio, insulin resistance

E. Raised testosterone, raised LH/FSH ratio, insulin resistance

The clinical description is consistent with polycystic ovary syndrome (PCOS). This presents with hirsutism, acne, oligo/amenorrhoea and subfertility. Biochemical findings in PCOS include insulin resistance, raised testosterone, raised LH/FSH ratio, raised prolactin and low HDL.

Question 100 of 114

A 45-year-old patient presents to the endocrinology clinic. She presented to her GP with a small palpable thyroid nodule and the GP referred her to the endocrinology clinic because of this. She has a roughly 6mm thyroid nodule which is not painful or tender which she noticed incidentally. She has had no problems with her throat or systemic features. Following the GP appointment, she has had an ultrasound which demonstrates no evidence of malignant features. She has a past medical history of post-partum depression and fibroids. She takes no regular medications.

What is the most appropriate plan?

A. Thyroid function tests
B. US guided FNAC
C. CT neck
D. MRI neck
E. Thyroid autoantibodies

A. Thyroid function tests

A new thyroid nodule does not need FNAC if the ultrasound features are benign

This patient has a small thyroid nodule. If the US features are benign in nature and there are no worrying features then a biopsy is not needed. A CT or MRI of the neck can be useful in the case of a large goitre of which the extent cannot be seen on ultrasound. Thyroid function tests would be useful to determine any level of thyroid dysfunction to exclude a toxic thyroid nodule. Autoantibodies can be useful in determining Grave's disease.

Question 101 of 114

A 69-year-old lady presents for a follow-up appointment. She had presented 3 months previously under the acute medical take with headaches, sweating, abdominal pain and wild fluctuations in blood pressure. She is currently being followed up by the appropriate surgical team and her symptoms are currently well controlled with medical treatments. On examination today, you note a lump in her anterior neck and you are given the following blood tests:

Calcium (corrected)	3.68 mmol/l
Phosphate	0.38 mmol/l
Vitamin D3	115 nmol/l (75-200 nmol/l)
Parathyroid hormone	19 pmol/l (0.8 - 8.5 pmol/l)

You have referred the patient to endocrine surgeons for neck biopsies and urgent review. Her daughter, who came with her to the consultation) is concerned she may have the same symptoms later in life.

What should you offer the daughter?

A. Reassurance
B. Genetic testing for patient and daughter
C. Offer annual follow up for surveillance
D. CT abdomen/pelvis with contrast
E. Serum bone and calcium homeostasis profile

B. Genetic testing for patient and daughter

The patient describes features consistent with phaeochromocytoma, the appropriate surgical team would be endocrine surgery, who would arrange for a resection. The new blood tests are suggestive of primary hyperparathyroidism, the neck lumps possible medullary thyroid tumour, resulting in a unifying diagnosis of MEN 2a. The daughter can undergo genetic testing for RET-mutation.

Question 102 of 114

A 25 year old woman presents to the endocrinology clinic. She is concerned because her father had a 'brain tumour' removed 2 years ago and has now been told he has another tumour in his abdomen after going to his doctor with reflux and indigestion. He has been told it might be a genetic problem and is awaiting testing. She is concerned she might also have the condition and so her GP has referred her to the clinic.

She is currently asymptomatic.

On examination there is no abnormality on the cardiovascular, respiratory, abdominal or neurological examinations.

The doctor explain that the most appropriate person to see would be the geneticist to whom her father has been referred. He asks her to obtain the details if her father is willing to provide them and says that he will refer her. In the meaning time he offers to carry out some screening blood tests.

Given the likely underlying diagnosis, which of the following is most likely to be abnormal?

A. Cortisol
B. Fasting glucose
C. Parathyroid hormone
D. Prolactin
E. Thyroid stimulating hormone

C. Parathyroid hormone

This lady's father is likely to have multiple endocrine neoplasia type 1 (MEN1) as evidenced by 2 MEN1 associated tumours, a pituitary adenoma and a gastrinoma.

Although this lady is asymptomatic, it is important she is offered genetic screening, as the condition is autosomal dominant.

Fasting glucose, parathyroid hormone and prolactin are all biochemical screening tests used for MEN1 associated tumours (insulinoma, parathyroid adenoma and pituitary adenoma respectively). However, hyperparathyroidism is by far the most common initial manifestation and will eventually develop in 90% of MEN1 patients.

Question 103 of 114

A 34-year-old woman is referred to endocrinology clinic for assessment after reporting symptoms of heat intolerance, tremors and diarrhoea to her General Practitioner. Blood tests in primary care showed evidence of thyrotoxicosis. Further assessment at clinic revealed the symptoms had been present for approximately 4 weeks. The patient had initially attributed the symptoms to the stress of caring for her new baby, who had been born 6 weeks previously. She denied any symptoms of pain on eye movements, diplopia or skin rashes.

Past medical history included only her recent pregnancy with delivery by vaginal delivery. The patient took no regular medications. There was no family history of thyroid disorders. Prior to taking maternity leave, the patient worked as a lawyer. She did not drink or smoke.

Examination revealed a small, diffuse and mildly tender goitre with no evidence of thyroid bruit. There was fine tremor of outstretched hands but no exophthalmos or proptosis. Investigations requested following clinic review are listed below.

Thyroid stimulating hormone	0.1 microU / L (reference 0.4-5.0)
T4 free serum	19.5 pmol / L (reference 8.5-15.2)
T3 free serum	8.1 pmol / L (reference 3.5-6.5)
Thyroid peroxidase antibodies	250 mU / L (reference < 150)
Erythrocyte sedimentation rate	21 ml / h

Thyroid scintiscanning (Technitium-99): no significant thyroid uptake

What is the most likely diagnosis?

A. Graves' disease
B. Toxic thyroid nodule
C. Viral thyroiditis
D. Toxic multinodular goitre
E. Post-partum thyroiditis

E. Post-partum thyroiditis

Post-partum thyroiditis is associated with elevated thyroid peroxidase antibodies but no significant uptake on thyroid scintiscanning and is, therefore, the correct answer in this case given that symptoms began approximately two weeks postpartum.

Viral thyroiditis is usually associated with an elevated ESR and negative anti-thyroid antibodies. Graves' disease, toxic multinodular goitre and a toxic thyroid nodule would be associated with different patterns of uptake on thyroid scintiscanning.

Question 104 of 114

A 43-year-old woman attends with a 1-year history of recurrent lethargy, shakiness, slurred speech, and diplopia. Symptoms most commonly develop in the mornings and then resolve after breakfast.

She has a past medical history of type 2 diabetes mellitus, alcohol excess and a functional neurological disorder.

Observations: SpO2 97% on room air, respiratory rate 22 breaths/minute, blood pressure 92/64, heart rate 116 beats per minute, apyrexial

Capillary blood glucose: 5.2

During your review, she becomes shaky and feels unwell. A repeat blood glucose is 2.4.

You send a set of blood tests and then administer 40% dextrose gel. The blood tests are reported as follows:

Insulin	43 pmol/L	(<25)
C-peptide	114 pmol/L	(<75)
Pro-insulin	23 pmol/L	(3.6-22)

What is the most likely diagnosis?

A. Alcohol-related hypoglycaemia
B. Exogenous insulin administration
C. Glimepiride use
D. Insulinoma
E. Non-insulinoma pancreatogenous hypoglycaemia syndrome

D. Insulinoma

An elevated C-peptide level following administration of IV insulin can confirm insulinoma

The most likely diagnosis here is insulinoma. This should be suspected in patients with symptoms of hypoglycaemia that improve after eating. Symptoms of hypoglycaemia can be varied and diagnosis of insulinoma can take months to years due to difficult diagnosis.

Insulinoma is correct. An insulinoma is a rare neuroendocrine tumour that secretes insulin, as a result insulin, pro-insulin, and C-peptide levels are raised.

Exogenous insulin administration is incorrect. Pro-insulin cleavage results in C-peptide and insulin which are then released from pancreatic beta cells. It is a marker of insulin secretion. C-peptide does not form part of exogenous insulin injections and so would not be raised if insulin was being administered erroneously.

Glimepiride use is incorrect. Glimepiride is a sulfonylurea and so can be associated with episodes of hypoglycaemia. It can cause a rise in insulin, C-peptide, and pro-insulin levels as seen here, however insulin levels would generally not be increased to this degree. To confirm or refute the diagnosis a supervised fast may be appropriate.

Alcohol-related hypoglycaemia is incorrect. Alcohol causes hypoglycaemia by inhibiting gluconeogenesis. In these cases, you would expect to see low levels of insulin and C-peptide in response to low blood glucose levels.

Non-insulinoma pancreatogenous hypoglycaemia syndrome is incorrect. This is a rare syndrome characterised by post-prandial hypoglycaemia episodes and a normal 72-hour fast. This is usually due to a genetic mutation and pancreatic hyperplasia. You may expect to see elevated insulin, pro-insulin, and C-peptide, but the differentiating factor here is that the hypoglycaemic episodes commonly occur after meals which would be unusual in insulinoma.

Question 105 of 114

A 32-year-old man attends as a day case for minor surgery. You are asked to see him on arrival as his blood pressure is 197/135mmHg. You organise ambulatory blood pressure monitoring which confirms an average BP of 184/111mmHg. Blood tests are sent and the results are below.

Na +	144 mmol/L	(135 - 145)
K +	2.4 mmol/L	(3.5 - 5.0)
Urea	5.2 mmol/L	(2.0 - 7.0)
Creatinine	71 µmol/L	(55 - 120)

What is the most appropriate initial treatment?

A. Amlodipine
B. Fludrocortisone
C. Oral potassium supplementation

D. Ramipril
E. Spironolactone

E. Spironolactone

Primary hyperaldosteronism: manage with spironolactone

The correct answer is **spironolactone**. Such marked hypertension in a young patient should prompt consideration of an underlying cause and the hypokalaemia would be very suggestive of primary hyperaldosteronism. This is managed initially with mineralocorticoid receptor antagonists such as spironolactone. These are associated with improvements in both his hypokalaemia and hypertension.

Amlodipine is incorrect. This is a dihydropyridine calcium channel antagonist usually used in the management of essential hypertension. It is unlikely to be effective in primary hyperaldosteronism. Other calcium channel antagonists such as verapamil may help control hypertension while awaiting the results of confirmatory tests, but amlodipine is not recommended.

Fludrocortisone is incorrect. This is a mineralocorticoid with more of a use in adrenal insufficiency. It is likely to worsen hypertension here. It can occasionally be used in a suppression test to confirm a diagnosis of primary hyperaldosteronism but has no place in treatment.

Oral potassium supplementation is incorrect. While this may lead to a temporary improvement in the patient's hypokalaemia, this is unlikely to be sustained. It will also not treat his hypertension. Such treatments may be used in other causes of hypokalaemia such as Bartter's or Gitelman's syndrome. However, these are generally associated with normotension or even hypotension.

Ramipril is incorrect. This would be an appropriate treatment for essential hypertension. However, this patient's young age should raise suspicion of a secondary cause and the hypokalaemia points to primary hyperaldosteronism as the most likely. Ramipril is unlikely to improve this patient's blood pressure significantly and can also interfere with the diagnostics in this condition.

Question 106 of 114

A 39-year-old woman has undergone a total thyroidectomy for a papillary thyroid tumour which was 3.2cm in size without lymph node or metastatic involvement and negative margins. She is reviewed in the endocrinology clinic following her treatment and would like to know how best she should be monitored for recurrence of malignancy.

What is the most appropriate monitoring?

A. Annual CT neck
B. Annual US neck
C. Annual MRI neck
D. Annual thyroglobulin
E. Annual TSH and free T4

D. Annual thyroglobulin

Following surgery and radioiodine therapy, patients with papillary thyroid cancer should be monitored with thyroglobulin annually

This patient has a very good prognosis as she has had papillary thyroid cancer which is less than 4cm in size without lymph node or other organ involvement. The most appropriate way to monitor for cancer recurrence is annual thyroglobulin. Imaging is unlikely to detect a recurrence early and is therefore inappropriate. TSH and free T4 levels should be measured frequently as this patient will need thyroxine replacement, but this is not to detect recurrence of malignancy.

Question 107 of 114

A 66-year-old man is referred to the endocrinology outpatient department with resistant hypertension and hypokalaemia. He is asymptomatic. He is on regular ramipril, amlodipine, indapamide and doxazosin. He has a further past medical history of hypercholesterolaemia. He has smoked five cigarettes daily for thirty years and drinks 2-3 bottles of wine per week. He is a non-executive director of a large multinational company.

His observations are heart rate 76 beats per minute, blood pressure 181/88 mmHg, respiratory rate 12/minute, oxygen saturations 97% on room air. He is apyrexial and appears clinically well. An examination is unremarkable other than a raised body mass index of 35.2 kg/m².

Blood tests:

Hb	141 g/L	Male: (135-180)Female: (115 - 160)
Platelets	222 * 10 /L $_9$	(150 - 400)
WBC	6.2 * 10 /L $_9$	(4.0 - 11.0)
Na $_+$	138 mmol/L	(135 - 145)
K $_+$	2.9 mmol/L	(3.5 - 5.0)
Urea	4.6 mmol/L	(2.0 - 7.0)
Creatinine	89 µmol/L	(55 - 120)
CRP	3 mg/L	(< 5)
Cortisol	589 nmol/L	(119 - 618)
Aldosterone: renin ratio	increased	

A CT of the abdomen demonstrates bilateral adrenal enlargement and adrenal vein sampling demonstrates the production of excess aldosterone bilaterally.

What is the most appropriate treatment?

A. Chemotherapy
B. IV hydrocortisone
C. Radiotherapy
D. Spironolactone
E. Surgery

D. Spironolactone

Primary hyperaldosteronism: manage with spironolactone

Spironolactone is the correct answer. Primary hyperaldosteronism caused by bilateral adrenal hyperplasia is treated by a mineralocorticoid receptor antagonist such as spironolactone.

Chemotherapy is the wrong answer. The first-line treatment for bilateral adrenal hyperplasia causing primary hyperaldosteronism is

a mineralocorticoid receptor antagonist such as spironolactone. If a patient had disseminated malignancy caused by an adrenal carcinoma, then this may be an option.

IV hydrocortisone is incorrect. This is the treatment for acute hypoadrenalism i.e. an Addisonian crisis. The patient's cortisol is within normal limits and he has no clinical features of hypoadrenalism.

Radiotherapy is incorrect. This type of treatment may be indicated for adrenal carcinoma. However, cancer is more likely to be a unilateral mass-like lesion, rather than bilateral diffuse enlargement.

Surgery is incorrect. This would be the correct option if the patient had a unilateral adrenal adenoma secreting excess aldosterone. This is not the case here.

Question 108 of 114

A 36-year-old woman is referred to the hypertension clinic with difficult to control hypertension. She describes having occasional aches and pains which mainly affect their arms and legs. They have also noticed an increase in their urinary frequency and urgency. Her blood pressure reading is taken at the clinic and reads as 175/95 mmHg.

They are currently being treated with amlodipine and lisinopril for hypertension.

Bloods tests taken prior to attending the clinic are shown below.

Na +	136 mmol/L	(135 - 145)
K +	4.3 mmol/L	(3.5 - 5.0)
Bicarbonate	23 mmol/L	(22 - 29)
Urea	6.5 mmol/L	(2.0 - 7.0)
Creatinine	110 µmol/L	(55 - 120)

An aldosterone and renin test is performed and demonstrates an increased ratio of aldosterone to renin. Based on this, a CT abdomen is arranged. This is reported as showing bilateral adrenal hyperplasia.

What is the most appropriate management of this?

A. Bilateral adrenalectomy
B. Unilateral adrenalectomy
C. Watch and wait
D. Low sodium diet
E. Spironolactone

E. Spironolactone

The correct answer is spironolactone. Bilateral adrenal hyperplasia can be safely and successfully managed with an aldosterone antagonist (either spironolactone or eplerenone) as this will reduce the effects of aldosterone.

There is no indication for surgical management in the treatment of bilateral adrenal hyperplasia so there is no rationale for an adrenalectomy. A low sodium diet is a reasonable lifestyle modification for hypertensive patients, but there is an underlying cause so medical management is more appropriate in this case.

Watch and wait would be inappropriate as there is a clear diagnosis and a treatment.

Question 109 of 114

A 45-year-old woman comes to the clinic some 6 months after thyroid resection for differentiated thyroid cancer. She is well, has recovered from her surgery and has a neatly healed scar across her anterior neck. Blood pressure is normal at 110/80 mmHg, and her pulse is 60 and regular. Her body mass index is unchanged at 25 kg/m². Only medication is thyroid hormone replacement.

Which of the following is the most appropriate way to monitor for a recurrence?

A. MRI neck
B. Technetium scanning
C. Thyroglobulin
D. Thyroid ultrasound scan
E. T3 levels

C. Thyroglobulin

Given this patient is prescribed thyroid hormone replacement, monitoring of T3 or T4 is not useful in monitoring for cancer recurrence. On the other hand the presence of thyroglobulin does indicate thyroid gland activity which isn't suppressed by thyroid hormone replacement and is therefore potentially cancerous in origin. Levels should be undetectable.

Radiological investigations such as MRI neck, technetium scanning or thyroid ultrasound scanning are potentially less sensitive than monitoring for thyroglobulin and are therefore not a preferred first line investigation. T3 levels are unsuitable as a marker of recurrence given that conversion of exogenously administered T4 to T3 confounds any measurement.

Question 110 of 114

A 34-year-old man is admitted after being found at home covered in his vomit and excrement. He is a known alcoholic and has not been seen for two weeks.

On examination, he is drowsy and complaining of generalised aches and pains.

An ECG shows prolonged PR interval and prolonged QTc of 620ms.

On examination JVP is not visible, his mucus membranes are dry, and his eyes are sunken. His chest is clear, his heart sounds normal, and his abdomen soft and non-tender. There is moisture damage to his buttocks.

CT head is unremarkable.

Blood tests are completed and results are detailed below:

Hb	114 g/L	Male: (135-180)Female: (115 - 160)
Platelets	98 * 10 /L 9	(150 - 400)
WBC	10 * 10 /L 9	(4.0 - 11.0)
Na +	131 mmol/L	(135 - 145)
K +	2.2 mmol/L	(3.5 - 5.0)
Urea	14.2 mmol/L	(2.0 - 7.0)
Creatinine	190 µmol/L	(55 - 120)
Calcium	1.9 mmol/L	(2.1-2.6)
Phosphate	0.6 mmol/L	(0.8-1.4)
Magnesium	0.4 mmol/L	(0.7-1.0)

What is the priority in the management of this man's electrolyte disturbances?

A. Replace magnesium intravenously
B. Replace potassium intravenously
C. Start insulin-dex infusion
D. Start oral magnesium, potassium, phosphate, and calcium
E. Start telemetry

A. Replace magnesium intravenously

Replace magnesium before correcting hypokalaemia. Hypomagnesemia prevents potassium absorption

In this question, the patient has multiple electrolyte abnormalities due to poor oral intake over the preceding 2 weeks.

Intravenous magnesium replacement would be recommended in this case due to the severity of magnesium deficiency. Severe hypomagnesaemia is defined as levels <0.4 mmol/L.

Although we do need to replace **potassium intravenously**, hypomagnesemia prevents potassium absorption, so we need to correct this before supplementing potassium.

Dex-saline can be used as part of the treatment of hyperkalaemia. It does not necessarily need to be used in hypokalaemia.

Start oral magnesium, potassium, phosphate, and calcium: this man has severe hypomagnesaemia (<0.4 mmol/L) and severe hypokalaemia (<2.5 mmol/L). He requires intravenous replacement and oral supplementation is not appropriate.

Telemetry would be required, but it is not the main priority here, electrolyte replacement should not be delayed in this case whilst awaiting telemetry.

Question 111 of 114

A 57-year-old female presents after noticing a lump in her neck. She reports it being non-tender and she is only concerned about it for cosmetic purposes. She reports no other symptoms. Her past medical history includes hypertension and constipation. Her current medications include ramipril, irbesartan, amlodipine and furosemide. No family history is unavailable. On examination, her neck lump is hard, non-tender and measures 2cm by 1 cm, moves with swallowing but not with tongue protrusion. Her heart rate is 84 beats/min and blood pressure 213/130 mmHg. Examination of her joints was unremarkable with no excessive laxity.

Her blood tests are as follows:

Hb	112 g/l
Platelets	349 * 10 /l9
WBC	7.2 * 10 /l9
Na +	138mmol/l
K +	4.2 mmol/l
Urea	6.9 mmol/l
Creatinine	72 µmol/l
Adjusted calcium	3.1 mmol/l
Phosphate	0.40 mmol/l

She undergoes an outpatient ultrasound and fine needle aspiration of her neck lump. After her procedure, the recovery nurses were concerned regarding her persistent hypertension and the patient is admitted for further investigation. She develops a mild headache with no visual disturbances, resolving on its own after her blood pressure falls to 182/101 mmHg two hours later. Urinary metanephrine collection was positive.

Which investigation is likely to produce the underlying diagnosis?

A. Genetic testing for RET mutation
B. Genetic testing for germline VHL mutation
C. Genetic testing for NF2 mutation on chromosome 22
D. CT chest/abdomen/pelvis with contrast
E. Urinary calcium collection

A. Genetic testing for RET mutation

The clinical collection of phaeochromocytoma and hyperparathyroidism should raise suspicion of multiple endocrine neoplasia type 2a, which is a genetic syndrome of near 100% penetrance caused by a mutation of the RET oncogene. The addition of a thyroid lump makes this scenario more suggestive of MEN 2, with medullary thyroid carcinoma completing the diagnosis. MEN2b is subtly differentiated from MEN2a by the lack of hyperparathyroidism and Marfanoid features. Unlikely MEN 1, genetic screening of family members for hyperparathyroidism and germline mutation is useful for long-term surveillance and monitoring. VHL and NF 2 mutations refer to von-Hippel Lindau syndrome and neurofibromatosis type 2 respectively: VHL presents classically as a combination of retinal haemangioblastomas, renal cell carcinomas and phaeochromocytomas while NF2 typically present with intracranial tumours, bilateral Schwannomas almost in all patients.

Question 112 of 114

A 43-year-old female presents with neck discomfort worsening over the past 2 months. She has no other past medical or family history. Examination reveals a firm neck lump moving with swallowing but not with tongue protrusion. Subsequent ultrasound of her neck with fine needle aspirate reveals a 2.5cm papillary thyroid carcinoma. A CT neck reveals one single lymph node in her left anterior cervical chain.

What is the optimum treatment?

A. Thyroidectomy and neck dissection with postoperative radioiodine ablation
B. Thyroidectomy and neck dissection without postoperativeradioiodine ablation
C. Lobectomy and neck dissection with postoperative radioiodineablation
D. Lobectomy and neck dissection without postoperativeradioiodine ablation
E. Monitor annually

A. Thyroidectomy and neck dissection with postoperative radioiodine ablation

Diagnosis of thyroid tumours are frequently made after the patient has self-palpated a neck lump or after an incidental finding following unrelated neck imaging. Most prevalent in young females, diagnosis is clinched on fine needle aspiration. The key considerations for treatment are the size of the thyroid mass and the presence of lymph node involvement: any lump greater than 1cm in

size or has any signs of metastatic spread should undergo thyroidectomy instead of a lobectomy. The postoperative use of concurrent radioiodine ablation enhances survival benefits in patients at high risk of disease recurrence and is recommended by both the American Thyroid association and a European consensus group. Patients with thyroid lumps greater than 4cm in diameter regardless of extrathyroid disease; those with lumps between 1 and 4cm in diameter and extra-thyroid disease; and all with high-risk histology such as aggressive histological subtypes benefit from radioiodine ablation. In the case of this patient with a 2.5cm mass and lymph node involvement, thyroidectomy, lymph node clearance with neck dissection and postoperative radioiodine ablation is appropriate. Annual monitoring is thus an unsafe option.

Question 113 of 114

A 47-year-old man attended his GP after checking his blood pressure at the local pharmacy. When he had it checked it was 179/102 mmHg. The GP confirmed it was high in the surgery at 186/103 mmHg. He started him on ramipril 2.5mg and titrated up to the dose to 10mg over the next few weeks. His repeat measurements showed consistently high readings so the GP added amlodipine, which had very little effect despite being tolerated at the maximum dose. After failing to get an adequate response with the addition of a third agent the GP referred the patient to the endocrine clinic.

Observations showed a blood pressure of 190/105 mmHg and a heart rate of 98 beats per minute. On examination, the man was thin with a body mass index of 23 kg/m². His apex was diffuse and displaced with normal heart sounds. The chest was clear and abdomen was soft and non-tender with no evidence of masses or renal bruits. He was noted to have a hard, painless nodule over the thyroid gland.

The 24 hour urinary catecholamines were raised and further investigations confirmed phaeochromocytoma. He was treated medically with an alpha blocker then beta blocker whilst awaiting surgery. In this period he had further investigation into the thyroid nodule, which was a cold nodule on radionucleotide scanning.

Which type of thyroid cancer would you expect this to be histologically?

A. Papillary
B. Follicular
C. Anaplastic
D. Lymphoma
E. Medullary

E. Medullary

This question tests your knowledge of multiple endocrine neoplasias (MEN). This patient could have MEN Type IIa or IIb, which both include phaeochromocytoma and medullary thyroid cancer.

Of all thyroid cancers, medullary thyroid cancer accounts for approximately 5% (and of these, 80% will be sporadic rather than associated with MEN).

Question 114 of 114

A 19 year-old man is referred by his GP to the outpatient department after having several episodes of collapse at college. He reports that during these episodes he feels tired and 'blacks out'. Afterwards, he feels shaky and weak. There is no tongue biting or incontinence during these episodes and the patient reports that he often feels dizzy after standing up too quickly from a chair. The only other symptoms he reports is a sore throat that has persisted for a few weeks and lethargy.

On examination of the patient's mouth and throat, there are some white plaques located at the back of the tongue and throat. His sitting blood pressure is 130/80 mmHg and his standing blood pressure is 95/70 mmHg. He is otherwise well.

Blood tests are performed and reveal:

Hb	13.9 g/dL
Platelets	200 * 10 /l9
WBC	6.2 * 10 /l9
Na +	132 mmol/l
K +	5.1 mmol/l
Urea	4.7 mmol/l
Creatinine	81 µmol/l
Calcium	1.9 mmol/l
Random glucose	3.9 mmol/l

What is the most likely diagnosis?

A. Type II polyglandular autoimmune syndrome
B. Thymoma
C. Type 1 polyglandular autoimmune syndrome
D. Type III polyglandular autoimmune syndrome
E. HIV

C. Type 1 polyglandular autoimmune syndrome

The most likely diagnosis is type 1 polyglandular autoimmune syndrome. This autosomal recessive syndrome is a subtype of autoimmune polyendocrine syndrome, whereby a number of endocrine glands dysfunction. The patient's oral candidiasis is caused by a mild immune deficiency and hyposplenism. Furthermore, the patient has hypocalcaemia, caused by autoimmune dysfunction of the parathyroid gland and hypoglycaemia with hypotension, caused by autoimmune dysfunction of the adrenal gland.

Alrajhi Jad

PASSMEDICINE

HEMATOLOGY & ONCOLOGY

Internal medicine

EDITED BY
DR. JAMIL ALRAJHI

2024

773-555-493
jameelalrajhi@gmail.com

PASSMEDICINE PART 2

HEMATOLOGY

2024

Question 1 of 191

A 40-year-old woman attends the emergency department with sudden onset abdominal pain. She feels nauseated and has vomited 4 times in the past 2 hours. Her past medical history includes hypothyroidism and vitiligo.

On examination, she has a temperature of 38.2ºC. She has a heart rate of 135bpm with a blood pressure of 116/66mmHg. There is guarding and tenderness on palpation of her right iliac fossa. Rovsing's sign is positive. It is noted that she has mild bruising and petechiae over her back and extremities and on further questioning, she tells you she has been suffering from frequent nosebleeds over the past 2 weeks.

She is reviewed by the general surgical team who want to prepare her for theatre. Investigations:

Hb	127 g/L	Male: (135-180)Female: (115 - 160)
Platelets	28 * 10 /L 9	(150 - 400)
WBC	15.2 * 10 /L 9	(4.0 - 11.0)
Na +	136 mmol/L	(135 - 145)
K +	4.1 mmol/L	(3.5 - 5.0)
Urea	5.6 mmol/L	(2.0 - 7.0)
Creatinine	87 µmol/L	(55 - 120)
Prothrombin time (PT)	11 secs	(10-14 secs)
Activated partial thromboplastin time (APTT)	26 secs	(25-35 secs)
Fibrinogen	2.1 g/L	(2 - 4)
D-Dimer	275 ng/mL	(< 400)
CRP	60 mg/L	(< 5)

What is the next step in this patient's management?

A. Intravenous immunoglobulin
B. Plasma exchange
C. Platelet transfusion
D. Prednisolone
E. Romiplostim

A. Intravenous immunoglobulin

Human immunoglobulin (IVIG) is an alternative to steroids in treating ITP, particularly if the platelets need to be raised quickly

This patient has immune thrombocytopenic purpura (ITP) the immune-mediated destruction of platelets. It is more common in older females and those with a history of autoimmune disease. It may present as easy-bruising, petechiae or epistaxis or may be an incidental finding on routine blood tests as seen here. The first-line treatment of choice is oral prednisolone. However, in patients who do not respond to or have contraindications to prednisolone use or those who require a rapid rise of platelets (for example, prior to having surgery) then intravenous immunoglobulin (IVIG) can be given. This patient requires a rapid rise of platelets due to her urgent surgery for likely appendicitis making IVIG the more appropriate choice in this scenario.

Plasma exchange is not a treatment for ITP. However, it is the treatment of choice for thrombocytopenic thrombotic purpura (TTP) a rare acquired disorder of coagulation that is associated with fever, acute kidney injury, central nervous system dysfunction (including confusion, seizures or coma), thrombocytopenia and microangiopathic haemolytic anaemia.

Platelet transfusion is a treatment of choice in ITP. However, it is typically reserved for cases of an acute haemorrhagic event or cerebral haemorrhage. As this patient is otherwise well with only evidence of minor bleeding, it would not be the first-line treatment of choice.

Romiplostim is a thrombopoietin receptor agonist which is a 3rd-line agent to treat ITP. It is considered in patients who have not responded to 1st and 2nd line treatment or have symptoms of chronic ITP persisting for beyond 12 months.

Question 2 of 191

A 50-year-old gentleman presents with fatigue and bruising to the emergency department. On examination, hepatosplenomegaly is noted, but no peripheral lymphadenopathy is palpable. The following blood tests are obtained:

Hb	73 g/l
Platelets	43 * 10 /l9
WBC	2.3 * 10 /l9
Neutrophils	0.8 * 10 /l9
Lymphocytes	1.3 * 10 /l9
Monocytes	0.0 * 10 /l9
Basophils	0.02 * 10 /l9
Eosinophils	0.1 * 10 /l9

He is diagnosed with hairy cell leukaemia.

Which gene is mutated in this malignant condition?

A. BRCA1
B. BRAF
C. TP53
D. NOTCH1
E. HER2

B. BRAF

BRAF is mutated in hairy cell leukaemia

Hairy cell leukaemia is caused by a mutation in exon 15 of the gene for the protein kinase BRAF. Patients may be asymptomatic with the disease is identified incidentally or present with symptoms of cytopenia.

The most common laboratory finding is cytopenia, usually affecting two or three lineages. Leucocyte counts tend to be low, with a monocytopenia present in 90% of patients. Hairy cells are often seen in peripheral blood films but their proportion is variable. They have a characteristic appearance; lymphocytes with villous cytoplasmic projections, oval nuclei and finely mottled pale grey-blue cytoplasm.

A germline mutation in BRCA1 underlies an increased risk of malignancy (especially breast and ovarian) seen in some families. This gene is also mutated, as a somatic mutation, in other cases of breast and ovarian cancer.

A germline mutation in TP53, encoding tumour suppressor protein p53, underlies Li Fraumeni syndrome. However, p53 is mutated in over 50% of malignancies, the most commonly mutated protein. Activating mutations in NOTCH1 are implicated in T cell acute lymphoblastic leukaemia and the HER2 gene is amplified in some forms of breast cancer. These are somatic mutations.

Question 3 of 191

A 59-year-old woman presents to the haematology clinic for review. Despite treatment with cyclophosphamide, rituximab and fludarabine (FCR), she has experienced a steady rise in her lymphocyte count, accompanied by symptomatic anaemia. Examination reveals evidence of anaemia, her body mass index is 22 kg/m², and there is obvious splenomegaly.

Which of the following is the most appropriate intervention?

A. Ibrutinib
B. Lapatinib
C. Everolimus
D. Pazopanib
E. Sunitinib

A. Ibrutinib

Ibrutinib is effective and can be used for the treatment of CLL in patients who have failed a previous therapy

Ibrutinib is a small molecule kinase inhibitor which binds to Bruton's tyrosine kinase which is involved in B cell clonal replication. It therefore has an important role in the treatment of B cell malignancies including mantle cell lymphoma and chronic lymphocytic leukaemia (CLL). It is recommended for patients who have had at least one previous therapy for CLL or those with a 17p deletion or TP53 mutation.

Question 4 of 191

A 76-year-old gentleman with myelofibrosis is found to have a haemoglobin of 67 g/l on admission. He is transfused two units of red cells and discharged home three days later. Six days later, he is readmitted febrile and jaundiced. Blood tests show:

Hb	71 g/l	Na +	137 mmol/l	Bilirubin	39 µmol/l
Platelets	97 * 10 /l 9	K +	3.9 mmol/l	ALP	82 u/l
WBC	2.8 * 10 /l 9	Urea	4.9 mmol/l	ALT	15 u/l
Neuts	1.5 * 10 /l 9	Creatinine	79 µmol/l	Albumin	26 g/l

What is the most appropriate management for this condition?

A. Prednisolone
B. Intravenous immunoglobulin
C. Conservative management
D. Packed red cells
E. Tazocin

C. Conservative management

A delayed transfusion reaction occurs 7-10 days after the transfusion

This is a delayed transfusion reaction, which typically occurs 7-10 days post-transfusion. It occurs when patients are sensitised from previous transfusions or pregnancy and have alloantibodies against red cell antigens which are not picked up by cross-matching, if below detectable limits.

Management includes informing the laboratory and sending a repeat group and save sample for further testing. Usually, supportive management is sufficient, though if symptomatic from anaemia, they may require further transfusion after the further crossmatching testing is carried out. Prednisolone and intravenous immunoglobulin are not used in this situation typically. If this were bacterial contamination of the blood product, tazocin would be appropriate, though this presentation is too late and there are no other signs of sepsis.

Question 5 of 191

A 44-year-old woman is referred to ambulatory care with a 10-day history of lethargy and exertional dyspnoea. Prior to this episode, she reports having mild coryzal symptoms that have since settled. Her past medical history is only significant for hypothyroidism which is well controlled on levothyroxine.

A full screen of blood tests is sent:

Hb	71 g/L	(135 - 180)
Platelets	110 * 10 /L 9	(150 - 400)
WBC	7.0 * 10 /L 9	(4.0 - 11.0)
Urea	6.3 mmol/L	(2.0 - 7.0)
Creatinine	110 µmol/L	(55 - 120)
Prothrombin time (PT)	11 secs	(10-14 secs)
Activated partial thromboplastin time (APTT)	26 secs	(25-35 secs)
Fibrinogen	3.1 g/L	(2 - 4)
D-Dimer	345 ng/mL	(< 400)
Reticulocytes	2.5 %	(0.5 - 1.5)
Blood film	Schistocytes	
Coombs test	Negative	
Bilirubin	59 µmol/L	(3 - 17)
ALP	128 u/L	(30 - 100)
ALT	40 u/L	(0 - 40)
AST	50 u/L	(< 35)
Blood glucose	5.2 mmol/L	(4 - 7)
Total cholesterol	7.1 mmol/L	(< 5)

What is the most likely diagnosis?

A. Autoimmune haemolytic anaemia
B. Disseminated intravascular coagulation
C. Paroxysmal nocturnal haemoglobinuria
D. Thrombotic thrombocytopenia purpura
E. Zieve syndrome

A. Zieve syndrome

Zieve syndrome is a rare clinical syndrome of Coombs-negative haemolysis, cholestatic jaundice, and transient hyperlipidaemia

This patient has a likely diagnosis of **Zieve syndrome** given the triad of non-autoimmune haemolysis, jaundice and hyperlipidaemia. The negative Coombs test indicates non-autoimmune haemolytic anaemia. In Zieve syndrome, jaundice can be cholestatic in nature but also secondary to haemolysis. The raised ALP supports a cholestatic picture. Zieve syndrome is a rare syndrome and generally resolves following the cessation of alcohol use.

Autoimmune haemolytic anaemia is incorrect. Although there is evidence of haemolysis given the low Hb, reticulocytosis, raised bilirubin and presence of schistocytes, the Coombs test is negative. A negative Coombs test makes autoimmune haemolytic anaemia very unlikely.

Disseminated intravascular coagulation (DIC) is incorrect. DIC is a disease of abnormal coagulopathy characterised by a prolonged prothrombin time, prolonged APTT, low fibrinogen and a very high D-dimer. Triggers for DIC include malignancy, surgery and significant infection of which none are mentioned in this presentation. The resolved coryzal symptoms are unlikely to be significant.

Paroxysmal nocturnal haemoglobinuria (PNH) is incorrect. PNH is characterised by intravascular haemolysis, bone marrow hypoplasia and increased risk of thromboses. This does not completely fit the patient's presentation making this answer incorrect.

Thrombotic thrombocytopenia purpura (TTP) is incorrect. This condition presents as a pentad of fever, acute kidney injury, thrombocytopenia, microangiopathic haemolytic anaemia and cerebral dysfunction. The absence of the majority of these features makes TTP unlikely.

Question 6 of 191

A 54-year-old gentleman is admitted under the medical team with a diagnosis of urosepsis. Twenty four hours after admission he develops numerous bruises across his abdomen, arms and legs. It is also noted that he is bleeding from his cannula site

His blood results are as follows

Hb	110 x10 /L 12	130-168 x10 /L12
WCC	19 x10 /L 9	4.2-10.6 x10 /L9
Platelets	50 x10 /L 9	130-370 x10 /L9
CRP	300 mg/L	0-5 mg/L
Fibrinogen	3.8 g/L	1.8-4.0 g/L
PT	20s	9-12s
APTT	38s	23-31s
Creatinine	150µmol/L	60-125 µmol/L
eGFR	60 mL/min/1.73 m 2	>90 mL/min/1.73 m2

What is the most likely cause?

A. Idiopathic thrombocytopenia purpura (ITP)
B. Thrombotic thrombocytopenia purpura (TTP)
C. Disseminated intravascular coagulation (DIC)
D. Heparin induced thrombocytopenia (HIT)
E. Haemolytic uraemia syndrome (HUS)

C. Disseminated intravascular coagulation (DIC)

In DIC fibrinogen can be normal or elevated in over 50% of cases especially early on (as fibrinogen is an acute phase marker)

Fibrinogen is an acute phase marker. Therefore in DIC fibrinogen levels can be normal or elevated in over 50% of cases especially early on.

PT/APTT are usually normal in ITP, TTP, HUS and HIT

Renal function is markedly abnormal in HUS

In HIT the plasma prothrombin time (PT) is decreased but the activated partial thromboplastin time (aPTT), and the fibrinogen levels are normal.

Question 7 of 191

You are called to see a 68-year-old female on the oncology ward who has developed coffee ground vomiting. She has a diagnosis of colorectal cancer which she is currently undergoing curative chemotherapy for.

Blood results are as follows:

Hb	95 g/l
Platelets	34 * 10 /l9
WBC	14 * 10 /l9
PT ratio	2.4
aPPT ratio	2.1
D-dimer	1540 ng/ml (normal < 500)
Fibrinogen	0.8 g/l (normal 1.5-4.0)

You administer fresh frozen plasma. Repeat blood results are as follows:

Hb	91 g/l
Platelets	24 * 10 /l9
WBC	18.4 * 10 /l9
PT ratio	2.2
aPPT ratio	1.9
Fibrinogen	0.85 g/l (normal 1.5-4.0)

What treatment is indicated?

A. Cryoprecipitate
B. Vitamin K
C. Factor VIII
D. Factor IX
E. No further treatment

A. Cryoprecipitate

In bleeding patients with DIC with severe hypofibrinogenaemia (<1 g/l) that persists despite FFP replacement, then fibrinogen concentrate or cryoprecipitate is indicated

The patient has DIC likely secondary to malignancy. In bleeding patients with DIC and prolonged prothrombin time (PT) and activated partial thromboplastin time (aPTT), administration of fresh frozen plasma (FFP) may be useful. Notably, this patient continues to have severe hypofibrinogenaemia despite administration of FFP. In bleeding patients with DIC with severe hypofibrinogenaemia (<1 g/l) that persists despite FFP replacement, then fibrinogen concentrate or cryoprecipitate is indicated.

Vitamin K would be indicated in Vitamin K deficiency. There is nothing in the history to suggest vitamin K deficiency.

Factor VIII and factor IX are indicated for Haemophilia A and B respectively.

Question 8 of 191

A 74-year-old woman is referred to the medical assessment unit with diffuse bone pain affecting her back and ribs that has persisted for two months. She has no significant past medical history and is not on any regular medications. She does not smoke and still workers as a potter.

Clinical examination is unremarkable and there are no focal areas of tenderness or swelling. There is no organomegaly or lymphadenopathy. Cardio-respiratory examination is unremarkable.

Blood tests:

Hb	111 g/L	Male: (135-180)Female: (115 - 160)
Platelets	389 * 10^9 /L	(150 - 400)
WBC	4.2 * 109 /L	(4.0 - 11.0)
Na +	138 mmol/L	(135 - 145)
K +	4.2 mmol/L	(3.5 - 5.0)
Urea	4.8 mmol/L	(2.0 - 7.0)
Creatinine	88 µmol/L	(55 - 120)
CRP	10 mg/L	(< 5)
Calcium	2.68 mmol/L	(2.20-2.60)
Immunoglobulin G	24.1 g/L	(6.6 - 15.9)
Immunoglobulin A	4.4 g/L	(0.6-5)
Immunoglobulin M	2.2 g/L	(0.53 - 2.47)

Serum protein electrophoresis:

Paraprotein	present

Given the likely diagnosis, what is the first-line imaging investigation?

A. Isotope bone scan
B. Low dose whole body CT
C. Fluorodeoxyglucose positron emission tomography CT (FDG PET-CT)
D. Plain radiographic skeletal survey
E. Whole body MRI

A. Whole body MRI

Whole body MRI 1st line imaging in suspected multiple myeloma

Whole-body MRI is the correct answer. This patient presents with diffuse bone pain, anaemia, hypercalcemia, raised IgG and a paraprotein. She likely has multiple myeloma. Recent NICE guidelines state that imaging should be offered to all patients with suspected myeloma and that whole-body MRI is the first-line choice in the absence of contraindication.

Low dose whole-body CT is incorrect. This investigation can be offered if an MRI is contraindicated or not tolerated.

A plain radiographic skeletal survey is incorrect. This is only offered if CT or MRI are unsuitable or not tolerated.

Isotope bone scan is incorrect. This is not recommended in the investigation of suspected myeloma.

FDG PET-CT is incorrect. This imaging investigation is only an option for those patients with confirmed multiple myeloma who have not yet had any whole body imaging

Question 9 of 191

A 67-year-old man with a previous diagnosis of myelodysplasia presents to a haematology clinic with rapid weight loss, an increase in nosebleeds and a greater feeling of fatigue than previously. He has the single lineage dysplasia subtype and usually requires monthly transfusions; he has presented two weeks after his last red blood cell top-up.

His blood results are as follows:

Hb	66 g/L	Male: (135-180)Female: (115 - 160)
Platelets	9 * 109 /L	(150 - 400)
WBC	1.1 * 109 /L	(4.0 - 11.0)

A blood film is sent for and he is admitted for blood and platelet transfusions.

What is the blood film likely to show?

A. Burr cells
B. Fragmented red blood cells
C. Lymphoblasts
D. Myeloblasts
E. Smear cells

D. Myeloblasts

Myelodysplasia may progress to acute myeloid leukaemia

Myeloblasts is the correct answer as this is most likely a malignant transformation of myelodysplasia to acute myeloid leukaemia (AML). This is evidenced by the myeloblasts coupled with the significant drop in haemoglobin, platelets and white blood cells, with the B symptoms of weight loss and fatigue becoming more prominent. The clue in the blood tests is that he previously had single lineage dysplasia which causes refractory anaemia, but now he has platelet and white blood cell reduction indicating bone marrow failure. The white cells overproduced in AML are the immature myeloblasts, not normally found in the blood.

Burr cells are incorrect as these are spiculated red blood cells commonly found in chronic renal failure due to high levels of nitrogen in the blood destabilising the red cell membrane.

Lymphoblasts are incorrect as these cells are only found in the peripheral blood film in acute lymphoblastic leukaemia.

Smear cells are incorrect because they are the result of artefactual damage to mature lymphocytes during film production, often characteristic of chronic lymphoblastic leukaemia.

Fragmented red blood cells are seen in many conditions which result in intravascular haemolysis, such as renal failure, microangiopathic haemolytic anaemia, haemolytic uraemic syndrome or thrombotic thrombocytopenic purpura. The reason for the anaemia, in this case, is bone marrow failure as opposed to haemolysis.

Question 10 of 191

An 18-year-old woman presents to the haematology clinic with spontaneous bruising, heavy periods and recurrent epistaxis. This has been a continuous problem during her teenage years but she was recently found to have anaemia and received an iron transfusion which prompted further investigation into these symptoms.

On examination, she is well and alert. There are numerous purpura across both arms and she denies trauma or any itchiness.

Hb	98 g/l
Platelets	314 * 10 /l9
WBC	5.6 * 10 /l9
PT	13.8
APTT	34.3
PFA-100 assay	prolonged closure time
Flow cytometry	GPIIb/IIIa negative
Ristocetin-induced platelet aggregation	normal agglutination

What is the likely diagnosis?

A. Bernard-Soulier disease
B. Glanzmann thrombasthenia
C. Von Willebrand disease
D. Haemophilia A
E. Evan's syndrome

B. Glanzmann thrombasthenia

Innate defective glycoprotein IIb/IIIa -> Glanzmann's thombasthenia

This is a test of the coagulopathies. Von Willebrand disease and haemophilia tend to have raised APTT. Evan's syndrome is an autoimmune disease with low platelets and red blood cells. The PFA- 100 assay shows that platelets are not aggregating and this may be due to platelet glycoproteins or their interaction with von willebrand factor. The ristocetin effectively allows aggregation of von willebrand factor to GPIb and does not require GPIIb/IIIa. Therefore Glanzmann thrombasthenia will have normal platelet aggregation once ristocetin is added.

Question 11 of 191

A 48-year-old man was admitted to the acute medical unit with history of haematemesis and melaena. No previous history was available. He required fluid resuscitation and had eight-units of red cell transfusion. On review after his transfusion, his pulse was found to be 80 beats per minute and blood pressure 130/78mmHg. Chest and abdominal examination were essentially normal. Repeat bloods were taken and results are as shown below:

Hb	110g/l (130 - 180)
Platelets	80 * 10 /l (150 - 400)9
WBC	10 * 10 /l (4.0 - 11.0)9
Prothrombin time	14.2secs (11.5 - 15.5)
INR	1.3 (<1.4)
APTT	32secs (30 - 40)
Thrombin time	17secs (15 - 19)
Fibrinogen	2.1g/l (1.8 - 5.4)

Which blood product should he be given at this stage?

A. Cryoprecipitate and fresh frozen plasma
B. Fresh-frozen plasma
C. Fresh-frozen plasma and platelets
D. Platelets
E. No blood products are indicated

A. No blood products are indicated

Fresh frozen plasma is offered to bleeding patients who have either a prothrombin time or activated partial thromboplastin time greater than 1.5 times normal

Decisions on transfusion of blood products should recognize that over-transfusion may be as damaging as under-transfusion by exposing patients to the risk of fluid overload and other complications of transfusion. Do not offer platelet transfusion to patients who are not actively bleeding and are haemodynamically stable. Platelets may be given to those actively bleeding and have a platelet count of less than 50 Ã— 109/L. Offer fresh frozen plasma to bleeding patients who have either a prothrombin time or activated partial thromboplastin time greater than 1.5 times normal; and cryoprecipitate or fibrinogen concentrate to those with a fibrinogen level of less than 1 g/L. Offer prothrombin complex concentrate to patients who are taking warfarin and actively bleeding. Treat patients who are taking warfarin and whose upper gastrointestinal bleeding has stopped in line with local warfarin protocols.

Question 12 of 191

A 65-year-old man has been admitted with worsening abdominal pain, hypotension and altered mental state. He is currently being treated for intra-abdominal sepsis secondary to peritoneal dialysis for chronic renal failure secondary to diabetic nephropathy. Previous medical history includes type 2 diabetes mellitus, angina, hypertension, hyperlipidaemia, and diabetic retinopathy. The patient is not anti-coagulated but does take aspirin.

He is started on intravenous antibiotics and intravenous fluid. His results are:

Hb	91 g/L	Male: (135-180)Female: (115 - 160)
Platelets	60 * 109 /L	(150 - 400)
WBC	24.5 * 10 /L 9	(4.0 - 11.0)
Na +	128 mmol/L	(135 - 145)
K +	5.8 mmol/L	(3.5 - 5.0)
Urea	31.7 mmol/L	(2.0 - 7.0)
Creatinine	645 µmol/L	(55 - 120)
CRP	317 mg/L	(< 5)
PT	22 seconds	(10-14 seconds)
APTT	56 seconds	(30-40 seconds)

He undergoes a CT scan which shows a spontaneous retroperitoneal haemorrhage and is referred to interventional radiology.

Given the patient's coagulation profile, what blood product is needed?

A. Cryoprecipitate
B. Fresh frozen plasma
C. Protamine
D. Prothrombin complex concentrates
E. Recombinant activated factor 7

B. Fresh frozen plasma

In bleeding patients with DIC and prolonged prothrombin time (PT) and activated partial thromboplastin time (aPTT), administration of fresh frozen plasma (FFP) may be useful

Fresh frozen plasma is correct. The clinical picture above is that of DIC. There is a prolongation of both PT and APTT. Patients with disseminated intravascular coagulopathy go through a hypercoagulable state followed by a hypo-coagulable state as they use up their reserves of clotting factors. Fresh frozen plasma replaces multiple clotting factors. The disturbed clotting state, as shown with prolongation of PT and APTT, indicates that clotting factor concentrations are below 30% of normal. A typical dose of fresh frozen plasma (15ml/kg) will increase circulating clotting factors by 10%.

Cryoprecipitate is incorrect because this is used to replace fibrinogen in haemorrhaging patients. It also contains Factor VIII and XIII, von Willebrand's Factor and fibronectin. It can be used in patients with von Willebrand's disease and haemophilia.

Protamine is incorrect because this is used to correct the effects of heparin, either unfractionated heparin or low molecular weight heparin. Administration can cause hypotension and high doses can inhibit platelet function.

Prothrombin complex concentrate is incorrect because this is used to reverse warfarin and can be used to reverse some direct oral anticoagulants. This includes clotting factors II, VII, IX and X. It is used for the rapid correction of an acquired coagulation disturbance, commonly warfarin. PT levels are monitored for dose-response. Repeat doses may be required 8-12 hours after the initial dose. An example is Octaplex.

Recombinant activated factor 7 is incorrect because this is used in haemophilia patients and off label in severe multiple trauma, non-traumatic intracranial haemorrhage and some surgeries. It is almost identical to native factor 7.

Question 13 of 191

A 45-year-old man is admitted from a local park. On arrival, he is in respiratory distress with a ventilatory rate of 34 breaths per minute and a peripheral oxygen saturation of 89% on 15L/min oxygen via a non-rebreathe mask. He is grey and sweaty with central cyanosis. Heart rate is 122bpm and blood pressure is 94/66mmHg. Examination of the chest discloses a normal cardiorespiratory examination and there is no clinical indication of heart failure. A chest x-ray shows no acute pathological lesion but background emphysematous changes and an ECG shows only sinus tachycardia with a corrected QT interval approaching the upper limit of normal with no ischaemic lesions.

An arterial blood gas taken on 15L/min oxygen shows:

pH	7.17	HCO3-	13.4	Glucose 6.9 mmol/l	MetHb	35%
pO2	10.9	Base excess	-5.8	Potassium 5.5 mmol/l	COHb	8%
pCO2	6.7	Lactate	4.1 mmol/l			

He is beginning to show signs of tiring and confusion and his GCS has fallen to 13/15 (E3V4M6) but he tells you he has been inhaling Liquid Gold (an alkyl nitrite).

Which of the following interventions is most appropriate in this patient's immediate management?

A. Continuous positive airways pressure ventilation (CPAP) pending transfer to a hyperbaric chamber
B. Reduce inspired oxygen concentration and controlled oxygen therapy via Venturi valve
C. 75mg 1% methylthioninium chloride solution IV over 5 minutes
D. 300mg 3% sodium nitrite solution IV over 10 minutes
E. 50ml 8.4% sodium bicarbonate solution IV over 20 minutes

D. 75mg 1% methylthioninium chloride solution IV over 5 minutes

Methylene blue can be used to treat methaemoglobinaemia. It causes a reduction of Fe3+ (ferric) to Fe2+ (ferrous)

The arterial blood gas in the above clinical description shows a mixed respiratory and metabolic acidosis with a relative hypoxia despite the inspired fraction of oxygen. However, the significant abnormality is the fraction of methaemoglobin on the gas at over 30% which is significantly elevated. The carboxyhaemoglobin level is slightly raised at 8% but levels such as this may be seen in smokers and are not necessarily pathological. In this case, the diagnosis is of acute methaemoglobinaemia which is a rare but potentially fatal consequence of inhalation of poppers (alkyl nitrites).

Poppers are a clear or yellowish, sweet-smelling volatile liquid which is inhaled as a recreational drug of abuse which yields euphoric and anxiolytic effects. It also is a vasodilator and is sometimes used as an adjunct in sexual acts, and previously was a treatment for angina pectoris. However, in some patients, particularly susceptible individuals such as those with sickle cell traits or a G6PD deficiency, inhalation of poppers may rapidly oxidise ferrous iron ions in haemoglobin to their ferric (Fe3+) states creating methaemoglobinaemia (MetHb). MetHb binds oxygen more avidly than haemoglobin and causes a shift in the dissociation curve to the left meaning that oxygen is not liberated in the tissues causing a cellular hypoxia. This manifests in a clinical cyanosis and often a deathly grey pallor to the patient. Oxygen saturations may not be as shocking as the patient appears since many pulse oximeters cannot distinguish between oxyhaemoglobin and methaemoglobin well. However, supplemental oxygen will not improve recorded values (although it should still be given). An impairment in gas exchange is often seen in acute methaemoglobinaemia with raised tensions of CO2 and low tension of O2 and consequent respiratory acidosis. An additional metabolic acidosis occurs due to cellular anoxic respiration. In smokers, this condition may be mistaken for a type 2 respiratory failure due to chronic obstructive lung disease. The methaemoglobin count and low bicarbonate make this unlikely and reducing the inspired oxygen is likely to worsen the situation. CPAP may be tried but will not reverse the underlying problem and hyperbaric oxygen is helpful only in situations of high carboxyhaemoglobin, such as carbon monoxide poisoning, which is not the case here.

The first line treatment for methaemoglobinaemia is the administration of methylthioninium chloride, also known as methylene blue, which reduces the ferric ions back to their ferrous states rapidly. Several administrations may be required, especially in high concentrations of MetHb. Sodium nitrite is a recognised treatment for cyanide poisoning. It actually causes methaemoglobinaemia since cyanide ions are cleared more rapidly when complexed with MetHb. Administration of sodium nitrite is not indicated in this vignette and will make the situation worse. Sodium bicarbonate may well be indicated in this patient due to the profound acidosis but it is not the most pressing concern. Rectification of the oxygen-carrying capacity of the blood may improve the acid-base balance without the need for bicarbonate, and hypoxia is likely to kill the patient more quickly than acidosis with a normal potassium. Remember that 8.4% bicarbonate solution, if given, should ideally be infused through a central vein since it is extremely caustic.

Question 14 of 191

A 74-year-old gentleman presents with shortness of breath on exertion. He denies cough, wheeze, or chest pain. He has a past medical history of Hodgkin's lymphoma. Examination of the cardiopulmonary system is normal.

Blood results are as follows:

Hb	55 g/l
Platelets	346 * 10 /l9
WBC	8.4 * 10 /l9

You decide to transfuse him two units of red cells. What special request will you make?

A. Leukodepleted
B. CMV negative
C. No special request required
D. Irradiated red cells
E. Cell salvage

D. Irradiated red cells

The risk of transfusion associated graft versus host disease in Hodgkin's lymphoma is unrelated to the treatment modality and/or disease stage. Individuals with Hodgkin's lymphoma should therefore receive X- or gamma-irradiated blood for life

If an immunocompromised patient receives red cells that are not irradiated, the donor's T cells can cause transfusion-associated graft-versus-host disease (TA-GvHD), in which the donor T lymphocytes mount an immune response against the recipient's lymphoid tissue causing tissue and organ damage, potentially leading to death.

Typical clinical features of TA-GvHD include fever, an erythematous maculopapular rash, abdominal pain and profuse diarrhoea.

Irradiation of blood products destroys the donors T lymphocytes thereby helping to prevent this potentially fatal complication.

Indications for gamma irradiated blood products include:

- Immunocompromised marrow or organ transplant recipients
- Patients with haematological disorders who will be undergoing allogeneic marrow transplantation imminently
- Intrauterine transfusions
- Patients with Hodgkin's disease
- Patients treated with purine analogue drugs (e.g. fludarabine)

Interestingly, the risk of transfusion associated graft versus host disease in Hodgkin's lymphoma is unrelated to the treatment modality and/or disease stage. Individuals with Hodgkin's lymphoma should therefore receive X- or gamma-irradiated blood for life.

In the UK, all blood products are leukodepleted to prevent Creutzfeldt-Jakob disease transmission.

The use of CMV-seronegative blood products is reserved for CMV-seronegative individuals that are likely to proceed to haematopoietic stem cell transplant, or neonates

Question 15 of 191

A 28-year-old woman presents to the acute medical unit with fatigue, lethargy, and abdominal pain. She is pregnant at 32 weeks gestation.

Blood results are as follows:

Hb	62 g/L	Male: (135-180)Female: (115 - 160)
Platelets	182 * 10 /L 9	(150 - 400)
WBC	8.8 * 10 /L 9	(4.0 - 11.0)

You discuss the case with the obstetrics registrar on call who advises you that they will review the patient in the department and asks you to arrange a blood transfusion.

What special red cell requirements are required?

A. Cytomegalovirus (CMV) negative
B. High titre anti-A and anti -B negative
C. Irradiated
D. Leukodepleted
E. Washed

A. Cytomegalovirus (CMV) negative

The use of CMV-seronegative blood products is reserved for CMV-seronegative individuals that are likely to proceed to haematopoietic stem cell transplant, or neonates

Cytomegalovirus (CMV) negative is the correct answer. CMV is a type of herpes virus. Primary infection is usually asymptomatic but may cause a flu or glandular fever-like illness, leading to a lifelong infection in all age groups. The virus resides in monocytes and can reactivate from its latent state. More severe disease may occur in individuals with impaired immunity such as foetuses, neonates and patients of any age who have been immuno-suppressed by disease or treatment.

Transmission of CMV present in blood components can give rise to primary infection in CMV negative patients or to reinfection in previously infected patients.

With very few stated exceptions (e.g. granulocytes), from November 1999 all allogeneic blood components produced in the UK have been subjected to a leukocyte depletion process. This was primarily a vCJD risk reduction measure, however, it has resulted in other added benefits including reduced incidence of non-febrile haemolytic transfusion reactions, TRALI, and transfer of CMV.

The following groups of patients require **CMV-negative blood**:

Intra-uterine transfusions

Neonates up to 28 days post expected date of delivery Pregnancy

As the patient in this clinical case is pregnant, CMV-negative blood is required.

Historically, immunocompromised patients who have not been infected with CMV (CMV negative) received CMV-negative blood due to the possible life-threatening complication of infection. However recent studies have shown that leukodepletion is just as effective as CMV IgG-negative blood components. Therefore CMV IgG negative donations are no longer required in this cohort.

Irradiated blood is required in individuals with severe immunodeficiencies, those with a history of Hodgkin's lymphoma, patients who have been exposed to certain drugs (e.g. bendamustine and fludarabine), and following stem cell transplant. Pregnancy is not an indication for irradiated blood.

All blood products in the UK are **leukodepleted** (with very few exceptions eg granulocytes). This is therefore not a special requirement.

With transfusion of plasma-rich components (e.g. FFP and platelets), we are more concerned about the passive transfer of antibodies (e.g. Anti-A and Anti-B) in contrast to the transfer of antigens as in red cell transfusions (which contain negligible amounts of plasma). Thus for plasmarich components, group AB is the universal donor and group O is the universal recipient, in stark contrast to packed red cells. **High titre anti-A and anti -B** negative can be requested for incompatible plasma-rich transfusion. This clinical case is regarding red cell transfusions and thus this special requirement is not applicable.

Washed red cells can be requested for patients who have recurrent febrile or allergic reactions to standard packed red cells. Pregnancy is not an indication for washed red cells.

Question 16 of 191

A 35-year-old man presents to the emergency department with generalised weakness, nausea, and joint pain. He has a past medical history of depression for which he has been prescribed sertraline but has not taken it for several months. He is a current smoker and also uses intravenous recreational drugs.

On examination, he has a non-blanching, slightly raised purplish rash over his torso and part of his limbs. He is slightly jaundiced and has mild right upper quadrant tenderness.

Blood tests are taken:

Bilirubin	51 µmol/L	(3 - 17)
ALP	188 u/L	(30 - 100)
ALT	142 u/L	(3 - 40)
Î³GT	82 u/L	(8 - 60)
Albumin	29 g/L	(35 - 50)
HBsAg	Negative	
Anti-HBV	Negative	
Anti-HCV	Positive	
HCV RNA	Positive	
HIV antibodies	Negative	

What is the most likely diagnosis?

A. Cold agglutinin disease
B. Cryoglobulinaemia type 1
C. Cryoglobulinaemia type 2
D. Cryoglobulinaemia type 3
E. Warm autoimmune haemolytic anaemia

C. Cryoglobulinaemia type 2

Hepatitis C + purpuric rash → cryoglobulinaemia

This patient, in summary, has generalised weakness, arthralgia, and a rash consistent with palpable purpura on the background of a diagnosis of hepatitis C. The combination of hepatitis C and purpura is suspicious of cryoglobulinaemia. Meltzer's triad is a combination of palpable purpura, arthralgia, and weakness and are the classical symptoms of cryoglobulinaemia.

There are three types of cryoglobulinaemia. It is **cryoglobulinaemia type 2** that is associated with hepatitis C and therefore this is the correct answer. Other associations of type 2 include rheumatoid arthritis, Sjogren's, and lymphoma.

Cold agglutinin disease is an autoimmune disease caused by the presence of cold-sensitive antibodies which act on red blood cells. It has both primary and secondary forms, of which hepatitis C can be a trigger of secondary cold agglutinin disease. Symptoms can include arthralgia and weakness, but the presence of palpable purpura is not associated. Therefore, this diagnosis is less likely.

Cryoglobulinaemia type 1 is not associated with hepatitic C. Associations include multiple myeloma and Waldenstrom macroglobulinaemia.

Cryoglobulinaemia type 3 is not associated with hepatitic C. Associations include rheumatoid arthritis and Sjogren's.

Warm autoimmune haemolytic anaemia is the most common form of autoimmune anaemia. It is idiopathic in 50% of cases. In others, it is often associated with another autoimmune disease such as rheumatoid arthritis or systemic lupus erythematosus. Therefore, this diagnosis is unlikely in this case.

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A 40-year-old man presents to the Emergency Department with non-specific symptoms of lethargy, malaise, headache, body aches, a low-grade fever and a sore throat. The examination is essentially normal aside from multiple limb petechiae. Tympanic temperature is 37.6°C. Blood pressure, heart rate and pulse oximetry are normal.

Blood tests show:

Haemoglobin	112g/L	Sodium	136mmol/L
MCV	82fL	Potassium	4.5mmol/L
Platelets	77x10^9/L	Urea	6.9mmol/L
White cells	32x10^9/L	Creatinine	111µmol/L
Prothrombin time	23secs	CRP	54mg/L
Fibrinogen	0.45g/L	HIV test	Negative
Liver enzymes	normal		

The automated counter is unable to supply differential white cell count.

Manual blood film shows immature granulocytes with bilobed nuclei and Auer rods.

What is the most important immediate therapy to institute?

A. All trans retinoic acid (ATRA) [tretinoin]
B. Methotrexate
C. Imatinib
D. Rituximab
E. Arsenic trioxide

A. All trans retinoic acid (ATRA) [tretinoin]

APML is treated with all-trans retinoic acid (ATRA) to force immature granulocytes into maturation to resolve a blast crisis prior to more definitive chemotherapy

The above vignette gives a classical description of acute promyelocytic leukaemia (APML). This diagnosis is considered a haematological emergency. Approximately 10% of patients with an acute leukaemia are ultimately diagnosed with APML but it is a largely unconsidered diagnosis. If diagnosed, it is one of the most treatable leukaemias, however, in one-third of cases the initial presentation is of catastrophic haemorrhage.

The average age of presentation with APML is 30-40 which distinguishes it from other acute myeloid leukaemias which present in the seventh decade typically. Other presenting symptoms are similar however with fatigue and weakness due to anaemia, coryzal or influenza symptoms and easy bruising due to thrombocytopaenia and coagulopathy. Myeloid leukaemia may cause splenomegaly which is usually absent in APML. Lymphadenopathy does not occur in leukaemias of myeloid line.

A blood film may show characteristic Auer rods in both myeloid leukaemia and APML. The film will show abnormal myeloid precursor cells or granulocytes in APML which are often bilobar or multilobar, whereas leukaemic mature myeloid cells are present in other forms of myeloid leukaemia although distinguishing these is difficult. Automated counters may fail to determine a differential white cell count. Confirmation of AMPL is via polymerase chain reaction or fluorescent in-situ hybridisation (FISH) of the bone marrow which demonstrates translocation of chromosomes 15 and 17. A gene on chromosome 17 codes for the retinoic acid receptor alpha protein and in APML patients with this translocation, administration of all-trans retinoic acid forces the immature myeloid cell to mature and it then undergoes apoptosis. ATRA is considered an emergency treatment for an APML blast crisis in those with coagulopathy or severe anaemia or sepsis. ATRA is considered only an emergency drug to force cell maturation; ongoing chemotherapy is required to maintain remission. Current guidelines recommend arsenic trioxide as the first line agent to achieve remission once ATRA therapy is commenced and also in refractory disease.

Methotrexate may sometimes be used to maintain remission in some APML patients although it is not helpful in the acute presentation.

Imatinib is a tyrosine kinase inhibitor used in the treatment of chronic myeloid leukaemia, particularly those with the Philadelphia translocation. It is not commonly used in this scenario.

Rituximab is a monoclonal antibody against CD20 on B cells. It has many uses in inflammatory diseases, lymphoma and lymphocytic leukaemia but is rarely used in APML or myeloid leukaemia (since B cells are of lymphoid, not myeloid lineage). There are some studies which have suggested a link between rituximab usage and the development of APML.

Question 18 of 191

A 43-year-old man presents to the emergency department with melaena and haematemesis. He has a past medical history of immune thrombocytopenia and is not currently on any treatment. He does not drink alcohol.

Observations:

Heart rate 108 beats per minute Blood pressure 92/58 mmHg Respiratory rate 18/minute Oxygen saturations 96% on room air Temperature 37.1ºC

On examination, there is dried blood around the patient's mouth. Ecchymoses are noted on his arms and abdomen. There is melaena per rectum. There are no signs of chronic liver disease.

Blood tests:

Hb	73 g/L	Male: (135-180)Female: (115 - 160)
Platelets	9 * 10 /L	(150 - 400)
WBC	8.2 * 10 /L	(4.0 - 11.0)
Na	137 mmol/L	(135 - 145)
K	4.2 mmol/L	(3.5 - 5.0)
Urea	5.2 mmol/L	(2.0 - 7.0)
Creatinine	89 µmol/L	(55 - 120)
CRP	4 mg/L	(< 5)
Bilirubin	12 µmol/L	(3 - 17)
ALP	38 u/L	(30 - 100)
ALT	27 u/L	(3 - 40)
Î³GT	44 u/L	(8 - 60)
Albumin	38 g/L	(35 - 50)
Prothrombin time	11 seconds	(10-12)

Given the clinical history, what is the most appropriate choice of treatment to raise the platelet count?

A. Azathioprine
B. Intravenous immunoglobulin
C. Prednisolone
D. Rituximab
E. Romiplostin

B. Intravenous immunoglobulin

Human immunoglobulin (IVIG) is an alternative to steroids in treating ITP, particularly if the platelets need to be raised quickly

Intravenous immunoglobulin (IVIG) is correct. The patient presents with life-threatening bleeding and a platelet count of 9. The fastest way to increment the platelet count in ITP is with IVIG, which is indicated in cases of life-threatening bleeding. The expected response time is 1-3 days. In cases of life-threatening bleeding, platelets are typically given in addition.

Prednisolone is incorrect. This is the typical first-line treatment for ITP. However, in cases of lifethreatening bleeding and where the platelet count needs to be incremented quickly, IVIG is more effective. The expected response time is 4-14 days.

Rituximab is incorrect. This is used in refractory cases and takes between 7-56 days to raise the platelet count. It would not be used in the first instance.

Azathioprine is incorrect. This can be used as a steroid-sparing agent in ITP but it takes between 30-90 days to work and would therefore not be used in an emergency such as this case.

Romiplostim is incorrect. This is a fusion protein that activates the thrombopoietin (TPO) receptor and increases platelet production. It takes between 5-14 days to raise the platelet count

Question 19 of 191

A 36-year-old female was referred to the outpatient haematology clinic having been referred by her GP with a falling white cell count. Eight weeks ago she saw her own GP complaining of feeling continuously tired after a viral upper respiratory illness two weeks prior to the onset of her symptoms. She also complained of feeling generally unwell and of having intermittent pains in all her joints and muscles without swelling or stiffness. Her respiratory symptoms had fully resolved and she denied any night sweats or weight loss. Her GP organised a set of screening blood investigations which revealed a white cell count of 2.6 *10^9 g/dl (neutrophil count 2.0 *10^9 g/dl). This was repeated on two further occasions over the next four weeks revealing results of 2.2 and 1.9 respectively (neutrophil counts of 1.5 *10^9 g/dl and 1.2 *10^9 g/dl respectively). Her past medical history included hypothyroidism for which she was treated with levothyroxine 150mcg OD.

Examination at the clinic revealed the presence of a systemically well female. Her blood pressure was 118/74 mmHg, heart rate 82 bpm, respiratory rate 16/min and temperature 36.6ºC. Examination of her cardiovascular system was unremarkable. Similarly, examination of her gastrointestinal system was unremarkable, with no organomegaly identified. No cervical, axillary or inguinal lymph nodes were palpable. Examination of her ENT system was unremarkable.

Initial investigations at the clinic revealed the following results:

Hb	122 g/l
WCC	2.0 * 10 /l
Neutrophils	1.3 * 10 /l
Lymphocytes	0.6 * 10 /l
Monocytes	0.1 * 10 /l
Platelets	224 * 10 /l
Blood film	Neutropaenia
B12	224 (NR 160-900 ng/l)
ESR	15 mm/hr
CRP	9 mg/l
TSH	0.35 (NR 0.4-3.6mu/ml)
FT4	11.6 (NR 4.5-13.6 mcg/dl)
Monospot test	Negative
CMV serology	Negative

What is the single most appropriate management option?

A. Organise bone marrow aspirate and biopsy
B. Organise peripheral blood flow cytometry analysis
C. Repeat full blood count in four weeks
D. Organise CT neck, thorax, abdomen and pelvis
E. Organise blood cytogenetic analysis

C. Repeat full blood count in four weekssss

This is a very common scenario seen in the haematology outpatient clinic. This patient most probably has transient myelosuppression secondary to a viral infection with post viral fatigue and malaise. Although she is symptomatic, she is systemically well and there are no other red flag symptoms or signs including the presence of lymphadenopathy and splenomegaly. Her neutrophil count has been falling but it has remained above 1 and she is not febrile or septic. Her neutrophil count is beginning to improve (albeit very slowly) and therefore a repeat FBC would be justified to ensure that it does return to normal. If it remains low further investigation would be justified.

Question 20 of 191

You are asked to review a 74-year-old man on the haematology ward. He complains of widespread bruising and bleeding gums. He has a past medical history of myelodysplasia. He has required 6 red cell transfusion and 5 platelet transfusions in the past 12 months.

Blood results are as follows:

Hb	95 g/l
Platelets	8 * 10 /l9
WBC	4.2 * 10 /l9

You decide to transfuse him 1 unit of platelets. A repeat blood test the following day is reported below:

Hb	92 g/l
Platelets	12 * 10 /l9
WBC	3.8 * 10 /l9

What investigation will you order next?

A. Anti HLA antibodies
B. Platelet Factor 4 Antibody
C. Von Willebrand factor (vWF) levels
D. Lupus anticoagulant
E. Anti-β2 glycoprotein

A. Anti HLA antibodies

HLA-matched platelets and single-donor platelets are used for individuals that are refractory to platelet transfusions and have developed anti-HLA or antiplatelet antibodies

Platelet refractoriness is a failure to obtain a satisfactory response to a platelet transfusion on two or more occasions. An unsatisfactory response is defined by a platelet rise of less than 10 * 10 /l. When a patient does not have a satisfactory response to a platelet transfusion it is important to establish exactly why this has occurred. There are many mechanisms which can play a role including both immunological (e.g. alloimmunisation) and non-immunological (e.g. splenomegaly, DIC and sepsis).

This patient has had multiple previous platelet transfusions, making alloimmunisation high up in the differential. It is therefore important to check for anti-HLA or antiplatelet antibodies. HLAmatched platelets and single-donor platelets are used for individuals that are refractory to platelet transfusions and have developed anti-HLA or antiplatelet antibodies.

Platelet Factor 4 (PF4) antibodies are checked when heparin induced thrombocytopenia is suspected.

Anti-β2 glycoprotein and lupus anticoagulant are checked when antiphospholipid syndrome is suspected.

Question 21 of 191

The hospital is running low on its stock of cytomegalovirus (CMV) seronegative blood products. All the blood products are routinely leuko-depleted to reduce the risk of transferring CMV. You are asked to prioritise patients for CMV seronegative blood.

From the following options, which patient is the highest priority?

A. Patient having post-partum haemorrhage after delivery of twins
B. Patient on high dose oral steroids for temporal arteritis
C. Patient with HIV with CD4 count 50, not on anti-retroviral therapy
D. Patient with acute myeloid leukaemia who is planned for haematopoietic stem celltransplant next week
E. Patient with previous CMV pneumonitis

D. Patient with acute myeloid leukaemia who is planned for haematopoietic stem celltransplant next week

The use of CMV-seronegative blood products is reserved for CMV-seronegative individuals that are likely to proceed to haematopoietic stem cell transplant, or neonates

The correct answer is patient 4 who is planning for a haematopoietic stem cell transplant. The patient with the post-partum haemorrhage and the patient with severe anaemia secondary to upper GI bleed both require emergency transfusion but do not require CMV negative blood.

Other patients that should receive CMV-negative blood are neonates and pregnant women (other than during delivery). Blood from a patient who is seropositive for CMV is potentially infectious to the recipient due to either infectious virus in the plasma or latent virus in the monocytes in the blood. Blood products are leuko-depleted to remove the white blood cells to reduce the risk of transmission via monocytes. The use of seronegative blood has a lower risk of transfer to CMV but there is still a possible risk of transmission, as the antibodies take time to become positive after the donor has been infected.

Post-partum haemorrhage: pregnant women should be given seronegative blood if possible during pregnancy to prevent CMV infection of the fetus. This does not apply during or after delivery because there is no risk to the fetus, so this patient would not require seronegative blood.

High-dose oral steroids: this patient does not require seronegative blood because immune suppression is not an indication for seronegative blood products.

HIV infection is not an indication for seronegative blood products even in advanced untreated disease.

Previous CMV pneumonitis: this patient has already been infected with CMV and therefore does not require seronegative blood products.

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A 23-year-old man presents with diffuse bruising, abdominal pain, and loss of appetite. He feels unwell and has no history of taking any medications, does not use dietary supplements, and does not use illicit drugs. His past medical history is negative for any prior illnesses or hospitalisations. No family history of any bleeding disorders.

On examination, he has a heart rate of 115 bpm with a blood pressure of 130/75mmHg. His respiratory rate is 18 breaths per minute and he is afebrile with a temperature of 37.2ºC. His abdominal exam demonstrates rebound tenderness in the right lower quadrant.

Investigations:

Hb	13.7 g/L	(135-180)
Platelets	48 * 10 /L $_9$	(150 - 400)
WBC	5 * 10 /L $_9$	(4.0 - 11.0)
Haematocrit	41%	(41-50%)
INR	1.1	(0.9-1.2)
Fibrinogen	2.2 g/L	(2 - 4)

A diagnosis of acute appendicitis is made and the general surgical team decides that the patient needs to be taken to theatre for an urgent laparoscopic appendectomy.

What is the next best step in his management?

A. Antithymocyte globulin plus ciclosporin
B. Give intravenous glucocorticoid
C. Haematopoietic stem cell transplant
D. Start intravenous human immunoglobulin
E. Urgent platelet transfusion

D. Start intravenous human immunoglobulin

Human immunoglobulin (IVIG) is an alternative to steroids in treating ITP, particularly if the platelets need to be raised quickly

Primary immune thrombocytopenia (ITP, also called idiopathic thrombocytopenic purpura, immune thrombocytopenic purpura) is acquired thrombocytopenia caused by autoantibodies against platelet antigens leading to platelet destruction and platelet underproduction that is not triggered by an apparent associated condition. The likely diagnosis is ITP here as the patient has normal white cell counts and haemoglobin, no other causes of coagulopathy with normal PT/INR, no family history of bleeding disorder and clinically no organomegaly.

IVIG can raise the platelet count within 12 to 24 hours in many patients with ITP; this effect is reproducible enough that a platelet count response to IVIG has been used as a diagnostic criterion for ITP. The effect of IVIG usually persists for two to six weeks. Thus, IVIG is most useful for patients who require a rapid, temporary increase in platelet count (for urgent management of bleeding associated with thrombocytopenia or before an urgent invasive procedure) or those who are unable to tolerate glucocorticoids and are awaiting initiation of second-line therapy.

IV glucocorticoid therapy is incorrect as it's not preferred therapy in this situation where there is a need for urgent surgery. IVIG may be used either as an adjunct to other therapies in an individual with critical bleeding or need for urgent surgery or as an alternative to glucocorticoids in patients with severe bleeding.

Urgent platelet transfusion will be an ineffective treatment in immune (idiopathic) thrombocytopenic purpura especially when planning for immediate surgery as it is controversial, as transfused platelets are short-lived and platelet transfusion has been linked to arterial thrombosis and mortality in platelet consumptive disorders like ITP.

Antithymocyte globulin plus ciclosporin is not the right option for treatment here as it is used in immune-mediated aplastic anaemia.

Haematopoietic stem cell transplant is not applicable in this situation as it is the best treatment for young patients with aplastic anaemia with the best match available.

Question 23 of 191

A 56-year-old woman presents with lethargy and vomiting to her GP. She is normally fit and well. Blood tests and a urine sample are sent.

Hb	110 g/L /L 9	Female: (115 - 160)
Platelets	160 * 10 /L 9	(150 - 400)
WBC	5 * 10 /L 9	(4.0 - 11.0)
Na +	132 mmol/L	(135 - 145)
K +	4.8 mmol/L	(3.5 - 5.0)
Urea	11.1 mmol/L	(2.0 - 7.0)
Creatinine	140 µmol/L	(55 - 120)
Calcium	3.0 mmol/L	(2.1-2.6)
Phosphate	1.2 mmol/L	(0.8-1.4)
Magnesium	0.8 mmol/L	(0.7-1.0)

Urine electrophoresis is positive for Bence Jones protein.

Given the likely underlying diagnosis, what imaging is recommended to assess for bony involvement?

A. DEXA scan
B. Isotope bone scan
C. No imaging required at diagnosis
D. Whole body MRI
E. X-rays of spine and pelvis

D. Whole body MRI

Whole body MRI 1st line imaging in suspected multiple myeloma

The presence of hypercalcaemia, acute kidney injury and Bence Jones proteins in the urine is highly suggestive of a diagnosis of multiple myeloma. In their 2016 guidelines, NICE recommends whole-body imaging at diagnosis of multiple myeloma to assess for bony involvement. They recommend whole-body MRI as first line. Whole-body CT or positron emission tomography (PET) scans can be used as alternatives if MRI is inappropriate or unavailable. The other options are not recommended as first-line imaging modalities in this circumstance by NICE.

A DEXA scan is used in combination with a FRAX tool assessment to assess for future fracture risk in patients with known or suspected osteoporosis.

Nuclear medicine scans such as an isotope bone scan are not recommended as first-line imaging in this situation. Some indications for isotope bone scans include investigating for Paget's disease, osteomyelitis or in primary bone malignancies.

In their 2016 guidelines, NICE recommends whole-body imaging at the time of diagnosis of myeloma to assess for bony involvement.

X-rays can show typical lytic lesions associated with bony involvement in myeloma. However, Xrays are inadequate to exclude bony involvement and are therefore not recommended as first-line imaging in this circumstance.

Question 24 of 191

A 43-year-old man with acute pancreatitis is being treated in the surgical ward. Whilst an inpatient, he experiences recurrent epistaxis, gingival bleeding, and haematuria.

He is re-examined and multiple petechiae are noted as well as some mild confusion. He is somewhat hypotensive when observations are recorded.

Blood tests are taken:

Platelets	83 * 10 /L 9	(150 - 400)
Prothrombin time (PT)	26 secs	(10-14 secs)
Activated partial thromboplastin time (APTT)	48 secs	(25-35 secs)
Fibrinogen	0.3 g/L	(2 - 4)

As well as general resuscitation, what else is most useful, first-line?

A. Cryoprecipitate
B. Fibrinogen concentrate
C. Fresh frozen plasma
D. Platelet transfusion
E. Tranexamic acid

C. Fresh frozen plasma

In bleeding patients with DIC and prolonged prothrombin time (PT) and activated partial thromboplastin time (aPTT), administration of fresh frozen plasma (FFP) may be useful

This patient has developed disseminated intravascular coagulation (DIC), a syndrome characterised by coagulation pathway activation, leading to intravascular thrombi and depletion of platelets and coagulation factors. It is triggered by an underlying condition - in this scenario, acute pancreatitis. As well as treatment of the underlying disorder, the most useful additional option here is **fresh frozen plasma**. This is the preferred method of replacing coagulation factors when bleeding is present or when fibrinogen levels are low.

Cryoprecipitate is a second-line alternative. Fresh frozen plasma is still preferred with regards to DIC.

Similarly, **fibrinogen concentrate** may be used as second-line alternatives. However, fresh frozen plasma is the first-line choice for DIC.

Platelet transfusion is generally considered when the platelet count is less than 20 * 10 /L, or less than 50 * 10 /L in the presence of active bleeding. In this scenario, therefore, this is not as important a consideration as the administration of fresh frozen plasma.

Tranexamic acid may be used in the management of chronic DIC, under specialist guidance. It should be used with extreme caution as it inhibits fibrinolytic pathways, which may worsen thrombosis. In the acute setting, such as this scenario, it does not play a role.

Question 25 of 191

A 23-year-old man presents to the emergency department acutely unwell for the last 72 hours. He has been fatigued for the last month and has been having night sweats and has had several colds. Over the last 3 days, he has become more unwell, shivery and vomiting. He has noticed bruising on forearms and thighs. On examination, he is drowsy and has a temperature of 38.5ºC. His blood pressure is 90/50 mmHg, heart rate 120/min. He is peripherally shut down with a cap refill time of 5 seconds. He has conjunctival pallor. He is given IV fluids and antibiotics by the emergency department. His blood results show:

Hb	89 g/l
Platelets	43 * 10 /l
WBC	13.0 * 10 /l
Neutrophils	9.0 * 10 /l
D-Dimer	5.8mg/L (<0.5)
INR	8.5
PT	89 seconds (9-12)
APTT ratio	1.7 (0.8-1.2)
Fibrinogen	0.1g/L (1.5 - 4.5)
Blood film	Faggot cells seen

Na	138 mmol/l
K	5.8 mmol/l
Urea	18 mmol/l
Creatinine	195 µmol/l
CRP	170 mg/l

Bilirubin	8 µmol/l
ALP	102 u/l
ALT	300 u/l
Albumin	38 g/l

He is transferred to ITU for ionotropic support. He is treated with fresh frozen plasma which corrects his coagulopathy. Haematology is involved and he has a bone marrow analysis performed. Cytogenetics shows a translocation of chromosomes 15 and 17. ***What is the appropriate treatment to give***?

A. All-trans retinoic acid
B. R-CHOP
C. Chlorambucil
D. High dose prednisolone
E. Ribavirin

A. All-trans retinoic acid

This patient has acute promyelocytic leukaemia (M3 subclass). This can present with DIC in young patients the prolonged prothrombin time, with platelets consumption and low fibrinogen. Faggot cells are hypergranular promyelocytes, so called because the high concentrations of Auer rods in the cytoplasm give the cells a bundle of sticks appearance. The gene translocation of 15:17 results in a fusion gene of PML:RARa (retinoic acid receptor). The exact process by which this gene fusion results in AML is unclear but the condition responds well to retinoic acid therapy hence this is the correct answer here. The other options are for other types of haematological malignancy.

Question 26 of 191

A 37-year-old woman was admitted to the Intensive Care Unit (ICU) two days following chemotherapy. She had received her third of six cycles of chemotherapy for malignant ovarian cancer and without initial complication and was discharged the same day. Two days later she felt unwell and developed a fever. After liaising with the oncology unit she was admitted directly to the Medical Admission Unit. A full septic screen was conducted and she was treated empirically for neutropaenic sepsis with intravenous tazocin and gentamicin. Other than malignant ovarian cancer her past medical history was unremarkable, and there was no known metastasis. She was not taking any other drug therapy and was a non smoker. She did not consume alcohol.

Unfortunately whilst on the Medical Admission Unit she continued to deteriorate, developing fluctuating hemiparesis of initially the left lower limb, and then the right upper limb. Her level of consciousness dropped and her speech had become slurred. An urgent transoesophageal echocardiogram and CT head scan was conducted, pending availability of a MRI scan. She was promptly transferred to the Intensive Care Unit.

Upon arrival at the ICU she appeared very unwell, with a GCS of 12/15. Her blood pressure was 188/96 mmHg, her heart rate was 112, her respiratory rate was 22/min and her temperature was 38.5 degrees celsius. Examination of the cardiovascular system revealed the presence of normal heart sounds, a JVP of 3cm and warm well perfused peripheries. Examination of the respiratory system revealed good air entry in both lungs, with an oxygen saturation of 95% on air. Examination of her gastrointestinal system was unremarkable. Examination of her neurological system revealed localization to pain stimulus, with confusion and eye opening only in response to verbal prompting. Her speech was slurred. There was no other apparent focal neurological deficit, with otherwise normal cranial nerve and peripheral nervous system testing.

The results of the investigations conducted are as follows:

Hb	89g/l
Platelets	38 * 10 /l
WBC	15.2 * 10 /l
Reticulocyte count	4% (ie above normal range)
Blood film	presence of schistocytes, normocytic normochromic anaemia

Na	136 mmol/l
K	6.1 mmol/l
Urea	10.1 mmol/l
Creatinine	154 µmol/l
CRP	22 mg/l
ESR	45 mm/hr
Protein	78 g/l
Albumin	36 g/l
Adj calcium	2.42 mmol/l
Phosphate	0.95 mmol/l
Bilirubin	44 µmol/l
ALT	39 u/l
LDH	1286 u/l
ALP	102 u/l
PTT	14s
APTT	44s
INR	1.1
D-dimer	136 ng/ml

Chest x-ray: normal heart and lung appearances

ECG: heart rate 107bpm normal sinus rhythm, normal QRS and QTc intervals Urinalysis: proteinuria ++, haematuria +, leuc/nit/glu negative

Blood MCS x3: pending result Urine MCS: pending result

CT head: no space occupying lesion, mass shift or intracerebral haemorrhage seen Transoesophageal echocardiogram: normal systolic function, normal appearance of all valves, no evidence of vegetation seen

In the context of the likely underlying diagnosis, what is the best immediate management step?

A. Commence immediate plasma exchange
B. Commence high dose intravenous hydrocortisone
C. Commence platelet infusion
D. Commence IV meropenem and IV antifungal therapy
E. Commence fresh frozen plasma infusion

A. Commence immediate plasma exchange

This patient has developed thrombotic thrombocytopaenia purpura (TTP) secondary to chemotherapy, as manifested by the combination haemolytic anaemia, thrombocytopaenia and acute renal impairment. The presence of a fever and neurological involvement differentiates this condition from haemolytic-uraemic syndrome. Of the above options, plasma exchange holds the best prognosis for this condition and is, therefore, the best option to be initiated without delay, but in practice glucocorticoids and fresh frozen plasma infusions are often given pending arranging plasma exchange.

Question 27 of 191

A 22-year-old man with sickle cell disease is seen in the Emergency Department. He has had worsening pain in his arms and legs for the last 2 days and 5 hours ago developed a painful sustained erection.

He has felt otherwise well recently and has no other past medical history. He is on regular paracetamol, ibuprofen, folate and penicillin. He does not receive regular transfusions and has been admitted with a crisis only once before. He has never had an episode of painful sustained erection.

On examination he has a heart rate of 110 beats per minute and a blood pressure of 132/95 mmHg. His oxygen saturations are 96% on room air and he is afebrile. His chest is clear and abdomen is soft. He has no swelling or erythema of his limbs, though they are generally tender. He continues to have an erection, though there is no sign of ischaemia.

His chest x-ray shows clear lung fields. His blood tests are as follows:

Hb	78 g/l	Na +	141 mmol/l
Platelets	331 * 10 /l 9	K +	3.7 mmol/l
WBC	9 * 10 /l 9	Urea	5 mmol/l
Neuts	7 * 10 /l 9	Creatinine	86 µmol/l
Lymphs	1.6 * 10 /l 9	CRP	14 mg/l

He is treated with intravenous fluids and generous analgesia with diamorphine. His limb pain is improved but he continues to have a painful erection.

What is the next most appropriate step

A. Adrenalin
B. Diethylstilbestrol
C. Exchange transfusion
D. Review by urologist
E. Sildenafil

D. Review by urologist

This gentleman has priapism, a sustained painful erection lasting 4 hours. If left untreated it can result in impotence. Current guidelines recommend initial conservative management with fluids and analgesia. Although any interventions beyond this have variable results, evidence supports prompt urology review to determine the need for surgical management including drainage. There is insufficient evidence to recommend routine exchange transfusions. Sildenafil, diethylstilbestrol and adrenalin may be used in consultation with urology but evidence of benefit is variable.

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A 23 year old male of Nigerian descent is referred from the ED with acute shortness of breath associated with fever and dry cough. His shortness of breath has limited him to an exercise tolerance of around 10 yards. He also complains of excruciating right sided chest pain. He is known to have sickle cell anaemia and has had no admissions to the hospital in the last 3 years.

Observations

- heart rate 94bpm regular
- blood pressure 112/74 mmHg
- temperature 38.3
- respiratory rate 22
- urine output under 30ml/hr oxygen saturations 93% on room air

Examination

respiratory system shallow breathing, bronchial breath sounds with crepitations to right base

Blood results:

Hb	7.7 g/dl
Platelets	200 * 10 /l9
WBC	13.2 * 10 /l9
Bilirubin	36 µmol/l
Urea	8.2 mmol/l
Creatinine	146 µmol/l

What is the most important initial steps in management?

A. Oxygen, IV fluids, antibiotics and analgesia
B. 2 unit blood transfusion and analgesia
C. Oxygen and analgesia only
D. Antibiotics and IV fluid only
E. Plasmapheresis and analgesia

A. Oxygen, IV fluids, antibiotics and analgesia

It is important to recognise and treat sickle cell crises (as detailed above) as a matter of urgency. This condition can be quickly fatal. This patient should be treated as per sepsis guidelines. It is crucial to adequately control the pain in this patient cohort as not being able to inhale deeply due to pain will only serve to worsen the lung damage. Early assessment for the involvement of the critical care team is required as acute respiratory distress syndrome is a real possibility. Oxygen saturation levels should be maintained above 95% in a patient where a normal steady state level is not known. Management of this and the haemaglobin level should be specific for the patient and compared to past levels. The question states that the patient has had no admissions to the hospital in the last 3 years and as such the aim should be to keep oxygen saturations above 95%.

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A 64-year-old female undergoing chemotherapy treatment with mitomycin for bladder cancer presents to the Emergency Department with confusion, epistaxis and a widespread rash. On examination, she is febrile and has a diffuse petechial rash.

Hb	10.6g/dl
Platelets	54 * 10 /l9
WBC	11.2 * 10 /l9
Urea	11mmol/l
Creatinine	117µmol/l
Bilirubin	58µmol/l
ALP	42u/l

A blood film shows fragmented erythrocytes.

What is the most appropriate treatment for this patient?

A. Renal dialysis
B. Supportive treatment
C. A pool of platelets and fluid resuscitation
D. Urgent plasma exchange
E. IV tazocin, a pool of platelets and fluid resuscitation

D. Urgent plasma exchange

Thrombotic thrombocytopenic purpura (TTP) is characterised by fluctuating neurological signs, fever, renal dysfunction, microangiopathic haemolysis and thrombocytopenia. The key to diagnosis is differentiating between TTP and haemolytic-uraemic syndrome (HUS). The presence of neurological signs in TTP and more severe renal dysfunction in HUS usually helps in distinguishing the two.

Congenital TTP is caused by a deficiency in von Willebrand factor. However, the majority of cases are secondary and can occur in pregnancy, HIV and associated with some drugs. In particular, the chemotherapeutic agents mitomycin C, bleomycin, tamoxifen and gemcitabine are all associated with secondary TTP. Other drugs in association include penicillin, rifampicin and mmunosuppressive drugs eg. ciclosporin A.

The standard treatment for TTP is IV plasma exchange which should be initiated as soon as possible.

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A 22-year-old man attends the emergency department with a group of friends from a university party. He complains of worsening shortness of breath and light-headedness. He appears panicked and is clutching his chest. There is no past medical history of note and he has no allergies.

His observations are as follows:

- Temperature 36.2ºC
- Heart rate 112 beats/min
- Blood pressure 120/75mmHg
- Respiratory rate 24 breaths/min
- Oxygen saturations 90% on air

On examination, his lips are cyanosed. His chest is clear with good air entry bilaterally and his heart sounds are normal. Peripheral pulses are present with a capillary refill of <2 seconds and his calves are soft and non-tender.

Arterial blood gas:

pH	7.21	(7.35 - 7.45)
pO_2	12.1 kPa	(11 - 14.4)
pCO_2	2.4 kPa	(4.6 - 6.4)
Bicarbonate	16 mmol/L	(22 - 29)
Chloride	100 mmol/L	(95 - 108)
Lactate	1.2 mmol/L	(0.5 -2.2)
Glucose	5.6 mmol/L	(4 - 7)

Given the likely diagnosis, which of the following is the most appropriate next step in the management of this patient?

A. Fomepizole
B. Hyperbaric oxygen
C. Methylene blue
D. Thrombolysis
E. Intravenous 1.26% sodium bicarbonate

C. Methylene blue

Methylene blue can be used to treat methaemoglobinaemia. It causes a reduction of Fe3+ (ferric) to Fe2+ (ferrous)

This patient has methaemoglobinaemia, likely secondary to drug misuse (e.g. amyl nitrite poppers) at a party. In patients with methaemoglobinaemia, haemoglobin has been oxidised from Fe^{2}+ to Fe^{3}+. This oxidised form of haemoglobin cannot bind to oxygen and therefore symptoms include cyanosis, shortness of breath and headache. In more severe illnesses, investigations may detect metabolic acidosis and arrhythmias. During methaemoglobinaemia, a normal PO may be seen with reduced oxygen saturations, similar to this patient. The treatment of choice for methaemoglobinaemia is intravenous methylene blue, which causes a reduction reaction to convert Fe^{3}+ back to Fe^{2}.

Fomepizole is used in the treatment of ethanol toxicity. Symptoms of ethanol toxicity are initially similar to alcohol excess with confusion, slurred speech and altered gait. Metabolic acidosis and acute kidney injury follow. However, ethanol toxicity does not explain this patient's hypoxia and cyanosis and is less likely to be correct.

Hyperbaric oxygen therapy is used in the treatment of carbon monoxide poisoning. Unlike methaemoglobinaemia, symptoms of carbon monoxide poisoning include headache, confusion and nausea and vomiting. Respiratory symptoms are less predominant compared to methaemoglobinaemia. Carbon monoxide poisoning tends to produce a normal oxygen saturation level with a low PO , unlike this patient. It also does not explain this patient's metabolic acidosis and suggests an alternative diagnosis.

Thrombolysis would be an option for the treatment of a thrombotic cause of this patient's symptoms (e.g. pulmonary embolism). However, patients with a pulmonary embolism are more likely to have a metabolic alkalosis secondary to hyperventilation, in contrast to this patient with metabolic acidosis.

Sodium bicarbonate can be used to treat severe metabolic acidosis. However, it is not used firstline in the treatment of methaemoglobinaemia and the priority should be to reverse the oxidation process of haemoglobin..

Question 31 of 191

A 33-year-old patient presents to the emergency department feeling very lethargic and tired over the last week. He has been increasingly short of breath on exertion and his exercise tolerance has fallen to a few hundred yards before he is out of breath. He is a known sickle cell disease patient who is managed by the Haematology team in your hospital. On examination he is afebrile and heart rate and blood pressure are within normal limits. His respiratory rate is 16/min and his oxygen saturation is 98% on air. He does become noticeably short of breath on minimal movement.

His blood tests show:

Hb	65 g/l
Platelets	46 * 10 /l
WBC	2.5 * 10 /l
Neuts	1.2 * 10 /l
Haptoglobins	1.9 g/L (0.3-2.0)
Reticulocytes	8.9 x10^9/L (25-80)
Na	136 mmol/l
K	3.9 mmol/l
Urea	7.4 mmol/l
Creatinine	78 µmol/l
CRP	<3 mg/L(<10)
LDH	200 IU/L (200-500)
Bilirubin	4 µmol/l
ALP	89 u/l
ALT	34 u/l
Albumin	39 g/l

His chest x-ray is normal.

On further questioning, he tells you that his 5-year-old daughter was unwell 3 weeks ago. He took her to see the GP and was told it was likely to be a viral illness.

What is the most likely cause for his blood results as shown?

A. Parvovirus B19
B. Acute chest crisis
C. Splenic sequestration
D. HIV
E. Legionella pneumoniae

A. Parvovirus B19

This gentleman has had an aplastic sickle cell crisis as a result of parvovirus infection. He has symptomatic anaemia. There is nothing to suggest splenic sequestration and he is too well for an acute chest syndrome. The normal chest x-ray is not in keeping with a pneumonia. HIV is a possibility but less likely given the clinical picture.

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A 79-year-old woman has been referred to the clinic due to weight loss, right-sided chest pain, lower back pain, and general fatigue.

Hb	96 g/L	Male: (135-180)Female: (115 - 160)
Platelets	399 * 10 /L 9	(150 - 400)
WBC	10.2 * 10 /L 9	(4.0 - 11.0)
Na +	145 mmol/L	(135 - 145)
K +	4.5 mmol/L	(3.5 - 5.0)
Urea	15.2 mmol/L	(2.0 - 7.0)
Creatinine	190 µmol/L	(55 - 120)
Calcium	3.1 mmol/L	(2.1-2.6)
Phosphate	1.4 mmol/L	(0.8-1.4)
Magnesium	0.7 mmol/L	(0.7-1.0)

Urine Bence Jones: positive

She is usually fit and well with no past medical history.

What would be the first-line imaging to confirm the most likely diagnosis?

A. CT thorax, abdomen, and pelvis
B. Chest radiograph
C. PET CT
D. Skeletal survey
E. Whole-body MRI

A. Whole-body MRI

Whole body MRI 1st line imaging in suspected multiple myeloma

The most likely diagnosis here is multiple myeloma given the weight loss, bone pain, anaemia, renal impairment, and hypercalcemia.

Further tests should be sent to clarify this diagnosis including serum free light chains, serum protein electrophoresis, and immunoglobulins.

It is worth noting that guidance regarding the answer to this question may vary from hospital to hospital depending on availability, but national guidance recommends whole-body MRI as firstline imaging.

Whole-body MRI is the correct answer. Part of the diagnostic criteria for multiple myeloma is the presence of 1 or more osteolytic lesions of ≥5 mm. This is most sensitively picked up on MRI scanning, which can also pick up pre-lytic lesions that may be amenable to biopsy.

CT thorax, abdomen, and pelvis is incorrect. CT scanning has a lower sensitivity for osteolytic lesions. There is also higher radiation exposure.

PET CT is incorrect. This type of scanning can be helpful for looking at intramedullary involvement, but the consensus from current best evidence is that MRI is preferable particularly to identify osteolytic lesions.

Skeletal survey is incorrect. It is less sensitive and specific compared to PET CT and MRI scanning. This is the preferred option for patients who are unable to undergo whole-body MRI, PET CT, or CT. It takes less time to perform compared to whole-body MRI (25 minutes as opposed to 60 minutes).

Chest radiograph is incorrect. Although this is a sensible first-line test to perform in the acute setting, this question asks specifically about the first-line test for the most likely diagnosis here - multiple myeloma. Chest radiograph is therefore incorrect.

Question 33 of 191

A 23-year-old female has presented with her first episode of seizure on the labour ward, 2 days after delivering her first child by normal vaginal delivery. She reports a fluctuating generalised headache over the past 3 months but had not previously sought medical attention. In addition, she had spiked 2 fevers over 38ºC over the past 48 hours, with no dysuria, diarrhoea or vomiting, productive cough or signs of meningism. She has no past medical history, is a life-long nonsmoker and has been abstinent from alcohol for 9 months, previously drinking 4 units per week. Her seizure was witnessed and described as tonic-clonic jerking of all 4 limbs, associated with loss of consciousness, terminated after 4mg of intravenous lorazepam after 4 minutes. On examination, she appears post-ictal but responding to voice despite being sleepy. Pupils are reactive and equal. Plantars are downgoing bilaterally. Cardiovascular, abdominal and respiratory examinations are unremarkable. No skin rashes, neck stiffness or photophobia are noted.Her blood results are as follows:

Hb	75 g/l
MCV	87 fl
Platelets	23 * 10 /l9
WBC	9.2 * 10 /l9
Blood film	Schistocytes
Coombs' test	Negative
CRP	30 mg/l
Urea	12.6mmol/l
Creatinine	154 µmol/l
Bilirubin	28 µmol/l
ALP	98 u/l
ALT	28 u/l
γGT	23 u/l

A CT head with contrast demonstrated no areas of ischaemia, haemorrhage or space occupying lesion.

Which is the next most appropriate immediate management?

A. Plasma exchange
B. Intravenous 3rd generation cephalosporin antibiotics
C. Intravenous phenytoin loading
D. MRI head with contrast
E. Intravenous steroids

A. Plasma exchange

This young lady's blood tests demonstrated a normocytic anaemia associated with schistocytes (fragmented red cells) with negative Coombs' test, suggestive of mechanical destruction of red cells. In addition, thrombocytopaenia is present with renal impairment and normal liver function tests. The initial presentation is of first seizure and fluctuating headache, with two unexplained fevers. The pentad of fever, fluctuating neurological symptoms, microangiopathic haemolytic anaemia, thrombocytopaenia and renal impairment represents thrombotic thrombocytopaenic purpura-haemolytic uraemic syndrome spectrum (TTP-HUS), resulting in microvascular thrombus formation. There is little to suggest an underlying epilepsy syndrome and no indication for phenytoin loading after a single seizure without status epilepticus. While meningitis may cause headaches, a fluctuating 3-month history without meningism is unlikely.

Immediate treatment of TTP-HUS is reliant on plasma exchange without delay in order to remove the high-molecular weight von-Willebrand factor (vWF) driving platelet aggregation and replacing ADAMTS-13 protease, a cleavage enzyme of high molecular weight vWF often deficient in TTP patients, resulting in microvascular thrombi formation. High dose steroids may be appropriate in the setting of refractory disease despite plasma exchange but is an adjunct to plasma exchange, not in place of. Persistent recurrent or refractory TTP-HUS despite plasma exchange and steroids should be considered for the addition of rituximab or increased frequency of plasma exchange. TTP-HUS is a medical emergency and invariably results in death secondary to progressive renal failure if not treated immediately.

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A 22-year-old man presents to the emergency department with shortness of breath. He takes no regular medicines and denies the use of herbal, or over-the-counter medications. However, he does admit to using amyl nitrites recreationally.

Observations are as follows: heart rate 125 beats per minute, respiratory rate 24 breaths per minute, blood pressure 125/75 mmHg, temperature 37.2 ºC, and SpO2 92%. His chest is clear on auscultation.

He is started on 15L O2 via a non-rebreather mask. Blood gas analysis results are as follows:

pH	7.26	(7.35 - 7.45)
PaO2	72.4 kPa	(10.3 - 13.3 kPa)
PaCO2	2.9 kPa	(4.7 - 6 kPa)
HCO3	18 mmol/L	(22 - 28 mmol/L)
SaO2	48%	(94 - 98%)
Lactate	3.2 mmol/L	(<1.0)

What treatment is indicated?

A. Ascorbic acid
B. Dicobalt edetate
C. Hydroxocobalamin
D. Hyperbaric oxygen
E. Methylthioninium chloride

A. Methylthioninium chloride

Methylene blue can be used to treat methaemoglobinaemia. It causes a reduction of Fe3+ (ferric) to Fe2+ (ferrous)

The striking feature, in this case, is the significant discrepancy between the PaO2 and SaO2 on arterial blood gas analysis.

Given the patient is on 15L O2, the PaO2 is at a level expected indicating that gas exchange at the level of the alveoli is not impaired. There are various formulas for predicting the expected PaO2 for any given inspired O2 concentration. The Advanced Life Support (ALS) guidelines suggest that the PaO2 for any given inspired concentration should be approximately 10 less than the inspired concentration (%). For example, in this case, 15L O2 is equivalent to approximately 80-85% so we would expect the PaO2 to be around 70 kPa.

However, it is important to note that the SaO2 (oxygen saturation of haemoglobin) is markedly low at 48% indicating impaired oxygen binding to haemoglobin. It is also important to note that the peripheral SpO2 is 92% which is clearly discrepant with the SaO2.

The two main causes of this picture are methaemoglobinaemia and carboxyhaemoglobinaemia. Because standard pulse oximeters are not capable of differentiating between different forms of bound haemoglobin, the SpO2 is usually falsely high in these conditions.

Tissue hypoxia can be a consequence of 4 main causes:

1. Hypoxaemia (low PaO2 due to poor gas exchange at the alveoli)

2. Toxic haemoglobin (low SaO2 due to methaemoglobinaemia or carboxyhaemoglobinaemia)

3. Perfusional (e.g. shock, or localised causes such as compartment syndrome or bowel

ischaemia)

4. Severe anaemia

Regardless of the cause, tissue hypoxia will result in lactic acidosis. Therefore, in patients with lactic acidosis, all the aforementioned causes should be considered. The patient in this case most certainly has lactic acidosis as a consequence of toxic haemoglobin-induced tissue hypoxia. He is acidotic with low serum bicarbonate since this acts as a buffer for lactic acid.

Methylthioninium chloride is correct. The patient has a diagnosis of acquired methaemoglobinaemia. As discussed above, the biochemical results are suggestive of toxic haemoglobin. The history of nitrate exposure favours the diagnosis of acquired methaemoglobinaemia. The treatment of choice is IV methylthioninium chloride (methylene blue).

Ascorbic acid is incorrect. This drug can be used to treat congenital methaemoglobinaemia. Although this remains within the differential diagnosis, the absence of previous episodes and the presence of a known risk factor (e.g. nitrate exposure) favours the acquired form.

Dicobalt edetate and **Hydroxocobalamin** are incorrect. These drugs can be used to treat cyanide poisoning. Cyanide binds the ferric (Fe3+) ion of cytochrome oxidase causing 'histotoxic hypoxia' and lactic acidosis. It would not cause a marked reduction of SaO2 as seen in this case unless it was associated with coexistent carbon monoxide poisoning (e.g. due to smoke inhalation). There is nothing in this clinical case to suggest that this is the case.

Hyperbaric oxygen is incorrect. This can be used to treat severe carboxyhaemoglobinaemia.

Although carbon monoxide poisoning presents similarly and remains within the differential diagnosis, the history of nitrate exposure favours the diagnosis of methaemoglobinaemia.

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A 31-year-old lady presents to the emergency department with abdominal pain.

Her medical and surgical history is unremarkable other than mild depression and hay fever. Medications include paroxetine and PRN loratadine.

She complains of generalised colicky abdominal pain and has vomited once in the department.

On examination blood pressure is 155/86mmHg, heart rate is 95bpm and temperature is 37.9ºC. Digital rectal examination reveals hard stool in the rectum.

Investigations reveal:

Hb	131 g/l
Platelets	362 * 10 /l9
WBC	7.3 * 10 /l9
Na +	121 mmol/l
K +	3.3 mmol/l
Urea	6.2 mmol/l
Creatinine	87 µmol/l
Urine dipstick	protein ++, leucocytes ++

What is the most likely diagnosis?

A. Cholecystitis
B. Acute porphyria
C. Intestinal obstruction
D. Pyelonephritis
E. Systemic lupus erythematosus

B. Acute porphyria

The normal white cell count make infective diagnoses such as pyelonephritis and cholecystitis less likely. Intestinal obstruction usually presents with an empty rectum.

The history of neuropsychiatric symptoms along with hypertension and hyponatraemia are suggestive of acute porphyria.

Systemic lupus erythematosus doesn't tend to present with abdominal pain.

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A 34-year-old woman is rushed to the emergency department by her husband. Her husband says that she has been lethargic for the last 2 days and this morning has become increasingly more confused and behaving differently to normal. She has no past medical history of note. She is 16 weeks pregnant.

On examination, she is febrile with a temperature of 38.5ºC. She is tachycardic with a heart rate of 125 bpm and has a blood pressure of 115/95mmHg. On auscultation, her chest is clear with normal heart sounds. Her abdomen is soft and non-tender with a palpable uterus in keeping with 16 weeks gestation.

Investigations:

Hb	92 g/L	Male: (135-180)Female: (115 - 160)
Platelets	43 * 10 /L 9	(150 - 400)
WBC	6.3 * 10 /L 9	(4.0 - 11.0)
Na +	138 mmol/L	(135 - 145)
K +	3.8 mmol/L	(3.5 - 5.0)
Urea	11.2 mmol/L	(2.0 - 7.0)
Creatinine	185 µmol/L	(55 - 120)
Bilirubin	32 µmol/L	(3 - 17)
ALP	35 u/L	(30 - 100)
ALT	85 u/L	(3 - 40)
Albumin	37 g/L	(35 - 50)
CRP	< 1 mg/L	(< 5)

Urinalysis: negative.

Pregnancy test: positive. What is the next immediate treatment for this patient?

A. Intravenous antibiotics
B. Intravenous immunoglobulin
C. Magnesium sulphate
D. Plasma exchange
E. Platelet transfusion

D. Plasma exchange

TTP - plasma exchange is first-line

The pentad of symptoms: AKI, fever, cerebral dysfunction, thrombocytopenia and microangiopathic haemolytic anaemia should point towards a diagnosis of thrombotic thrombocytopenic purpura (TTP). TTP is a serious condition that causes dysregulation of the clotting cascade. The underlying mechanism is not completely understood but a deficiency in the enzyme ADAMTS13 has been identified. If left untreated, TTP has a high mortality. Pregnancy is a known cause of TTP although it does not have to be present to make the diagnosis. If TTP is suspected, the first-line treatment of choice is plasma exchange. Plasma exchange removes any antibodies which inhibit ADAMTS13 whilst also replacing the functioning version of the enzyme. Steroids and rituximab have also been used to suppress the patient's immune system from producing enzymes to ADAMTS13.

Intravenous antibiotics are an appropriate consideration, given that this patient is febrile. In the absence of raised inflammatory markers, it seems more likely that fever is a symptom of TTP rather than an underlying infection. Nevertheless, even if given, antibiotic administration will not treat TTP.

Intravenous immunoglobulin is used in the treatment of refractory TTP. However, it is not the firstline treatment of choice.

Magnesium sulphate is used in the treatment of eclampsia. It is not used in the treatment of TTP.

Platelet transfusion is not recommended in TTP. Transfusion is associated with an increased risk of thrombosis and mortality.

Question 37 of 191

A 28-year-old man is admitted to the Medical Admissions Unit with a 2-day history of itching, right upper quadrant pain, and abdominal distension. The pain started as a dull ache but became constant and severe over the course of several hours.

His past medical history is remarkable only for a left lower limb DVT diagnosed at age 20. He takes no regular medications and he is a non-smoker. He drinks 2-3 units of alcohol per week and denies intravenous drug use.

Examination reveals a jaundiced young man with pale conjunctivae. He appears deeply uncomfortable. His abdomen is moderately distended with marked right upper quadrant tenderness. His liver and spleen are both palpable 2cm below the costal margin. Shifting dullness is demonstrable on percussion of the abdomen.

His blood results are as follows:

Hb	101 g/l	Na +	139 mmol/l	Bilirubin	109 µmol/l
MCV	102.4 fl	K +	4.2 mmol/l	ALP	284 u/l
Platelets	63 * 10 /l 9	Urea	6.7 mmol/l	ALT	684 u/l
WBC	12.9 * 10 /l 9	Creatinine	108 µmol/l	γGT	179 u/l
Neuts	10.8 * 10 /l 9	Albumin	27 g/l		
Lymphs	0.9 * 10 /l 9	LDH	759 u/l		

His abdominal ultrasound scan is consistent with hepatic vein thrombosis and the patient is started on low molecular weight heparin. Following a review by the Haematologists, a diagnosis of paroxysmal nocturnal haemoglobinuria is made and the patient is advised to start treatment with eculizumab.

Given the proposed treatment strategy, which of the following vaccinations should the patient be offered?

A. Hepatitis B
B. Neisseria meningitidis
C. Varicella zoster virus
D. Streptococcus pneumoniae
E. Haemophilus influenzae type b

B. Neisseria meningitidis

Eculizumab is a recombinant humanised monoclonal antibody that specifically binds to terminal complement protein C5. Patients with C5 deficiency are at elevated risk of serious meningococcal infections and all patients being treated with eculizumab should receive a quadrivalent vaccine against the meningococcal strains A, C, W, and Y

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A 32-year-old male presents with a progressive worsening non-specific lethargy. 9 months ago, he had returned from an active holiday from New Zealand and now feels lethargic to the point that he can no longer work in his job as a computer programmer. In this period, he has been treated for two deep vein thromboses with low molecular heparin, the first initially attributed to his return flight from New Zealand. He reports three episodes of rose coloured urine over the past 4 months and intermittent episodes of abdominal cramps that his GP had diagnosed to be irritable bowel syndrome.

On examination, you note mild conjunctival pallor and jaundiced sclera. Respiratory, cardiovascular and abdominal examinations are unremarkable. His blood results are as follows:

Hb	76 g/l
MCV	92 fl
Platelets	276 * 10 /l9
WBC	4.1 * 10 /l9
Reticulocytes	18%
Haptoglobin	2 (normal range 41-165 mg/dL)
LDH	2128 (normal range 140-280 units/L)
Coombs' test	negative at 4 and 37 degrees

What is the definitive treatment for the underlying condition?

A. Packed red blood cell transfusion

B. Anti-retroviral treatment
C. Bone marrow transplant
D. R-CHOP chemotherapy
E. Intravenous iron replacement

C. Bone marrow transplant

This is a tricky clinical scenario with a number of red herrings: a young man in his 30s has presented with recurrent DVTs, episodes of haematuria, abdominal cramps and a blood picture suggestive of intravascular haemolysis (low haptoglobin, raised LDH) not secondary to an autoimmune cause (Coombs negative): there is thus an underlying red cell fragility predisposing to thrombosis, strongly suggestive of paroxysmal nocturnal haemoglobinuria (PNH). Note that haemoglobinuria is not restricted to night-time alone. The majority of patients present in their 30s with thromboses being the most common cause of death.

PNH is caused by an underlying reduced CD 59 on the red cell surface, leading to increased susceptibility to complement lysis. Complications can present following the release of haemoglobin, such as pulmonary hypertension, dystonia and renal impairment while PNH can overlap with aplastic anaemia and myelodysplasia. PNH patients are managed by blocking complement lysis with eculizumab and red cell transfusions but the only curative solution is allogenic bone marrow transplantation.

Question 39 of 191

A 24-year-old female presents with severe abdominal pain. This is her fourth presentation this year. She was previously told that the abdominal pain was medically unexplained. On examination you note peripheral neuropathy of her lower limbs. Her blood pressure is 160/110 mmHg and heart rate 125 beats per minute.

Blood results are as follows:

Hb	115 g/l	Na^+	132 mmol/l
Platelets	468 * 10 /l 9	K^+	3.8 mmol/l
WBC	14.2 * 10 /l 9	Urea	8.6 mmol/l
Neuts	10.8 * 10 /l 9	Creatinine	72 µmol/l
Lymphs	1.6 * 10 /l 9	CRP	28 mg/l
Eosin	0.2 * 10 /l9		
Urine	Increased levels of delta aminolevulinic acid and porphobilinogen		

What treatment is indicated?

A. Dimercaprol
B. Penicillamine
C. Dimercaptosuccinic acid (DMSA)
D. Haem arginate
E. Supportive management

D. Haem arginate

IV haem arginate can be used to treat flares of acute intermittent porphyria

The clinical features and laboratory data are suggestive of acute intermittent porphyria (AIP). This is one of the hereditary hepatic porphyrias. Its inheritance is autosomal dominant. The deficient enzyme is porphobilinogen deaminase (PBGD). A deficiency of PBGD is not sufficient by itself to produce AIP, and other activating factors must also be present. These include hormones, drugs and dietary changes.

Hematin and heme arginate is the treatment of choice during an acute attack of AIP. Heme is not a curative treatment, but can shorten attacks and reduce the intensity of an attack.

Dimercaprol, penicillamine and dimercaptosuccinic acid (DMSA) can be used to treat lead toxicity. Lead toxicity can present similarly to AIP (e.g. abdominal pain and peripheral neuropathy). In lead poisoning we would expect increased urinary coproporphyrin.

Question 40 of 191

A 30-year-old woman presents to the emergency department with severe, progressive abdominal pain over the past day. The pain is accompanied by nausea, vomiting and diarrhoea. The patient recalls similar episodes in the past that progressed over a few days and lasted for a week. Temperature is 37°C, blood pressure is 140/100 mmHg, pulse is 120/min and respirations are 16/min.

On examination: minimal abdominal tenderness and rebound tenderness. She has a history of abdominal surgery for suspected appendicitis and biliary disease, neither of which was confirmed once inside the abdomen.

Which of the following will help to confirm the diagnosis?

A. Erythrocyte porphyrins
B. Faecal porphyrins
C. Plasma porphyrins
D. Urine porphobilinogen
E. Urine porphyrins

D. Urine porphobilinogen

In acute intermittent porphyria, urinary porphobilinogen is typically raised

This patient has acute intermittent porphyria, which classically presents with neurovisceral symptoms which can mimic an acute abdomen. This mimicry occurs because the abdominal pain is produced by a nerve problem rather than inflammation, hence why exploratory surgery is uneventful. In long-standing cases, patients may have damage to their motor nerves resulting in upper limb weakness.

Question 41 of 191

A 40-year-old woman of Mediterranean descent was found to be anaemic during a workup for fatigue. Past medical history includes hypothyroidism, which is currently well controlled on 125mcg levothyroxine, and hypertension for which she takes amlodipine 5mg.

She denies any recent weight loss or change in bowel habits and her periods remain regular and are no heavier than normal. Blood tests results showed:

Hb	85g/l	115-165g/l
MCV	60fL	79-95fL
Iron	32nmol/L	13-32nmol/l
Ferritin	250µg/l	11-307µg/l

TIBC	50µmol/l	45-70µmol/l
Folate	2.5µg/l	3.1-19.9µg/l
B12	253ng/l	145-910ng/l

Which test is most likely to identify the underlying cause of her anaemia?

A. Anti-tissue transglutaminase (anti-tTG) antibodies
B. Colonoscopy
C. Prussian blue stain
D. Haemoglobin electrophoresis
E. Blood film

D. Haemoglobin electrophoresis

Disproportionately low MCV think thalassaemia

The differential diagnosis for microcytic anaemias includes iron deficiency anaemia (IDA), thalassaemia, and sideroblastic anaemia. Colonoscopy is used to investigated unexplained IDA but serum iron and ferritin levels were raised in this patient, thus excluding IDA.

The disproportionately low MCV compared with haemoglobin levels points towards thalassaemia where iron and ferritin levels can often be normal or high. Whilst a blood film would aid the diagnosis, HbA2 >3.5% on haemoglobin electrophoresis is diagnostic.

Prussian blue stains are used in the diagnosis of sideroblastic anaemia but one would expect significantly raised iron levels and reduced TIBC.

Anti-tTGs are used in the diagnosis of coeliac disease which often causes a mixed iron and folate deficiency. Whilst folate levels are low in this patient, this can also be explained by a chronic background level of haemolysis in thalassaemia and the other results are not in keeping with a coeliac picture.

Question 42 of 191

A 46-year-old man with a diagnosis of chronic hepatitis C is reviewed in hepatology clinic. He complains of 3 weeks of lethargy and generalised muscle pain.

On examination there are erythematous macules and purpuric papules on both lower limbs with some small areas of ulceration. Light touch and pain sensation is reduced in the toes bilaterally.

What is the most likely cause of these symptoms?

A. Eosinophilic granulomatosis with polyangiitis
B. Leukocytoclastic vasculitis
C. Granulomatosis with polyangiitis
D. Cryoglobulinaemia
E. Microscopic polyangiitis

D. Cryoglobulinaemia

Hepatitis C is associated with mixed (type II) cryoglobulinaemia

A vasculitic rash and neuropathy in a patient with hepatitis C is suggestive of cryoglobulinaemia.

Small-vessel vasculitides can all cause neuropathy, however the association with hepatitis C makes cryoglobulinaemia most likely.

Patients with eosinophilic granulomatosis with polyangiitis (formerly known as Churg-Strauss syndrome) typically present with a history of allergic rhinitis and asthma. The disease progresses to eosinophilic infiltrative disease (eg, eosinophilic pneumonia or gastroenteritis) and systemic medium- and small-vessel vasculitis with granulomatous inflammation.

Granulomatosis with polyangiitis (GPA), formerly known as Wegener's granulomatosis has a spectrum of presentations but often includes chronic sinusitis (failure to respond to conventional treatment is suggestive). Rhinitis and epistaxis occur in 22% and 11% of patients with GPA, respectively.

Question 43 of 191

A 65-year-old man attends the Emergency Department with a one-month history of persistent left-sided chest pain that is unresponsive to analgesia. He describes the pain as a constant ache, which does not worsen with exertion or radiate elsewhere. His medical background includes hypertension and diabetes, and he has a significant smoking history. His current medication regimen comprises enalapril, metformin, vildagliptin, and glimepiride.

Hb	110 g/L	Male: (135-180)
Platelets	210 * 10 /L $_9$	(150 - 400)
WBC	6.4 * 10 /L $_9$	(4.0 - 11.0)
Calcium	2.8 mmol/L	(2.1-2.6)
Na $^+$	138 mmol/L	(135 - 145)
K $^+$	4.4 mmol/L	(3.5 - 5.0)
Urea	7.2 mmol/L	(2.0 - 7.0)
Creatinine	235 µmol/L	(55 - 120)

No cardiac abnormalities were detected on ECG or echocardiography.

What is the most appropriate imaging modality for this patient?

A. Bone scan
B. Chest x-ray
C. PET-CT
D. Skeletal survey
E. Whole body MRI

A. Whole body MRI

Whole body MRI 1st line imaging in suspected multiple myeloma

Whole body MRI is the correct answer. The constellation of symptoms, including left-sided chest pain (bony aches), anaemia, hypercalcaemia, and renal impairment, should prompt consideration of multiple myeloma. Whole-body MRI is the recommended modality for diagnosis when multiple myeloma is suspected, as per NICE and BSH guidelines. Diagnostic features of myeloma on MRI include a diffuse pattern of bone marrow involvement and numerous focal lesions throughout the skeleton. These lesions typically present with a 'salt-and-pepper' appearance due to varying signal intensities caused by active myeloma cells and fatty marrow replacement.

Bone scan is not the appropriate choice. Although bone scans use a radioactive tracer to evaluate bone metabolism, they are limited in detecting small focal lesions and do not adequately assess bone marrow involvement in multiple myeloma. Furthermore, they may demonstrate nonspecific uptake in other conditions affecting bone remodelling and repair.

Chest x-ray, while capable of revealing osteolytic lesions, does not provide sufficient information to differentiate between multiple myeloma and metastatic disease; thus, it is not advocated for definitive diagnosis.

PET-CT, although an alternative diagnostic tool for patients with newly diagnosed nonsecretory or oligo secretory multiple myeloma or those presenting with the extramedullary disease, lacks sufficient evidence to support its routine use in all cases of newly diagnosed multiple myeloma. One limitation is the potential for high false positive and false negative findings rates.

Skeletal survey is less sensitive than CT, MRI, and PET/CT; therefore, it is considered suboptimal and is not advisable in this clinical scenario.

Question 44 of 191

An 18-year-old woman presents to the emergency department with dark urine. She had recently been diagnosed with 'mono' following a sore throat confirmed by swab testing, originally becoming unwell nine days ago. She had been recovering and was starting to feel well but then noticed passing dark urine after having a walk outside in the park but she started to return after it started to rain heavily and she became very cold. She also had abdominal cramping sensation. She has no other past medical history, takes no medication apart from the oral contraceptive pill, and has no allergies.

Blood tests:

Hb	101 g/l
Platelets	379 * 10 /l9
WBC	5.1 * 10 /l9
Bilirubin	43 µmol/l
ALP	161 u/l
ALT	15 u/l
Blood film	spherocytes

What is the most appropriate investigation to establish a diagnosis?

A. Hepatitis C serology
B. Syphilis serology
C. Anti-nuclear antibody
D. Direct Coombs test
E. ESO titre

D. Direct Coombs test

Paroxysmal cold haemoglobinuria should be confirmed with urinary heamosiderin

This is a young patient who has developed dark urine associated with exposure to going outside, implying exposure to cold. This has been associated with abdominal cramps and following a recent viral infection, most likely with Ebstein-Barr virus. In addition, blood tests show anaemia, elevated bilirubin and spherocytes. This implies haemolysis, and the history suggests that the haemolysis is associated after cold exposure. This history suggests is typical for paroxysmal cold haemoglobinuria. Paroxysmal cold haemoglobinuria is due to cold-reacting IgG causing complement fixation leading to haemolysis. Diagnosis is best established with direct Coombs test to demonstrate the intravascular nature of haemolysis. Further testing can demonstrate the presence of temperature specific IgG antibodies which react to red blood cells below 37 degrees C and cause hemolysis on re-warming. Syphilis was historically associated with this condition, but only in tertiary syphilis which such a young patient is unlikely to have. Determining ESO titre to establish the viral infection is unlikely to be helpful in the current situation, and whilst hepatitis C is associated with cryoglobulinaemia it is not associated with paroxysmal cold haemoglobinuria. In older patients, paroxysmal cold haemoglobinuria can be associated with auto-immune conditions, making ANA a useful screening test, but there is no chronic history suggesting a prolonged autoimmune condition.

Question 45 of 191

A 74-year-old man attends with worsening fatigue and shortness of breath. A systematic enquiry was otherwise unremarkable. He was treated with radical chemo-radiotherapy for metastatic squamous cell carcinoma 5 years ago.

Blood results are as follows:

Hb	72 g/L	Male: (135-180)Female: (115 - 160)
Platelets	42 * 10 /L 9	(150 - 400)
WBC	2.1 * 10 /L 9	(4.0 - 11.0)
Neuts	0.4 * 10 /L 9	(2.0 - 7.0)
Lymphs	0.6 * 10 /L 9	(1.0 - 3.5)
Mono	1.0 * 10 /L 9	(0.2 - 0.8)
Eosin	0.1 * 10 /L 9	(0.0 - 0.4)
Blood film	Occasional hypogranular and hypolobulated neutrophil; Blasts 8% of all nucleated cells	

What is the most likely diagnosis?

A. Acute lymphoblastic leukaemia
B. Acute myeloid leukaemia
C. Metastatic infiltration
D. Myelodysplastic syndrome
E. Myelofibrosis

D. Myelodysplastic syndrome

Pancytopaenia 5 years post-chemotherapy/radiotherapy → ?myelodysplastic syndrome

Myelodysplastic syndrome is correct. The patient most likely has therapy-related myelodysplastic syndromes (t-MDS) which is defined as MDS occurring as a complication of cytotoxic chemotherapy and/or radiation. The median onset of occurrence is 5 years post-chemotherapy (however this can vary widely with the type of chemotherapy given). The blood film appearances of hypogranular and hypolobulated neutrophils are classic of MDS. The presence of myeloblasts is also in keeping with this condition, and since the blast count is < 20% on peripheral blood the diagnosis is MDS rather than acute myeloid leukaemia. The patient requires an urgent bone marrow aspirate and trephine for morphology, cell markers, and cytogenetics.

Acute lymphoblastic leukaemia is incorrect. Acute lymphoblastic leukaemia (ALL) is rare in adults. It is predominantly a disease of children. The blood film appearances are not in keeping with ALL which would present with lymphoblasts and the absence of dysplasia in the myeloid lineage.

Acute myeloid leukaemia is incorrect. The peripheral blast count is < 20% confirming a diagnosis of MDS. Acute myeloid leukaemia (AML) is characterised by a peripheral blast count > 20%. It is important to note that the main risk of MDS is transformation to AML.

Metastatic infiltration is incorrect. Metastatic infiltration would be characterised by leukoerythoblastosis and teardrop poikilocytes on the blood film.

Myelofibrosis is incorrect. Myelofibrosis would also be characterised by leukoerythoblastosis and teardrop poikilocytes on the blood film.

Question 46 of 191

A 45-year-old gentleman has a diagnosis of renal cell carcinoma and is booked for a nephrectomy in four days time. He presents with an acutely swollen, tender left calf and a diagnosis of deep vein thrombosis is made, with the ultrasound report describing a clot extending to the femoral vein.

Which management is most appropriate?

A. Low molecular weight heparin and IVC filter insertion
B. Low molecular weight heparin
C. Warfarin
D. Apixiban
E. None

A. Low molecular weight heparin and IVC filter insertion

If a diagnosis of DVT is made less than 7 days before surgery, an IVC filter must be inserted pre-op

This gentleman has a malignancy that needs urgent operative management. An IVC filter should be inserted pre-operatively to bridge the operative period when low molecular weight heparin will be held.

The British Society of Haematology recommends that 'IVC filters should be considered in any preoperative patient with recent VTE (within 1 month) in whom anticoagulation must be interrupted'.

Whilst DOACs such as apixaban are now recommended first-line for venous thromboembolism they would be less suitable as surgery is planned in 4 days time.

Question 47 of 191

A 34-year-old woman presents to the emergency department with fever and bruising. She has no past medical history and does not take any regular medications. She does not smoke or drink alcohol and works as an accountant.

Observations:

- Heart rate 94 beats per minute
- Temperature 38.1ºC
- Blood pressure 170/101 mmHg
- Respiratory rate 18/minute
- Oxygen saturations 96% on room air

On examination, there is no meningism. You note that she is jaundiced but there are no signs of chronic liver disease. There are scattered petechiae on her arms, legs and abdomen.

Bloods tests:

Hb	72 g/L	Male: (135-180)Female: (115 - 160)
MCV	104fL	(80-96)
Platelets	33 * 10 /L	(150 - 400)
WBC	4.6 * 10 /L	(4.0 - 11.0)
Na	136 mmol/L	(135 - 145)
K	4.2 mmol/L	(3.5 - 5.0)
Urea	9.2 mmol/L	(2.0 - 7.0)
Creatinine	155 µmol/L	(55 - 120)
CRP	4 mg/L	(< 5)
Bilirubin	53 µmol/L	(3 - 17)
ALP	88 u/L	(30 - 100)
ALT	24 u/L	(3 - 40)
Î³GT	44 u/L	(8 - 60)
Albumin	36 g/L	(35 - 50)
Prothrombin time	11 seconds	(10-14)
Blood film		schistocytes
ADAMTS13 enzyme		absent

Urinalysis:

Protein	++
Blood	++
Leucocytes	+
Nitrites	-ve
Glucose	-ve

Based on the likely diagnosis, what treatment is most likely to be effective?

A. Antibiotics
B. Antiplatelet therapy
C. High dose intravenous steroids
D. Intravenous labetalol
E. Plasma exchange

E. Plasma exchange

TTP - plasma exchange is first-line

Plasma exchange is correct. The patient has thrombotic thrombocytopenic purpura as evidenced by fever, thrombocytopenia, renal failure, microangiopathic haemolytic anaemia (jaundice, schistocytes) and absent ADAMTS13 enzyme activity. Haematoproteinuria is also common in this condition. The first-line treatment for this condition is plasma exchange (PEX), which removes inhibitory antibodies and replenishes the deficient protease.

Antiplatelet therapy is incorrect. While this is a thrombotic condition, there is a risk of haemorrhage with antiplatelet treatment.

High dose intravenous steroids is incorrect. Steroids are sometimes administered in this condition but have no proven benefit over PEX alone.

Intravenous labetalol is incorrect. While the patient is hypertensive, the blood pressure is not so high that we would label this as malignant hypertension, which is a differential for a cause of microangiopathic haemolytic anaemia.

Antibiotics are not the right answer. While no one would criticise someone for administering antibiotics to someone with a petechial rash and fever, on the basis that it could be meningococcal septicaemia, antibiotics have no role in the management of TTP and may even worsen the condition.

Question 48 of 191

A 24-year-old female presents to the emergency department with severe abdominal pain which had developed over for the last few hours. The pain was central, severe and stabbing in nature associated with vomiting. She was in hysterics and was extremely agitated and confused. Past medical history included asthma and depression. Two days earlier she had seen her GP for dysuria and been prescribed trimethoprim.

She was a student studying chemistry at university and had recently been out late several nights drinking excess alcohol to celebrate passing her exams. On examination, she was unwell, extremely clammy, distressed with generalised abdominal tenderness and weakness in both legs with areflexia. Heart sounds and chest were clear. Observations showed a blood pressure 190/100 mmHg, heart rate 126/min, regular and temperature 37.9ºC.

Which investigation is most likely to be diagnostic?

A. Urinary catecholamines
B. Abdominal ultrasound
C. Urinary porphobilinogen
D. Lumbar puncture
E. Blood cultures

C. Urinary porphobilinogen

In acute intermittent porphyria, urinary porphobilinogen is typically raised

This patient has acute intermittent porphyria (AIP). AIP is the most common type of porphyria. AIP is an autosomal dominant condition caused by a genetic mutation in the gene encoding the enzyme porphobilinogen (PBG) deaminase on chromosome 11. The condition is more common in females than males and usually occurs between the ages 14-30 years. Porphyrias occur due to a problem within the haem biosynthesis. Defective PBG deaminase causes PBG to accumulate and this can be found in the urine. Attacks are usually precipitated by drugs, alcohol, fasting and sepsis. This patients attack has been caused by a combination of antibiotic use and alcohol intake.

Patients most commonly present with gastrointestinal symptoms (most commonly severe abdominal pain) neurological symptoms (autonomic dysfunction, peripheral motor neuropathies, areflexia, delirium, seizures, coma) and psychiatric symptoms such as common. Observations commonly show tachycardia and hypertension. Fever can be present. Most patients are completely free of symptoms between attacks.

The treatment for acute attacks of porphyria is to decrease haem synthesis and reduce the production of porphyrin precursors. Withdrawal of culprit medication is essential. A high-calorie intake and high doses of glucose can help inhibit haem synthesis and are useful for the treatment of mild attacks. In severe attacks, intravenous haematin is used. Pain is treated with narcotics. Beta blockers can be used to treat tachycardia and hypertension.

Advice can be obtained from National Acute Porphyria Centres: Cardiff, Kings College Hospital, London and Cambridge.

Question 49 of 191

A 22-year-old man attends with progressive tiredness over the past week. A systematic enquiry was otherwise unremarkable. He has a past medical history of hereditary spherocytosis. His regular medications include folic acid however he states that he often forgets to take this. On examination, you note 5cm splenomegaly.

Blood results are as follows:

Hb	55 g/L	Male: (135-180)Female: (115 - 160)
Platelets	142 * 10^9/L	(150 - 400)
WBC	14.2 * 10^9/L	(4.0 - 11.0)
Reticulocytes	0.2 %	(0.5 - 1.5)
Direct antiglobulin test (DAT)	Negative	
Bone marrow examination	Marked erythroid hypoplasia and occasional giant erythroblasts	

Blood results from the clinic 3 months ago are as follows:

Hb	105 g/L	Male: (135-180)Female: (115 - 160)
Reticulocytes	5.8 %	(0.5 - 1.5)

What is the most likely explanation?

A. Acute erythroblastic leukaemia
B. Folic acid deficiency
C. Haemolytic crisis
D. Parvovirus infection
E. Splenic sequestration crisis

D. Parvovirus infection

Parvovirus infection may trigger an aplastic crisis in patients with hereditary spherocytosis

Parvovirus infection is correct. Of note, the patient has significant reticulocytopaenia suggestive of bone marrow failure. Two of the commonest causes of this would include infection (e.g. Parvovirus) and megaloblastic anaemia (e.g. B12 and folate deficiency). In health, the usual life span of a red cell is approximately 120 days. However in conditions such as hereditary spherocytosis (HS), due to premature destruction by the spleen, the lifespan could be as low as 10- 30 days. Thus acute insults to the bone marrow can manifest much more rapidly in the peripheral blood. Acute parvovirus B19 infection can cause a transient erythroid hypoplasia as demonstrated on the bone marrow results in this case. In healthy populations this would unlikely cause more than mild anaemia, however, in patients with chronic haemolytic states (e.g. red cell membrane disorders and haemoglobinopathies), the effects can be much more rapid and profound as demonstrated in this case.

Acute erythroblastic leukaemia is incorrect. The giant erythroblasts within the bone marrow are indicative of acute parvovirus infection, not leukaemia.

Folic acid deficiency is incorrect. Patients with HS have chronic low-grade haemolysis and therefore an increased red cell turnover. Since folic acid is essential for haematopoiesis, all patients with HS should continue on lifelong folic acid replacement. This is even more essential in a haemolytic crisis. Importantly this remains the case even when folic acid levels are normal. Although the patient, in this case, has poor compliance with folic acid, the bone marrow examination is more suggestive of acute parvovirus infection rather than megaloblastic anaemia.

Haemolytic crisis is incorrect. The presence of a marked reticulocytosis would be suggestive of a haemolytic crisis. However, it would be important to compare this to the patient's baseline reticulocyte count since patients with hereditary spherocytosis will often have chronic reticulocytosis due to low-grade haemolysis of spherocytes. In this case, the patient's reticulocyte count is low, making a haemolytic crisis less likely.

Splenic sequestration crisis is incorrect. Although the patient has mild splenomegaly, this would be expected due to the chronic haemolytic state associated with hereditary spherocytosis. A splenic sequestration crisis would present with a marked and painful enlargement of the spleen.

Question 50 of 191

A 35-year-old male Caucasian presents to the emergency department. He was asked to come to hospital after his GP noticed that his recent community blood tests showed severe anaemia. He attended his GP two days ago due to feeling increasingly tired and therefore the GP did routine blood tests for him. Apart from increasing fatigue and shortness of breath on exertion he had noticed nothing wrong. He had no past medical history apart from appendectomy when he was 20 years old and has recently significantly increased his exercise in an attempt to loose weight, and developed severe muscle aches, for which he has started to take diclofenac regularly. He denies any pain currently. On examination he is mildly jaundiced, has no stigmata of liver disease and his abdomen is soft without tenderness.

Blood tests:

Hb	96 g/l
Platelets	357 * 10 /l$_9$
WBC	4.8 * 10 /l$_9$
Blood film	spherocytosis
Na $^+$	138 mmol/l
K $^+$	4.9 mmol/l
Urea	4.7 mmol/l
Creatinine	79 µmol/l
Bilirubin	58 µmol/l
ALP	188 u/l
ALT	26 u/l

Further investigations are pending.

What is the most likely diagnosis?

A. G6PD deficiency
B. Warm autoimmune haemolytic anaemia
C. Peptic ulcer disease
D. Sickle-cell anaemia
E. Thalassaemia

B. Warm autoimmune haemolytic anaemia

Warm autoimmune haemolytic anaemia can be confirmed with the presence of anaemia, spherocytes on blood film and positive direct Coombs test for IgG or C3d

This is a patient who has developed symptomatic anaemia and jaundice following taking diclofenac, and has no symptoms of gastric irritation or signs of epigastric tenderness. Furthermore, the presence of elevated bilirubin suggests a haemolytic cause for the anaemia. These factors make peptic ulcer disease less likely.

Warm autoimmune haemolytic anaemia is the most likely explanation due to the evidence of haemolysis occurring following the administration of a drug known to cause this problem, and the presence of spherocytes supports this as well, but confirmation would be with a positive direct Coombs test.

G6PD deficiency is a cause for acute haemolytic anaemia but is more common in those of African ancestry and occurs typically after oxidating drugs such as primaquine.

Sickle-cell anaemia is very unlikely in a Caucasian patient and is also unlikely to be diagnosed at this age, and also would likely have a history of sickle-cell crisis.

Thalassemia is a disorder of globin genes causing ineffective erythropoiesis and haemolysis, endemic in the Mediterranean region and Africa. The blood film would be expected to show microcytosis and target cells rather than spherocytosis, and there is no evidence of extramedullary haematopoiesis.

Question 51 of 191

A 65-year-old man attends the clinic with a three-month history of generalised, dull-aching bone pain and increased fatiguability. He has a history of hypertension, which is well-controlled with amlodipine 10 mg daily. On examination, he exhibits pallor and has bruises and petechiae on his shins and forearms. Laboratory results are attached.

Hb	7.6 g/L	Male: (135-180)
Platelets	39 * 10 /L $_9$	(150 - 400)
WBC	3.1 * 10 /L $_9$	(4.0 - 11.0)
Calcium	3.1 mmol/L	(2.1-2.6)
Creatinine	140 µmol/L	(55 - 120)

What is the most appropriate next investigation to determine the cause of this patient's bone pain?

A. MRI spine
B. Whole body MRI
C. Whole body isotope bone scan
D. Whole body low dose CT scan
E. X-ray skull and spine

B. Whole body MRI

Whole body MRI 1st line imaging in suspected multiple myeloma

Whole body MRI is the preferred initial imaging modality for detecting myeloma-related bone disease and extra-medullary plasmacytomas. The clinical features of bone pain, fatigue, anaemia, thrombocytopenia, deranged creatinine, and hypercalcaemia in an older man are suggestive of multiple myeloma. It is standard practice to offer imaging to all patients with a suspected diagnosis of myeloma to investigate potential bone involvement, and a whole-body MRI is the most suitable investigation. This imaging modality can detect both lytic lesions and bone marrow infiltration, which are characteristic of multiple myeloma.

Whole body low dose CT scan is recommended as the first-line alternative for imaging in patients with myeloma in instances where an MRI is contraindicated due to the presence of a pacemaker, metallic heart valves, or aneurysm clips. For comprehensive assessment in this context, a whole-body MRI would be necessary rather than just an **MRI spine**.

A **whole body isotope bone scan** is not indicated for initial screening of myeloma-related bone disease. An isotope bone scan relies on osteoblastic activity and may yield a false negative result, particularly in cases where bone turnover is not high.

An **x-ray skull** is not a primary imaging choice for evaluating myeloma-associated bone pathology. While a raindrop skull on an x-ray can support the diagnosis of multiple myeloma, it is not sensitive or specific enough to be used as a primary diagnostic tool.

Question 52 of 191

A 20-year-old female complains of feeling tired. She has no past medical history and takes no regular medications.

The following blood tests were obtained:

Hb	84g/L
MCV	70fL
WBC	8x10^9/L
Platelets	460x10^9/L

Blood film analysis comments on hypochromic anaemia with pencil cells, target cells, acanthocytes and Howell Jolly bodies.

What is the most likely diagnosis?

A. Sickle cell anaemia
B. Hereditary spherocytosis
C. Thalassaemia trait
D. Iron deficiency anaemia
E. Coeliac disease

E. Coeliac disease

The blood film shows hypochromic anaemia with the presence of pencil cells that indicates the patient has iron deficiency anaemia. Target cells, acanthocytes and Howell Jolly bodies are features of hyposplenism. Iron deficiency and functional hyposplenism are both seen in coeliac disease, which is therefore the most likely unifying diagnosis. Thalassaemia trait is associated with mild reduction in haemoglobin, low MCV and low MCH; there would not be evidence of hyposplenism on the film in thalassaemia trait. Hereditary spherocytosis is due to a deficiency in spectrin, which leads to an abnormal red blood cell cytoskeleton. The film would show spherocytic red blood cells and reticulocytes. In sickle cell anaemia MCV is usually normal and sickle cells would be seen on the blood film.

Question 53 of 191

A 34-year-old man presents to the emergency department with abdominal pain. He has a background of multiple previous attendances with similar symptoms, which typically last for 3-7 days and then resolve. On a previous occasion, he underwent an exploratory laparoscopy, which did not identify a cause. He has a past medical history of depression. He takes citalopram. He does not smoke cigarettes or drink alcohol. He is a ceramicist. He has been under a lot of stress at work recently.

His observations are heart rate 111 beats per minute, blood pressure 153/74 mmHg, respiratory rate 18/minute, oxygen saturation 96% on room air and temperature 37ºC.

Examination reveals a soft and non-tender abdomen with normal bowel sounds. There are no abnormalities of the external genitalia. A bilateral foot drop is noted on neurological examination.

Blood tests:

Hb	136 g/L	Male: (135-180)Female: (115 - 160)
Platelets	189 * 10 /L 9	(150 - 400)
WBC	5.6 * 10 /L 9	(4.0 - 11.0)
Na +	129 mmol/L	(135 - 145)
K +	4.2 mmol/L	(3.5 - 5.0)
Urea	8.2 mmol/L	(2.0 - 7.0)
Creatinine	89 µmol/L	(55 - 120)
CRP	23 mg/L	(< 5)
Lactate	1.2 mmol/L	(0-2)

A urine sample is requested and it is noted to turn dark red when left standing. Porphobilinogen is increased in the urine.

Plain radiography of the abdomen and chest are normal.

Based on the likely diagnosis, what is the most appropriate treatment?

A. Chelation therapy
B. IV hydrocortisone
C. Supportive treatment only
D. 5% dextrose
E. Haem arginate and 10% dextrose

E. Haem arginate and 10% dextrose

IV haem arginate can be used to treat flares of acute intermittent porphyria

Haem arginate and 10% dextrose is the correct answer. This presents with features suggestive of acute intermittent porphyria (unexplained abdominal pain, depression, neuropathy, hyponatraemia, hypertension, tachycardia, red urine) and increased levels of urine porphobilinogen. Intravenous glucose inhibits the activity of aminolaevulinic acid synthase (an enzyme in the haem pathway). This reduces the overproduction of porphyrins. More hypotonic dextrose infusions should be avoided as they may worsen hyponatraemia. Haem arginate itself inhibits 5-aminolevulinic acid, which is another component in the porphyrin synthesis pathway. While the randomized control evidence of is lacking for this treatment, it is felt that patients typically respond well to this treatment and it should be given early in a severe attack.

5% dextrose is incorrect. Hypotonic dextrose solutions should be avoided as they may worsen hyponatraemia.

Chelation therapy is incorrect. Lead poisoning is a mimic of acute intermittent porphyria and can cause unexplained abdominal pain and neuropathy. However, the key distinction is that urine levels of porphobilinogen will not be increased in this condition. The diagnosis is typically confirmed by measuring levels of lead in the blood.

IV hydrocortisone is incorrect. Addison's disease can cause abdominal pain and hyponatraemia but you would expect hypotension rather than hypertension and the urinary findings are not typical of this condition.

Supportive treatment only is incorrect. This patient has a significant attack of acute intermittent porphyria with evidence of raised urinary porphobilinogen and motor neuropathy. This indicates a need for treatment with the above regimen.

Question 54 of 191

A 70-year-old man is referred to the haematology clinic by his general practitioner with anaemia. He has experienced progressive fatigue and shortness of breath for four months. On further questioning he also describes waking up at night soaked in sweat on one or two nights per week for the last month. His weight is stable. He has a past medical history of hypertension and COPD.

On examination he is pale. His heart sounds are normal and his chest is clear.. He has no ankle oedema and JVP is not raised. His abdomen is soft and he has splenomegaly 3cm below the costal margin with no hepatomegaly.

Test results sent with him by his GP are a follows:

Hb	92 g/l	Na +	143 mmol/l
Platelets	143 * 10 /l 9	K +	3.7 mmol/l
WBC	4 * 10 /l 9	Urea	7 mmol/l
Neuts	2 * 10 /l 9	Creatinine	86 µmol/l
Lymphs	1 * 10 /l 9	CRP	5 mg/l

Blood film: Anisocytosis with mild hypochromia. Tear drop cells. Mild thrombocytopenia with no platelet clumping.

Chest x-ray: Mildly hyperexpanded lung fields. No focal consolidation. No masses or lymphadenopathy.

Upper GI endoscopy & colonoscopy: Normal

Presence of which mutation is required to confirm the likely diagnosis?

A. BCR-ABL
B. BCL2
C. C-MYC
D. JAK2
E. TP53

D. JAK2

This gentleman has B symptoms, anaemia and splenomegaly in the presence of characteristic tear drops cells on the blood film, making myelofibrosis the most likely diagnosis. The British Committee for Standards in Haematology list several mutations which together with these symptoms and fibrosis on bone marrow form the diagnostic criteria. The most common of these is JAK2.

Presence of BCR-ABL is diagnostic for CML. BCL2 and TP53 mutations are seen in diffuse large B cell lymphoma.

C-MYC mutations are seen in Burkitt's lymphoma.

Use of JAK inhibitors in the management of myelofibrosis: a revision of the British Committee for Standards in Haematology Guidelines for Investigation and Management of Myelofibrosis 2012.

British Journal of Haematology, 2014;:167;418438.

Question 55 of 191

A 48-year-old male with hepatitis C is admitted to the acute medical assessment unit having noticed a significant reduction in his urine output over the last few days. Urinalysis revealed haematoproteinuria, and his blood results are as follows:

Hb	9.8 g/dl
Platelets	75 * 10 /l9
WBC	12.1 * 10 /l9
Na +	143 mmol/l
K +	5.4 mmol/l
Urea	18.9 mmol/l
Creatinine	205 µmol/l

A renal biopsy was subsequently performed and microscopy of the sample showed generally enlarged and hypercellular glomeruli with an increase in mesangial cellularity and matrix. A histological diagnosis of membranoproliferative glomerulonephritis is made.

What is the most likely underlying pathology for this finding?

A. Cryoglobulinaemia
B. Haemolytic uraemic syndrome
C. Hepato-renal syndrome
D. Systemic lupus erythematosus
E. Thrombotic thrombocytopaenic purpura

A. Cryoglobulinaemia

Hepatitis C is associated with mixed (type II) cryoglobulinaemia

This gentleman presents with acute kidney injury secondary to membranoproliferative glomerulonephritis. All of the listed options are recognised causes of membranoproliferative glomerulonephritis, but the key to this question is the patient's history of hepatitis C which is known to be closely associated with cryoglobulinaemia.

Question 56 of 191

A 77-year-old man presents with a general decline in mobility, extreme lethargy, and weight loss. His admission blood tests show:

Hb	98 g/L	Male: (135-180)Female: (115 - 160)
Platelets	119 * 10 /L 9	(150 - 400)
WBC	3.8 * 10 /L 9	(4.0 - 11.0)

Na +	137 mmol/L	(135 - 145)
K +	4.4 mmol/L	(3.5 - 5.0)
Urea	6.1 mmol/L	(2.0 - 7.0)
Creatinine	73 µmol/L	(55 - 120)
Calcium	2.46 mmol/L	(2.1-2.6)

A CT TAP is performed:

No evidence of solid organ malignancy or bony lesions. Splenomegaly noted - clinical correlation advised.

Given the ongoing concern for malignancy, a myeloma screen is sent:

IgA	0.56 g/L	(0.64-2.97)
IgG	4.7 g/L	(5.8-15.4)
IgM	12.52 g/L	(0.71-2.3)
Serum electrophoresis	Shows a paraprotein band	
Paraprotein level	44g/L	
Serum immunofixation	IgM type kappa paraprotein	
Serum free kappa light chains	13.65mg/L	(3.30-19.40)
Serum free lambda light chains	18.09 mg/L	(5.71-26.30)
Serum kappa / lambda ratio	0.75	(0.26-1.65)

What is the most likely diagnosis?

A. Acute lymphoblastic leukaemia
B. Monoclonal gammopathy of uncertain significance
C. Multiple myeloma
D. Smouldering myeloma
E. Waldenstrom's macroglobulinaemia

A. Waldenstrom's macroglobulinaemia

IgM paraproteinaemia - ?Waldenstrom's macroglobulinaemia

Waldenstrom's macroglobulinaemia is the correct diagnosis. The patient presents with relatively non-specific symptoms; however, initial findings of pancytopenia and radiological evidence of splenomegaly are concerning for a haematological malignancy. Subsequent serological investigations revealing elevated serum free light chains and electrophoresis indicating a raised IgM level, alongside an IgM-type kappa paraprotein, strongly suggest Waldenstrom's macroglobulinaemia.

Acute lymphoblastic leukaemia (ALL) is not the appropriate diagnosis. ALL is characterised by the malignant proliferation of lymphoid progenitor cells and typically presents with lymphadenopathy and signs consistent with bone marrow infiltration by malignant clones, such as anaemia and thrombocytopenia. While initial blood tests may show leukocytosis, anaemia, and thrombocytopenia, elevated IgM levels (12.52 g/L) and an associated IgM-type kappa paraprotein indicate a monoclonal gammopathy rather than ALL. Lymphoid progenitor cells in ALL do not secrete immunoglobulins in large quantities; thus, the detection of a paraprotein band, specifically an IgM-type kappa paraprotein on serum electrophoresis, does not align with ALL but instead suggests plasma cell disorders such as Waldenstrom's macroglobulinaemia or multiple myeloma.

Monoclonal gammopathy of uncertain significance (MGUS) is also incorrect. Although MGUS with an IgM paraprotein could yield similar serum electrophoresis and immunofixation results to those observed in this case, MGUS is asymptomatic by definition—contrary to our patient who exhibits symptomatic anaemia. Furthermore, diagnostic criteria for MGUS stipulate that the paraprotein concentration must be below 30g/L; this patient's level exceeds this threshold at 44g/L. The combination of symptomatic presentation and a higher paraprotein level excludes MGUS as a diagnosis.

Multiple myeloma does not fit this clinical picture either. Defined by CRAB features (hypercalcaemia, renal failure, anaemia, bony lesions), multiple myeloma involves monoclonal plasma cell proliferation detectable via serum electrophoresis showing a paraprotein presence. However, an IgM-type kappa paraprotein is more indicative of Waldenstrom's macroglobulinaemia than multiple myeloma; the latter more commonly involves IgG or IgA production. Additionally, splenomegaly is less common in multiple myeloma cases. The absence of skeletal lesions further argues against multiple myeloma as a diagnosis here. While multiple myeloma can present with variable levels of serum free light chains depending on disease burden and subtype—a finding not prominent in this patient—the normal range kappa/lambda ratio observed supports Waldenstrom's macroglobulinaemia over multiple myeloma.

Smouldering myeloma, like smouldering forms of other malignancies, presents without symptoms or end-organ damage—which includes CRAB features or any indication of organ impairment—and typically involves either IgG or IgA rather than IgM paraproteins. Given that our patient has symptomatic anaemia, smouldering myeloma is ruled out as a diagnosis in favour of Waldenstrom's macroglobulinaemia which accounts for both the symptomatology and laboratory findings observed.

Question 57 of 191

A 35-year-old patient with known sickle cell disease presents to the emergency department with new onset of left arm and facial weakness. The symptoms began earlier in the day. He is normally very careful with his sickle disease and ensures he is well hydrated and avoids the cold. His wife admits that over the last few days he has been suffering from nausea and vomiting and diarrhoea after he had a takeaway meal 3 days ago, On examination he his observations are within normal parameters. He has slurred speech and an obvious left facial droop with forehead sparing. He has power of 0/5 in his left arm but is otherwise neurologically intact.

His blood test show:

Hb	100 g/l
Platelets	330 * 10 /l9
WBC	8.9 * 10 /l9
INR	1.0
Na +	138 mmol/l
K +	3.5 mmol/l
Urea	9.9 mmol/l
Creatinine	135 µmol/l
CRP	19 mg/L(<10)
Bilirubin	12 µmol/l
ALP	89 u/l
ALT	39 u/l
Albumin	39 g/l

He is seen by the stroke team who arrange an urgent CT head which is reported as normal.

What is the appropriate treatment for this gentleman?

A. Thrombolysis
B. Aspirin
C. Plasmapheresis
D. Methylprednisolone
E. Exchange transfusion

A. Exchange transfusion

Exchange transfusions are a way of reducing the number of sickle red cells and increasing the number of normal red cells, in order to improve oxygenation

This gentleman has a cerebral vaso-occlusive episode as a result of his sickle cell disease. The treatment of choice in these situations is urgent and aggressive exchange transfusion followed by transfusions to maintain HbS <30%. Thrombolysis and aspirin are not in the management guidelines for acute stroke in this situation.

Question 58 of 191

A 29-year-old man who is known to be HIV positive is reviewed. He has been taking anti-retroviral therapy for the past 2 years and has remained relatively well. Over the past few weeks however he has developed abdominal distension with some discomfort in the right iliac fossa. On examination a mass can be felt in the right lower quadrant. A biopsy shows a B cell lymphoma. Sheets of a medium sized lymphoid cells with high proliferative activity, forming a 'starry sky' appearance, are noted.

What cytogenic abnormality is most likely to be found?

A. t(11;14)
B. t(14;18)
C. t(8;14)
D. t(9;22)
E. t(11;18)

C. t(8;14)

Burkitt's lymphoma - t(8:14)

This patient has an immunodeficiency-associated Burkitt lymphoma

Question 59 of 191

A 27-year-old woman is admitted after developing dyspnoea associated with pleuritic chest pain. A D-dimer taken on admission is elevated and subsequent CTPA shows a pulmonary embolism. Her past medical history includes a giving birth to her son 12 months ago (full term, vaginal delivery) and anxiety. She reports that her 47-year-old mother has had two deep vein thromboses in the past 10 years.

Which one of the following is the most likely underlying cause?

A. Factor V Leiden
B. Antithrombin III deficiency
C. Antiphospholipid syndrome
D. Protein C deficiency
E. Prothrombin gene mutation

A. Factor V Leiden

Activated protein C resistance (Factor V Leiden) is the most common inherited thrombophilia

NICE would recommend testing for thrombophilia given the unprovoked venous thromboembolism and family history

Question 60 of 191

A 19-year-old man with hereditary spherocytosis is reviewed in the clinic. He was last admitted to the hospital some 6 months earlier for an acute haemolytic crisis related to parvovirus infection. He complains that he feels chronically tired and has persistent left upper quadrant pain and discomfort. Abdominal examination confirms splenomegaly.

Investigations:

Abdominal ultrasound scan reveals splenomegaly and a solitary gallstone visualised within the gallbladder.

Hb	90 g/l
MCV	101 fL
Platelets	301 * 10 /l9
WBC	7.1 * 10 /l9

Which of the following is the most appropriate intervention for him?

A. Prednisolone
B. Splenectomy
C. Cholecystectomy
D. Oral iron supplementation
E. IV iron supplementation

B. Splenectomy

Elective splenectomy is appropriate in a patient with hereditary spherocytosis who has recurrent anaemia

This patient has hereditary spherocytosis with evidence of chronic haemolysis. Although this patient doesn't yet have symptoms of cholecystitis they have evidence of possible pigment gallstone formation on the ultrasound scan and symptomatic splenomegaly. Coupled with the anaemia and raised MCV (suggestive of increased reticulocytes), this clinical picture points to elective splenomegaly as the most appropriate option, with vaccination at least against pneumococcus before the procedure is carried out.

Cholecystectomy is carried out where patients present with cholecystitis related to hereditary spherocytosis. Prednisolone may help stimulate red cell production in situations where there is an acute haemolytic crisis. There is no role for iron supplementation here. IV iron supplementation is usually considered for patients with chronic renal impairment.

Question 61 of 191

A 60-year-old woman presents to the clinic with a one-year history of generalised body pain and fatigue. She intermittently experiences epistaxis and bleeding gums. She underwent a fundoscopy for blurred vision, which showed retinal haemorrhages and dilated retinal veins. Examination reveals pallor and hepatosplenomegaly. Serum protein electrophoresis demonstrates a sharp, narrow spike of monoclonal IgM. A skeletal survey is normal. A bone marrow biopsy is scheduled.

What are the expected findings of the bone marrow biopsy for this patient?

A. <10% infiltration of bone marrow with lymphoplasmacytic cells
B. <10% monoclonal plasma cells
C. >10% infiltration of bone marrow with lymphoplasmacytic cells
D. >10% monoclonal plasma cells with clock-face chromatin
E. Abnormal B cells

C. >10% infiltration of bone marrow with lymphoplasmacytic cells

IgM paraproteinaemia - ?Waldenstrom's macroglobulinaemia

> 10% infiltration of the bone marrow with lymphoplasmacytic cells is the typical bone marrow finding observed in patients with Waldenstrom's macroglobulinemia, which is the case here. The patient's presentation and investigations are consistent with Waldenstrom's macroglobulinaemia, a lymphoplasmacytic malignancy characterised by the secretion of a monoclonal IgM paraprotein. The disease typically presents with systemic symptoms such as fatigue and weight loss, hyperviscosity syndrome (e.g., blurred vision due to retinal haemorrhages), and hepatosplenomegaly.

< 10% infiltration of bone marrow with lymphoplasmacytic cells is typically observed in monoclonal gammopathy of undetermined significance (MGUS). The features such as anaemia, hepatosplenomegaly and hyperviscosity symptoms outlined in this scenario are rarely associated with MGUS; hence, this option is not applicable.

< 10% monoclonal plasma cells is incorrect because it is < 10% monoclonal plasma cells can be seen in MGUS. It is important to distinguish this condition from multiple myeloma. The lack of bony lesions and normal skeletal survey findings exclude multiple myeloma in this case. Additionally, IgM myeloma is exceedingly uncommon. **> 10% monoclonal plasma cells with clock-face chromatin** typify multiple myeloma; therefore this choice is incorrect.

Chronic lymphocytic leukaemia (CLL) represents another differential diagnosis characterised by **abnormal B cells** within the bone marrow. Monoclonal gammopathy is uncommon in CLL. Serum protein electrophoresis typically appears normal in CLL cases and only demonstrates abnormal protein spikes in approximately 15% of instances.

Question 62 of 191

A 28-year-old Afro-Caribbean male presents with a two-hour history of sudden onset left sided weakness. He denies any sensory involvement, dysarthria or dysphasia. He has a known history of sickle cell disease, with two previous episodes of transient ischaemic attacks and an episode of acute chest syndrome attack 10 days ago. On examination, he displays power of 1/5 in his left arm, 2/5 in his left leg, 5/5 in his right side. He reports no sensory disturbances, plantar responses are downgoing bilaterally, he is unable to perform finger-nose testing. He denies any illicit drug use, is a non-smoker and does not drink alcohol. He has no other past medical history. A hyperacute CT head demonstrates an area of acute ischaemia in the right internal capsule region.

What is the most appropriate immediate treatment?

A. Intravenous thrombolysis
B. Aspirin 300mg
C. Intravenous thrombolysis and mechanical thrombectomy
D. Exchange transfusion
E. Clopidogrel 300mg

D. Exchange transfusion

Exchange transfusions are a way of reducing the number of sickle red cells and increasing the number of normal red cells, in order to improve oxygenation

This patient has presented with an acute vaso-occlusive crisis on a background of known sickle cell disease. He is at high risk of an infarctive stroke given the background of previous TIAs (increased relative risk by 56 times) and a recent chest crisis within the past 2 weeks (increased relative risk by 7 times). The other risk factors include a high systolic pressure (RR 1.3 increase per 10mmHg) and

a low steady state haemoglobin (RR 1.9 per 1g/dL decrease)1. The management of an acute sickle infarct is different to that of thromboembolic stroke or atheromatous disease. The immediate aim is to reduce the proportion of HbS, either with immediate transfusion or optimally, with exchange transfusion. The latter reduces the concentration of HbS faster with a lower risk of transfusional volume overload, possibly resulting in increased blood viscosity and consequent pulmonary oedema.

Question 63 of 191

A 25 year old man presented to the Emergency Department with acute onset, rapidly progressive weakness in all the four limbs following a bout of severe colicky abdominal pain which lasted for 10 days but eventually subsided. The abdominal pain was located around the umbilicus, stabbing in nature, radiated towards the back, and was associated with nausea and intermittent constipation. Patient reports weakness originating in both arms then affecting both the lower limbs. He also complains of difficulty in closing his lips and eyes, and has uncontrolled salivation from the angles of the mouth. He denied any history of paraesthesia, sphincteric disturbances, epileptic fits or dark colored urine during any of the episodes. No definite history of any drug intake prior to both the episodes could be ascertained.

Physical examination revealed bilateral facial palsy of lower motor neuron (LMN) type together with flaccid quadriparesis which was more marked distally with bilateral wrist drop. Deep tendon reflexes were diminished in both upper and lower limbs. There was no sensory loss.

Initial lab values showed WBCs 8.9x10^9/L, Hemoglobin of 12g/L, Sodium of 132mmol/L, and ESR of 35mm/Hr. His temperature was 36.5 C and pulse 83/min. His blood pressure was 130/83 mm/Hg and oxygen saturations were 98% on air. A lumbar puncture was performed and CSF studies were normal.

Which of the following is most likely to be diagnostic?

A. CT scan with contrast of abdomen
B. MRI brain with contrast
C. Urine screen for Vanillylmandelic acid (VMA)
D. Urine screen for Aminolevulinic Acid (ALA) and Porphobilinogen (PBG)
E. Measurement of urinary and stool porphyrins

D. Urine screen for Aminolevulinic Acid (ALA) and Porphobilinogen (PBG)

n acute intermittent porphyria, urinary porphobilinogen is typically raised

Acute attacks of Acute Intermittent Porphyria (AIP) are often precipitated by drugs like barbiturates, sulfonamides, chloroquin, griseofulvin, diphenyl hydantoin and many other drugs, acute infections and over-indulgence in alcohol. Acute attacks are due to greatly increased activity of delta-aminolaevulinic acid synthetase. During acute attacks, the urine contains large amounts of porphobilinogen (PBG) and delta-aminolaevulinic acid (ALA) which, on standing, gives dark 'portwine' colour to the urine. Mental disturbances during acute episodes are of metabolic origin whilie neurological weakness of the extremities is due to focal demyelination and/or axonal degeneration of peripheral and autonomic nerves.

Onset of symptoms usually occurs in adolescence. Females are more commonly affected than males. The clinical picture is dominated by gastrointestinal and neurological manifestations. Symptoms during acute episodes usually comprise of severe colicky pain in the abdomen which may be diffuse or localized- usually to the umbilical or epigastric region and is often associated with nausea and vomiting and occasionally diarrhea. Diagnosis in such patients may be missed, unduly delayed or confused with acute surgical abdomen especially if neurological manifestations are lacking. Gastrointestinal manifestations are related to severe intestinal spasm due to autonomic dysfunction. Neurological manifestations comprise of flaccid paralysis, neuropsychiatric disturbances and rarely generalized epileptic fits. Paralysis may be confined to lower or upper extremities or may affect all the four limbs. It may be more marked proximally or distally or may be generalized and is due to predominant motor neuropathy. Sensory symptoms may also occur but objective sensory loss is unusual. Epileptic fits in AIP have been reported in about 15 to 20% cases. Bulbar paralysis and respiratory involvement can also occur and may threaten the life of the patient. Sphincteric disturbances are uncommon. Skin lesions are seldom seen.

Diagnosis of AIP should be suspected in any patient presenting with rapidly progressive flaccid paralysis with severe abdominal pain and history of passing dark reddish urine. Examination of the urine during acute attacks for PBG and ALA will help in establishing the diagnosis. In the patient in the above case, the diagnosis of AIP was based on clinical and urinary findings.

There is no specific treatment for porphyria and it is therefore very important to prevent the onset of acute attack. Treatment during acute episodes usually comprises symptomatic and general supportive measures for bulbar and respiratory paralysis, if it occurs. Chlorpromazine has been reported to be beneficial in relieving pain and other symptoms. Approximately 25% siblings of patients of AIP may be expected to have PBG in urine and therefore screening of siblings and other family members should be done. Prophylaxis in such cases as well as those known to clinically manifest AIP includes the avoidance of drugs known to precipitate the acute attacks as enlisted above.

Question 64 of 191

A 55-year-old man is referred by her GP to the haematology clinic. He reports a four-week history of night sweats and early satiety. On questioning, he elaborates and says that he has been feeling unwell for some time, but that he had just started work as a managing director and had attributed this to the added stress of his promotion.

On examination, he is pale with an unremarkable cardiovascular and respiratory exam. There is a fullness in the left upper quadrant which moves with inspiration and widespread small volume lymphadenopathy.

His blood tests are shown below:

Hb	100 g/L	Male: (135-180)Female: (115 - 160)
Platelets	117 * 10 /L 9	(150 - 400)
WBC	30.1 * 10 /L 9	(4.0 - 11.0)
Neuts	4.0 * 10 /L 9	(2.0 - 7.0)
Lymphs	25.0 * 10 /L 9	(1.0 - 3.5)
Mono	0.8 * 10 /L 9	(0.2 - 0.8)
Eosin	0.3 * 10 /L 9	(0.0 - 0.4)
Blood Film	lymphocytosis with smudge cells	

A bone marrow biopsy shows lymphocytosis in a diffuse growth pattern.

Chromosomal analysis is performed to guide further management.

Given the likely diagnosis,

which of the following findings would suggest a poor prognosis?

A. Wild-type TP53
B. >2% mutated immunoglobulin heavy chain variable region
C. Deletion of 13q
D. Mutated TP53
E. < 20% expression of CD38 lymphocytes

D. Mutated TP53

TP53 mutation is associated with a poor prognosis in CLL

This patient has aggressive chronic lymphocytic leukaemia (CLL) with both constitutional symptoms and bone marrow failure, indicating the need for treatment. The chromosomal analysis may be used to guide treatment offers significant insights into prognostication.

The presence of a mutated TP53 is a poor prognostic marker, where a 'wild type' refers to an unmutated form and is a good prognostic marker. A >2% mutated immunoglobulin heavy chain variable region, deletion of 13q or < 20% expression of CD38 lymphocytes are good prognostic markers.

Question 65 of 191

A 26-year-old gentleman presents to haematology clinic one week prior to starting chemotherapy for acute myeloid leukaemia (AML). He is currently feeling fatigued, suffering from night sweats, and has chronic lower back pain. He is anxious to start treatment.His past medical history includes ankylosing spondylitis and a clavicular fracture. His current medications include paracetamol and ibuprofen.

Blood tests:

Hb	113 g/l
Platelets	156 * 10 /l9
WBC	57 * 10 /l9
Na +	140 mmol/l
K +	3.6 mmol/l
Urea	4.2 mmol/l
Creatinine	63 µmol/l

What measure is the least useful to prevent tumour lysis syndrome?

A. IV fluids prior to chemotherapy
B. Urine alkalization
C. Prophylactic allopurinol
D. Prophylactic rasburicase
E. Stopping NSAID use

B. Urine alkalization

This patient has AML with a high white cell count and is about to start chemotherapy, and is therefore at high risk of tumour lysis syndrome (TLS).The key is to prevent TLS, which is done by ensuring good renal perfusion. Commonly this can be done by aggressive intravenous hydration prior to the start of treatment and stop NSAIDs, such as ibuprofen for this patient. Allopurinol and rasburicase both prevent uric acid accumulation and can, therefore, be used as well. Urine alkalization has not shown to be effective.

Question 66 of 191

A 50 year old woman was referred to haematology clinic for management of a persistent erythrocytosis that had been monitored by her General Practitioner over the previous 6 months. The patient was asymptomatic and in particular reported no headaches or visual changes. Past medical history was remarkable only for internal fixation of a tibial fracture sustained in a car accident 5 years previously. There is no strong family history of venous thrombosis or ischaemic heart disease. The patient took no regular medications and worked as an accountant. She is a lifelong non-smoker and drinks approximately 10 units of alcohol per week.

Following assessment at haematology, further investigations were requested as listed below.

Hb	# g/dl
Platelets	# * 10 /l
WBC	# * 10 /l
Haemoglobin	18.7 g / dL
White cell count	6.1* 10 /l
Neutrophils	3.5 * 10 /l
Lymphocytes	1.0 * 10 /l
Monocytes	0.7 * 10 /l
Eosinophils	0.4 * 10 /l
Basophils	0.5 * 10 /l
Platelets	276 * 10 /l
Packed cell volume	0.57
Urea	4.5 mmol / L
Creatinine	95 micromol / L
Sodium	142 mmol / L
Potassium	4.5 mmol / L
Ferritin	56 ng / mL
Albumin	35 g / L
Alkaline phosphatase	80 U / L
ALT	20 U / L
Bilirubin	18 micromol / L
JAK 2 V617F mutation	Postive
Serum erythropoietin	3 U / L (reference 0-19)

Blood film: no abnormality detected

Abdominal ultrasound: liver, hepatic duct system and gallbladder unremarkable; mild-moderate splenomegaly; kidneys and renal tract unremarkable

What is the appropriate management for the patient's erythrocytosis?

A. Aspirin and venesection with target PCV < 0.45
B. Aspirin and venesection with target PCV 0.45-0.50
C. Aspirin
D. Venesection with target PCV 0.45-0.50
E. Hydroxyurea

A. Aspirin and venesection with target PCV < 0.45

This patient has polycythemia vera as evidenced by the positive JAK 2 mutation, normal blood film, splenomegaly and low serum erythropoietin.

Management of polycythemia depends on the patient's risk of thrombosis. This patient has a low risk of thrombosis based on her age, absence of cardiac risk factors, normal white cell and platelet count and absence of hyperviscosity symptoms.

The mainstay of treatment of polycythaemia vera is aspirin and venesection. A target PCV < 0.45 has been shown to have a significantly lower rate of death from cardiovascular disease and major thrombosis than target PCV 0.45-0.50. Treatment with aspirin reduces the risk of non-fatal myocardial infarction, non-fatal stroke, pulmonary embolus, major venous thrombosis or death from cardiovascular cause compared to placebo. Aspirin does not increase the incidence of major bleeding episodes compared to placebo.

Cytoreductive treatments such as hydroxyurea are used to treat high-risk polycythaemia vera.

Question 67 of 191

A 65-year-old male presents with a 2 year history of fatigue. Over the past year he has had progressive early satiety, vomiting and weight loss. On examination you note 10cm splenomegaly.

Investigation results are as follows:

Hb	84g/l	Na +	138 mmol/l	Bilirubin	22 µmol/l
Platelets	65 * 10 /l 9	K +	3.7 mmol/l	ALP	88 u/l
WBC	2.2 * 10 /l 9	Urea	4.5 mmol/l	ALT	22 u/l
Neuts	0.8 * 10 /l 9	Creatinine	68 µmol/l	γGT	96 u/l
Lymphs	0.4 * 10 /l 9	Albumin	28 g/l		

Blood film: Tear-drop poikilocytes

What is the most likely diagnosis?

A. Myelodysplasia due to bone marrow metastases
B. Chronic myeloid leukaemia
C. Acute myeloid leukaemia
D. Myelofibrosis
E. Megaloblastic anaemia

D. Myelofibrosis

Massive splenomegaly may early satiety and vomiting which can mimic gastric cancer

The blood results confirm pancytopenia with tear drop cells, suggestive of a primary marrow problem. Teardrop cells may be seen in the setting of marrow infiltration (by fibrosis, granulomatous inflammation, haematological or metastatic malignancy), splenic abnormalities, megaloblastic anaemia, and thalassemia.

In this case, the differential is clearly between gastric cancer (with bone marrow metastases resulting in myelodysplasia), and myelofibrosis. Early satiety and vomiting can be a feature of both gastric cancer, and massive splenomegaly. The presence of splenomegaly, and the duration of symptoms are more suggestive of myelofibrosis.

Question 68 of 191

A 56-year-old man presents to the clinic with a 1-week history of yellowing of his skin. He also complains of itching. A systematic inquiry was otherwise unremarkable. He drinks approximately 160 units of alcohol per week. Observations are within normal limits. On examination, there is palpable hepatosplenomegaly.

Blood results are as follows:

Hb	115 g/L	Male: (135-180)Female: (115 - 160)
Platelets	52 * 10 /L 9	(150 - 400)
WBC	4.8 * 10 /L 9	(4.0 - 11.0)
Reticulocytes	195 * 10 /L 9	(50 - 100)
Urea	2.8 mmol/L	(2.0 - 7.0)
Creatinine	42 µmol/L	(55 - 120)
LDH	520 units/L	(140 - 280)
Haptoglobins	<0.5 g/L	(0.5 - 2.2)
Triglycerides	4.2 mmol/L	(<2.3)

Prothrombin time (PT)	16 secs	(10-14 secs)
Activated partial thromboplastin time (APTT)	38 secs	(25-35 secs)
Fibrinogen	2.8 g/L	(2 - 4)
D-Dimer	360 ng/mL	(< 400)

Bilirubin	58 µmol/L	(3 - 17)
ALP	580 u/L	(30 - 100)
ALT	126 u/L	(3 - 40)
Î³GT	860 u/L	(8 - 60)
Albumin	28 g/L	(35 - 50)
Direct antiglobulin test (DAT)		Negative

What is the treatment of choice?

A. Abstinence from alcohol
B. Eculizumab
C. Intravenous immunoglobulin (IVIg)
D. Plasma exchange
E. Prednisolone

A. Abstinence from alcohol

Zieve syndrome usually resolves with abstinence from alcohol

The presence of anaemia with reticulocytosis is suggestive of bleeding or haemolysis. The suppressed haptoglobins, raised bilirubin, and raised LDH are highly suggestive of haemolytic anaemia. The causes of haemolytic anaemia are wide and include hereditary (e.g. membranopathies, enzymopathies, haemoglobinopathies), immunological (e.g. autoimmune haemolytic anaemia), infective (e.g. malaria), and microangiopathic haemolytic anaemias (e.g. TTP, DIC).

It is also important to note that the patient is thrombocytopenic. The cause of this most likely relates to cirrhosis with portal hypertension and splenomegaly, given the significant alcohol history and the presence of hepatosplenomegaly. However, it may also be due to a recent alcohol binge since ethanol is toxic to platelets and can cause transient thrombocytopenia which should resolve within a few days following the binge. However, in the context of haemolytic anaemia, it would also be important to consider microangiopathic haemolytic anaemias (e.g. TTP, HUS, HELLP) which can cause thrombocytopenia due to platelet consumption.

The patient is also coagulopathic which is most certainly related to cirrhosis with decreased production of liver-derived clotting factors.

Abstinence from alcohol is correct. The most likely diagnosis is Zieve's syndrome (ZS) which is characterised by a triad of cholestatic jaundice (high ALP/GGT/bilirubin), DAT-negative haemolytic anaemia, and hyperlipidaemia that develops secondary to alcohol-induced liver injury (most commonly following a binge). The patient fulfils all the criteria of this triad favouring this diagnosis. The treatment for Zieve's syndrome is abstinence from alcohol. Abstinence from alcohol would also be essential for preventing the progression of cirrhosis.

Eculizumab is incorrect. Eculizumab is a terminal complement inhibitor that specifically binds to the complement protein C5 with high affinity, thereby inhibiting its cleavage to C5a and C5b and preventing the generation of the terminal complement complex C5b-9. This drug is used to treat paroxysmal nocturnal haemoglobinuria (PNH). PNH presents with pancytopenia, intravascular haemolytic anaemia, and arterial/venous emboli. Although it remains within the differential diagnosis, the clinical and laboratory features are more in keeping with Zieve's syndrome.

Intravenous immunoglobulin (IVIg) is incorrect. IVIg can be used to treat autoimmunehaemolytic anaemia (AIHA). The patient clearly has haemolysis, however, the negative DAT makes AIHA an exceptionally unlikely diagnosis. Although DAT-negative haemolysis can occur it is exceedingly rare.

Plasma exchange is incorrect. Plasma exchange can be used to treat thrombotic thrombocytopenic purpura (TTP). Thrombotic thrombocytopenic purpura (TTP) is a microangiopathic haemolytic anaemia classically characterised by the pentad of fever, haemolytic anaemia, thrombocytopenia, and renal and neurologic dysfunction. Although it remains within the differential diagnosis, the clinical and laboratory features are more in keeping with Zieve's syndrome.

Prednisolone is incorrect. Prednisolone can be used to treat autoimmune haemolytic anaemia (AIHA). The patient clearly has haemolysis, however, the negative DAT makes AIHA an exceptionally unlikely diagnosis. Steroids can also be used to treat alcoholic hepatitis, however, the liver function tests are predominantly cholestatic in this case.

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A 44-year-old woman presents to the clinic with bruising. This has progressively worsened over the past week. She has also been having frequent nose bleeds which self-terminate. Her past medical history includes rheumatoid arthritis for which she takes weekly methotrexate. She drinks approximately 64 units of alcohol per week.

Her observations are within normal limits. She is noted to have widespread petechia and purpura. On abdominal examination, there is palpable hepatosplenomegaly.

Blood results are as follows:

Hb	120 g/L	Male: (135-180)Female: (115 - 160)
Platelets	2 * 10 /L 9	(150 - 400)
WBC	8.0 * 10 /L 9	(4.0 - 11.0)
Neuts	6.0 * 10 /L 9	(2.0 - 7.0)
Urea	5.4 mmol/L	(2.0 - 7.0)
Creatinine	75 µmol/L	(55 - 120)

Bilirubin	16 µmol/L	(3 - 17)
ALP	88 u/L	(30 - 100)
ALT	146 u/L	(3 - 40)
Î³GT	582 u/L	(8 - 60)
Albumin	32 g/L	(35 - 50)

Prothrombin time (PT)	10 secs	(10-14 secs)
Activated partial thromboplastin time (APTT)	28 secs	(25-35 secs)
Fibrinogen	3.4 g/L	(2 - 4)
D-Dimer	360 ng/mL	(< 400)

What is the most likely cause of this patient's thrombocytopenia?

A. Disseminated intravascular coagulation (DIC)
B. Felty's syndrome
C. Immune thrombocytopenic purpura (ITP)
D. Portal hypertension
E. Thrombotic thrombocytopenic purpura (TTP)

C. Immune thrombocytopenic purpura (ITP)

ITP should be considered in the presence of symptoms that suggest isolated thrombocytopenia e.g. epistaxis, menorrhagia

The causes of thrombocytopenia are wide and include decreased production (e.g. bone marrow failure), increased consumption (e.g. MAHA), and sequestration (e.g. portal hypertension with splenomegaly).

Immune thrombocytopenic purpura (ITP) is correct. ITP is an acquired autoimmune disorder characterised by a low platelet count resulting from peripheral platelet destruction by autoantibodies. It can often be precipitated by malignancy, drugs, vaccinations, or infections. It is often commonly associated with other autoimmune disorders. A single-figure platelet count, with otherwise normal full blood count and a normal coagulation screen, is most likely a consequence of ITP.

Felty's syndrome is incorrect. Felty's syndrome is a rare, extra-articular manifestation of rheumatoid arthritis (RA), characterised by persistent, idiopathic neutropenia and, in some cases, splenomegaly. The absence of neutropenia makes this an unlikely diagnosis.

Disseminated intravascular coagulation (DIC) is incorrect. The normal coagulation profile excludes this diagnosis. DIC presents with prolonged PT, prolonged APTT, low fibrinogen and very high D-dimers.

Thrombotic thrombocytopenic purpura (TTP) is incorrect. Thrombotic thrombocytopenic purpura (TTP) is a microangiopathic haemolytic anaemia classically characterised by the pentad of fever, haemolytic anaemia, thrombocytopenia, and renal and neurologic dysfunction. The absence of any of the other criteria makes this an unlikely diagnosis.

Portal hypertension is incorrect. The patient certainly has features to suggest portal hypertension namely the significant alcohol history, deranged LFTs, and the presence of hepatosplenomegaly. Thrombocytopenia is a common complication in liver disease as a result of multiple factors, including splenic sequestration, reduced activity of the haematopoietic growth factor thrombopoietin, and bone marrow suppression by chronic hepatitis C virus infection. However, it is important to remember that the platelet count is rarely < 50 *10 /L in this setting. Therefore, although this may be a contributing factor in this clinical case, an alternative diagnosis should be considered, and indeed ITP is the most likely diagnosis.

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An 82-year-old man attends the clinic with a 6-month history of drenching night sweats and 10kg weight loss. He also complains of headaches and blurred vision. His observations are within normal limits. On examination, he has bulky bilateral cervical and inguinal lymphadenopathy.

Blood results are as follows:

Hb	115 g/L	Male: (135-180)Female: (115 - 160)
Platelets	185 * 10 /L 9	(150 - 400)
WBC	7.2 * 10 /L 9	(4.0 - 11.0)
Na +	135 mmol/L	(135 - 145)
K +	4.4 mmol/L	(3.5 - 5.0)
Urea	6.2 mmol/L	(2.0 - 7.0)
Creatinine	85 µmol/L	(55 - 120)
CRP	4 mg/L	(< 5)
Total protein	110 g/L	(60-80)

What is the most likely diagnosis?

A. Burkitt's lymphoma
B. Diffuse large B-cell lymphoma
C. Mantle cell lymphoma
D. Marginal zone lymphoma
E. Waldenstrom's macroglobulinaemia

E. Waldenstrom's macroglobulinaemia

Weight loss, raised total protein, hyperviscosity symptoms (headache, blurring) → ? Waldenstrom's macroglobulinaemia

Waldenstrom's macroglobulinaemia is correct. The clinical features are suggestive of lymphoma (e.g. B symptoms and lymphadenopathy). The presence of headaches, and visual impairment, in the context of a raised total protein, is suggestive of hyperviscosity. Although formal serum electrophoresis and immunofixation are required to confirm and classify the paraprotein, it is usually IgM paraproteins that are implicated in hyperviscosity. This is because the IgM molecule is a massive pentameric structure. IgM paraproteins can be associated with many types of lymphoma, however, the classic cause is Waldenstrom's macroglobulinaemia, making this the most likely diagnosis.

Burkitt's lymphoma is incorrect. Burkitt's lymphoma is a highly aggressive high-grade lymphoma. It is the fastest-growing tumour seen in humans with a doubling time of between 24 and 48 hours. Thus the chronicity of the patient's symptoms makes this an exceptionally unlikely diagnosis.

Diffuse large B-cell lymphoma is incorrect. Diffuse large B cell lymphoma (DLBCL) is a highly aggressive high-grade lymphoma. Although not as rapidly growing as Burkitt's, the chronicity of the patient's symptoms makes this a less likely diagnosis.

Mantle cell lymphoma is incorrect. Although mantle cell lymphoma looks like a low-grade lymphoma under the microscope, it often behaviours more like a high-grade lymphoma (e.g. rapid growth). Although it can occasionally produce an IgM paraprotein, it is Waldenstrom's macroglobulinaemia which is more commonly associated with IgM paraproteins making this the more likely diagnosis.

Marginal zone lymphoma is incorrect. There are three types of marginal zone lymphomas (MZL): extranodal MZL of mucosa-associated lymphoid tissue (MALT or gastric GALT), splenic MZL, and nodal MZL. Although IgM paraproteins are occasionally associated with MZL, they are much closer associated with Waldenstrom's macroglobulinaemia making this the favoured diagnosis.

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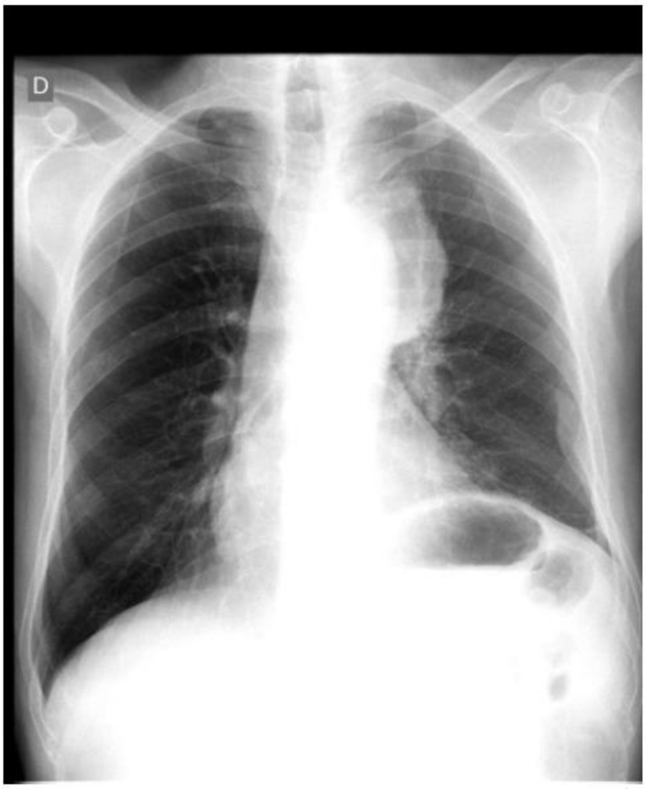
A 70-year-old man is investigated for dysphagia and chest pain. These symptoms have been getting progressively worse for the past 3 months and have not responded to a trial of a proton pump inhibitor. There is no history of weight loss or anorexia.

On examination you note a left-sided partial ptosis. The patient also complains of double vision when you are assessing eye movements. Sustained upward gaze exacerbates his ptosis.

A chest x-ray is requested:



What is the most likely diagnosis?

A. Lung cancer
B. Cardiac myxoma
C. Tuberculosis
D. Sarcodoisis
E. Thymoma

E. Thymoma

Anterior mediastinal mass + symptoms of myasthenia = thymoma

The chest x-ray shows is a partially delineated mediastinal mass (anterior mediastinum) with regular borders, bulging the left upper mediastinal contour. These findings are consistent with a thymoma.

The history is highly suggestive of myasthenia gravis which is seen in around a third of patients with a thymoma. Note how the ptosis worsened with sustained upward gaze, a demonstration of fatigability.

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A 53-year-old man presents to the emergency department with a rash and ulceration on his lower limbs. He has a past medical history of untreated hepatitis C virus. He is not on any regular medications. He is homeless. He is an intravenous drug user.

On examination, there is a purpuric rash on his lower limbs with ulceration of the digits. There is no clinical evidence of synovitis.

Blood tests:

Hb	121 g/L	Male: (135-180)Female: (115 - 160)
Platelets	422 * 10 /L	(150 - 400)
WBC	5.2 * 10 /L	(4.0 - 11.0)
Na	138 mmol/L	(135 - 145)
K	4.2 mmol/L	(3.5 - 5.0)
Urea	5.2 mmol/L	(2.0 - 7.0)
Creatinine	141 µmol/L	(55 - 120)
CRP	55 mg/L	(< 5)
Rheumatoid factor	654 IU/ml	(0-20)
Complement (C3)	0.81 g/L	(0.75 - 1.65)
Complement (C4)	.02 g/L	(0.16 to 0.48)
Antinuclear antibody	negative	(negative)
ESR	101 mm/Hr	(0-20)
IgA	10.2 g/L	(6.60 - 15.90)
IgG	24.1 g/L	(6-16)
IgM	4.2 g/L	(0.53-2.47)

Urinalysis:

Leucocytes	negative
Nitrites	negative
Blood	+++
Protein	++
Glucose	negative

What is the likely diagnosis?

A. Rheumatoid vasculitis
B. Systemic lupus erythematosus
C. Type I cryoglobulinaemia
D. Type II cryoglobulinaemia
E. Type III cryoglobulinaemia

D. Type II cryoglobulinaemia

Hepatitis C is associated with mixed (type II) cryoglobulinaemia

Type II cryoglobulinaemia is correct. This patient presents with a purpuric rash on the lower limbs with distal ulceration in association with hypocomplementaemia and a positive rheumatoid factor on a background of hepatitis C. This suggests a diagnosis of cryoglobulinaemia. Hepatitis C is associated with Type II cryoglobulinaemia. The haematoproteinuria and deranged renal function suggest glomerulonephritis. Rheumatoid factor may be very high in this condition.

Rheumatoid vasculitis is incorrect. This may cause a rash, ulceration and positive rheumatoid factor. However, it tends not to cause low complement and glomerulonephritis and is, therefore, less likely. Additionally, it tends to occur in longstanding rheumatoid, which is absent here.

Systemic lupus erythematosus is incorrect. This can be associated with rash, low complement, rheumatoid factor and ulceration. However, a negative ANA essentially excludes the diagnosis.

Type I cryoglobulinaemia is incorrect. This form of cryoglobulinaemia is typically associated with a monoclonal IgM or IgG rather than a polyclonal picture. It is associated with multiple myeloma and Waldenstrom's macroglobulinaemia.

Type III cryoglobulinaemia is incorrect. This type of cryoglobulinaemia is typically associated with rheumatoid arthritis and Sjogren's rather than hepatitis C. The absence of joint symptoms and the negative ANA make rheumatoid arthritis and Sjogren's unlikely.

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A 19-year-old African male was referred by his GP to the medical assessment unit with a complaint of dark coloured urine, abdominal pain and jaundice. He does not have any other co-morbidities and there is no family history of haemolytic anaemias. He recently tried a new Mediterranean diet containing fava beans. On examination, there is moderate splenomegaly.

Hb	70 g/l	Na +	137 mmol/l	Bilirubin	107 µmol/l
Platelets	280 * 10 /l 9	K +	4.3 mmol/l	ALP	98 u/l
WBC	8.7* 10 /l 9	Urea	5.6 mmol/l	ALT	25 u/l
Neuts	5.6 * 10 /l 9	Creatinine	77 µmol/l	γGT	37 u/l
Lymphs	1.3 * 10 /l 9	Albumin	35 g/l		
Eosin	0.9 * 10 /l9				

Serum haptoglobin was undetectable, and serum lactate dehydrogenase (LDH) was 1987 units/l. Peripheral blood film showed Heinz bodies. A direct Coombe's test was negative. G6PD levels return as normal.

What investigation should be performed next to confirm the diagnosis?

A. Repeat G6PD level immediately as the first specimen may have been inadequate
B. Ultrasound abdomen
C. Hepatitis serology
D. Bone marrow examination
E. G6PD analysis in few weeks when symptoms improved

E. G6PD analysis in few weeks when symptoms improved

G6PD deficiency: G6PD level may be normal during an acute haemolytic episode

G6PD testing should be performed when patients are in remission as the result may be falsely negative during acute haemolysis. The reason for this is that during an acute haemolytic episode, older erythrocytes with low G6PD levels are destroyed and there is a compensatory increase in immature erythrocytes and reticulocytes that have increased G6PD levels. Although ultrasound abdomen may be useful in assessing for splenomegaly and gallstones in this patient, it's not diagnostic of G6PD deficiency.

Hepatitis serology is more useful in the setting of jaundice secondary to hepatobiliary disease. Bone marrow examination is not an investigation of choice in G6PD deficiency.

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A 72-year-old man is brought into the emergency department following a fall. He was found by his daughter unresponsive on the floor after hearing a loud bang. His daughter tells you that he has been more unsteady on his feet for the last few days and is recovering from a urinary tract infection. There is a past medical history of benign prostatic hyperplasia and atrial fibrillation for which he takes tamsulosin, finasteride and rivaroxaban.

On examination, he is drowsy with a GCS of 12 (E3V4M5). He has a heart rate of 85bpm and blood pressure of 210/118mmHg. On auscultation, his chest is clear with normal heart sounds. Oxygen saturations are 90% on air. There is a deep laceration over the left side of his forehead which is oozing blood and there is bruising down the left side of his body. Pupils are equal and reactive to light.

CT Head: Evidence of a moderate intracranial haemorrhage in the left frontal lobe. There is no evidence of a mass effect.

What is the most appropriate next step in the management of this patient?

A. Andexanet alfa
B. Cryoprecipitate
C. Dexamethasone
D. Idarucizumab
E. Protamine

A. Andexanet alfa

Rivaroxaban and apixaban can be reversed by andexanet alfa

Important for me Less im This patient has had a traumatic intracranial haemorrhage. Initial treatment should be to maintain a patent airway and attempt to stop the bleeding in order to prevent raised intracranial pressures. With patients taking anticoagulant therapy, actions should be taken to reverse their action. Andexanet alfa is a recombinant form of factor Xa which binds specifically to apixaban or rivaroxaban to reverse their anticoagulant effects.

Cryoprecipitate is derived from fresh frozen plasma and contains factor VIII, factor XIII, fibrinogen, vWF and fibronectin. It is used to treat fibrinogen deficiency in cases of disseminated intravascular coagulation, trauma, invasive procedures or haemorrhage. It is more commonly used to treat an intracranial haemorrhage secondary to tissue plasminogen activator use for an acute ischaemic stroke.

Dexamethasone can be given in the case of increased intracranial pressure, which can be secondary to intracranial haemorrhage and subsequent mass effect. However, the current investigations do not show evidence of cerebral oedema or midline shift and therefore dexamethasone is not the next initial treatment to be given.

Idarucizumab is a specific reversal agent for dabigatran. As this patient is not taking dabigatran therapy, the use of idarucizumab is not indicated.

Protamine is used in the reversal of heparin. As this patient is not taking heparin therapy, the use of protamine is not indicated.

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A 24-year-old female presents to her GP with a 2 month history of tiredness. On further questioning she admits she has also been experiencing pain in the centre of her chest and in the lymph nodes in her neck when drinking alcohol.

What is the likely underlying cause?

A. Hodgkin's lymphoma
B. Somatisation disorder
C. GORD (gastro-oesophageal reflux disease)
D. Hiatus hernia
E. Pancreatitis

A. Hodgkin's lymphoma

Lymph node pain when drinking alcohol is very specific for Hodgkin's lymphoma (although it occurs rarely)

Lymph node pain when drinking alcohol is very specific for Hodgkin's lymphoma. It is however a rare symptoms and most patients do not experience it.

None of the other conditions mentioned cause pain in lymph nodes

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You are the medical registrar on call. The surgical registrar contacts you for a patient he has just seen in clinic who requires an elective cholecystectomy. The patient is a 65-year-old woman who has atrial fibrillation for which she takes rivaroxaban. The patient is otherwise well. The bloods performed in clinic that day are as follows:

Hb	131 g/l
Platelets	352 * 10 /l9
WBC	5.5 * 10 /l9
INR	1.5
Na +	137 mmol/l
K +	3.6 mmol/l
Urea	3.2 mmol/l
Creatinine	67 µmol/l

The surgical registrar would like to know how long the patient should omit their anticoagulation before the procedure?

A. 1 day
B. 2 days
C. 3 days
D. 5 days
E. 7 days

A. 1 day

The new oral anticoagulant drugs (NOACs) are being used increasingly in patients who require anticoagulation. They have the advantage of once or twice daily dosing regimes, oral formulation and do not require therapeutic monitoring like warfarin. They are particularly useful in patients that may have issues with warfarin compliance or those with erratic international normalised ratio (INR) readings. NOACs have been shown to convey equivalent efficacy to warfarin in most contexts the main exception being anticoagulation in patients with prosthetic heart valves where NOACs were not as effective as warfarin.

Rivaroxaban is one of the most commonly used agents. It acts as a direct factor Xa inhibitor. It is absorbed from the gut and has maximum factor Xa inhibition four hours post dose. It is indicated for anticoagulation in atrial fibrillation and in the treatment of venous thromboembolism. It has a half-life of 7-9 hours and is metabolised in the liver. Factor Xa levels do not return to normal for just over 24 hours so once daily dosing is appropriate.

The following table outlines how long anticoagulants need to be withheld before surgery:

Drug	Duration to withhold before procedure
Dabigatran	1-2 days with creatinine clearance >50ml/min
Rivaroxaban	1 day if creatinine clearance >90 ml/min
Apixaban	1-2 days if creatinine clearance >60
Fondaparinux	36-48 hours
LMWH	12 hrs for prophylactic dose, 24 hours for therapeutic dose
Warfarin	1-8 days, check INR
UFH	IV 4-6 hrs, SC 12-24 hrs

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A 45-year-old worker on demolition sites comes to the Emergency department for review. He has suffered increasing tiredness, lethargy, headache, and abdominal pains over the past 2 months. On examination he is hypertensive with a blood pressure of 155/90 mmHg, his pulse is 85 beats per minute and regular. He looks pale.

Investigations

Hb	97 g/l
MCV	78 fl
Platelets	175 * 10 /l9
WBC	6.2 * 10 /l9
Lead	5 µmol/l

Blood film reveals basophilic stippling

Which of the following is the most appropriate initial intervention?

A. Activated charcoal
B. Disodium EDTA
C. DMSA
D. Haemodialysis
E. Vitamin C

C. DMSA

DMSA is an oral chelation therapy that can be used for chronic lead poisoning, the diagnosis here. A lead level above 3.4 µmol/l indicates significant occupational exposure and that the patient should be withdrawn from work. 500mg twice per day is a usual initial therapeutic dose.

Given his lead exposure represents chronic exposure, there is no role for activated charcoal. Disodium EDTA is used in the management of acute lead poisoning and is given intravenously. Human trials of vitamin C with respect to treatment of lead toxicity are so far equivocal, and there is no role for haemodialysis in chronic lead poisoning.

Question 78 of 191

A 65-year-old man is seen in the haematology clinic for review of test results. He has been fatigued for the last three months and has experienced night sweats. On examination he is pale with a palpable spleen 4cm below the costal margin, mildly tender on palpation. His only other past medical history is of osteoarthritis.

His results are as follows:

Hb	89 g/l	Na +	140 mmol/l
Platelets	205 * 10 /l 9	K +	4.0 mmol/l
WBC	5 * 10 /l 9	Urea	5.8 mmol/l
Neuts	3.6 * 10 /l 9	Creatinine	72 µmol/l
Lymphs	1.2 * 10 /l 9	CRP	3 mg/l

Blood film: Anisocytosis with mild hypochromia. Tear drop cells. CT chest/abdomen/pelvis: Splenic enlargement. No suspicious mass lesions seen. 1cm simple right renal cyst. No lymphadenopathy.

Bone marrow biopsy: Fibrosis

Which initial therapy should be used to treat this gentleman?

A. Chlorambucil
B. Fludarabine
C. Hydroxycarbamide
D. Interferon alpha
E. Lenalidomide

C. Hydroxycarbamide

This gentleman has myelofibrosis as evidenced by bone marrow fibrosis, tear drop cells on blood film and constitutional symptoms. He has symptomatic splenomegaly and anaemia. First line treatment for this would be hydroxycarbamide.

Chlorambucil is used in chronic lymphocytic leukaemia and non-Hodgkin lymphoma. Fludarabine is used to treat acute myeloid leukaemia. Interferon alpha is used for myelosuppression in myelofibrosis in the presence of thrombocytosis or leucocytosis. Lenalidomide is used in the treatment of multiple myeloma

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A 30-year-old lady presents with shortness of breath and facial flushing. She had been to a restaurant that evening and noticed the symptoms when rushing home. She also had itchy skin and tingling lips. On examination she was tachycardic with a wheeze. She had no history of food allergies and reported eating lasagne for dinner alongside a glass of wine.

What is the most likely diagnosis?

A. Acute asthma attack
B. Carcinoid syndrome
C. Exercise induced wheat angioedema
D. Shellfish anaphylaxis
E. Thyrotoxicosis

C. Exercise induced wheat angioedema

Exercised induced anaphylaxis is now well described and is most associated with wheat ingestion. The clinical manifestations usually occur around 10 minutes after exercise and follow a sequence of pruritus, widespread urticaria and then subsequently respiratory distress and vascular collapse. The condition usually resolves on stopping exercise and is managed in the same manner as anaphylaxis. The patients can usually eat the causative food without problems so long as they do not exercise afterwards. The physiology of the condition remains slightly unknown, but it may be related to endorphin release during exercise. The endocrines cause excessive histamine release from mast cells in susceptible individuals.

The tingling lips and facial flushing are not in keeping with an asthma attack and there is no history of shellfish consumption. Thyrotoxicosis could explain some of the symptoms but not the wheeze. Carcinoid syndrome can cause episodes of flushing, shortness of breath and tachycardia and thus represents a plausible option, especially as exercise, food and stress can trigger these episodes. However these patients often have diarrhoea and abdominal symptoms alongside their other symptoms.

Question 80 of 191

A 54-year-old man presents to the emergency department with shortness of breath. He has a history of heart failure and takes ramipril, bisoprolol, eplerenone and isosorbide mononitrate.

Observations are as follows: heart rate 135 beats per minute, respiratory rate 26 breaths per minute, blood pressure 135/82 mmHg, temperature 37.4 ºC, and SpO2 92%. His chest is clear on auscultation.

The doctors initiate the patient on 15L O2 via a non-rebreather mask. Blood gas analysis results are as follows:

pH	7.24	(7.35 - 7.45)
PaO2	72.4 kPa	(10.3 - 13.3 kPa)
PaCO2	2.9 kPa	(4.7 - 6 kPa)
HCO3	16 mmol/L	(22 - 28 mmol/L)
SaO2	44%	(94 - 98%)
Lactate	3.4 mmol/L	(<1.0)

What is the most likely diagnosis?

A. Acquired methaemoglobinaemia
B. Carboxyhaemoglobinaemia
C. Congenital methaemoglobinaemia
D. Cyanide poisoning
E. Pulmonary embolism

A. Acquired methaemoglobinaemia

Nitrates, including recreational nitrates such as amyl nitrite ('poppers') may cause methaemoglobinaemia

The striking feature, in this case, is the significant discrepancy between the PaO2 and SaO2 on arterial blood gas analysis. Given the patient is on 15L O2, the PaO2 is at a level expected indicating that gas exchange at the level of the alveoli is not impaired. There are various formulas for predicting the expected PaO2 for any given inspired O2 concentration. The Advanced Life Support (ALS) guidelines suggest that the PaO2 for any given inspired concentration should be approximately 10 less than the inspired concentration (%). For example, in this case, 15L O2 is equivalent to approximately 80-85% so we would expect the PaO2 to be around 70 kPa.

However, it is important to note that the SaO2 (oxygen saturation of haemoglobin) is markedly low at 44% indicating impaired oxygen binding to haemoglobin. It is also important to note that the peripheral SpO2 is 92% which is clearly discrepant with the SaO2. The two main causes of this picture are methaemoglobinaemia and carboxyhaemoglobinaemia. Because standard pulse oximeters are not capable of differentiating between different forms of bound haemoglobin, the SpO2 is usually falsely high in these conditions.

Tissue hypoxia can be a consequence of 4 main causes:

1. Hypoxaemia (low PaO2 due to poor gas exchange at the alveoli)
2. Toxic haemoglobin (low SaO2 due to methaemoglobinaemia or carboxyhaemoglobinaemia)
3. Perfusional (e.g. shock, or localised causes such as compartment syndrome or bowel ischaemia)
4. Severe anaemia

Regardless of the cause, tissue hypoxia will result in lactic acidosis. Therefore, in patients with lactic acidosis, all the aforementioned causes should be considered. The patient in this case most certainly has lactic acidosis as a consequence of toxic haemoglobin-induced tissue hypoxia. He is acidotic with low serum bicarbonate since this acts as a buffer for lactic acid.

Acquired methaemoglobinaemia is the correct answer. As discussed above, the biochemical results are suggestive of toxic haemoglobin. The history of isosorbide mononitrate use favours the diagnosis of acquired methaemoglobinaemia. Nitrate-containing drugs are commonly implicated in this condition.

Carboxyhaemoglobinaemia is incorrect. Although carbon monoxide poisoning presents similarly and remains within the differential diagnosis, the history of nitrate exposure favours the diagnosis of methaemoglobinaemia.

Congenital methaemoglobinaemia is incorrect. Although this remains within the differential diagnosis, the absence of previous episodes and the presence of a known risk factor (e.g. nitrate exposure) favours the acquired form.

Cyanide poisoning is incorrect. Cyanide binds the ferric (Fe3+) ion of cytochrome oxidase causing 'histotoxic hypoxia' and lactic acidosis. It would not cause a marked reduction of SaO2 as seen in this case unless it was associated with coexistent carbon monoxide poisoning (e.g. due to smoke inhalation). There is nothing in this clinical case to suggest that this is the case.

Pulmonary embolism is incorrect. As stated above, the blood gas analysis confirms hypoxaemia as a consequence of toxic haemoglobin rather than poor gas exchange at the level of the alveolar. Therefore a pulmonary embolism is a less likely diagnosis.

Question 81 of 191

A 75-year-old man presents to the emergency department with a reduced level of consciousness after a recent fall and head injury. He has a past medical history of atrial fibrillation. His medications include apixaban and bisoprolol.

On examination, his Glasgow coma scale is 12/15 (E 3 V4 M5). There is evidence of an external head injury with bruising and laceration in the right occipital area.

An urgent CT head is arranged, which demonstrates a large right-sided subdural haematoma.

What is the most appropriate medication to administer?

A. Andexanet alfa
B. Fresh frozen plasma
C. Vitamin K
D. Prothrombin complex
E. Idarucizumab

A. Andexanet alfa

Rivaroxaban and apixaban can be reversed by andexanet alfa

Andexanet alfa is the correct answer. The patient has sustained a traumatic subdural haematoma. Clinically he has a low GCS. The method of anticoagulation needs to be reversed. The most appropriate reversal agent for apixaban is andexanet alfa.

Vitamin K is incorrect. This can be used as a reversal agent for warfarin. It is not the most appropriate agent to use given this patient's method of anticoagulation.

Fresh frozen plasma is incorrect. This is mainly used to replace coagulation factors in cases of excessive bleeding or in those with abnormal coagulation. It is not the most appropriate reversal agent available to choose in this case.

Prothrombin complex is incorrect. This can be used to rapidly reverse the effects of warfarin in those patients that are bleeding. It is under investigation currently to see if it is effective in reversing agents such as apixaban Idarucizumab is incorrect. This is a monoclonal antibody that can reverse the effects of dabigatran

Question 82 of 191

You are looking after a 35-year-old man who is in the oncology day unit receiving his second round of chemotherapy for a low grade non-Hodgkin's lymphoma. His lymphoma is confined to two lymph node groups in his anterior cervical chain and right inguinal region. In total there are 6 nodes with the largest being 4cm in size. He had no issues during his first round of chemotherapy apart from some nausea a week afterwards. He has no other medical problems and is on no other medications. His bloods pre-chemotherapy are as shown below:

Na +	137 mmol/l
K +	3.8 mmol/l
Urea	2.8 mmol/l
Creatinine	55 µmol/l
Corrected Calcium	2.39 µmol/l
Phosphate	1.05 µmol/l

What regimen would be most appropriate for prevention of tumour lysis syndrome in his case?

A. Allopurinol (200mg BD)
B. Fluids and allopurinol (200mg BD)
C. Fluids and rasburicase (0.2mg/kg)
D. Reduced dose chemotherapy
E. Fluids, allopurinol (200mg BD) and rasburicase (0.2mg/kg)

B. Fluids and allopurinol (200mg BD)

The British Society of Haematology suggest that patients undergoing chemotherapy can be categorised into three different risk groups for tumour lysis syndrome. While you are not required to know how to do this in detail it would be important clinically to be able to recognise factors that put people at increased risk:

- High tumour burden
- High grade tumours with rapid cell turnover
- Pre-existing renal impairment or renal involvement by the tumour Increased age
- Treatment with highly active, cell-cycle specific agents
- Concomitant use of drugs that increase uric acid levels (the list is available on the guidance)

Risk Group	Prophylaxis
Low Risk	Adequate hydration (consider IV fluids and allopurinol prophylaxis)
Intermediate Risk	Allopurinol (7 days) and IV fluids
High Risk	Rasburicase and IV fluids (consider low dose chemotherapy)

This patient has no high risk features in his history and is therefore considered low risk, so the closest answer is fluids and allopurinol.

It is important to know that allopurinol and rasburicase should not be used together, as allopurinol reduces the effectiveness of rasburicase. Allopurinol reduces the production of urate, allowing elimination of upstream compounds, which are more water soluble and thus more easy to excrete through the kidneys. Rasburicase, on the other hand, converts urate into more easily excreted downstream compounds.

In addition rasburicase cannot be given to patients with G6PD as it can precipitate haemolysis.

Question 83 of 191

A 24-year-old man is brought into the resuscitation room. They were struggling to walk across the emergency department reception area due to severe dyspnoea.

They admit to inhaling some substances at a party earlier in the evening but cannot recall what it was.

On examination, they have blue-tinged peripheries with blue lips and tip of nose but appear relatively undistressed. Their observations show oxygen saturations of 90% on 6 litres of oxygen and a heart rate of 120 beats per minute.

An arterial blood gas is taken while they are on 6 litres of oxygen. The results are below.

pH	7.40	(7.35-7.45)
paO2	18	(11-13)
paCO2	4.6	(4.5-6)
methaemoglobin	38%	(1-3%)

What is the most likely substance that they have ingested?

A. Amyl nitrite
B. Glyceryl trinitrate
C. Sodium nitroprusside
D. Aniline dye
E. Dapsone

A. Amyl nitrite

Nitrates, including recreational nitrates such as amyl nitrite ('poppers') may cause methaemoglobinaemia

The answer is amyl nitrite otherwise known as 'poppers' which is a drug of abuse. It induces a short lived euphoric high but can result in methaemoglobinaemia. The other answers all cause methaemoglobinaemia but are unlikely to be used in the above context.

Question 84 of 191

You are called to see a 55-year-old gentleman who is having a blood transfusion for symptomatic anaemia secondary to colon cancer. The nurses have been doing regular observations as documented below.

10am:

Baseline observations prior to blood transfusion

- Respiratory rate 20 breaths/min
- Saturations 96% on air
- Temperature 37.5 ºc
- Blood pressure 145/78 mmHg
- Heart rate 74 beats/min

10:15 am: repeat observations after 15 mins of transfusion

- Respiratory rate 19 breaths/min
- Saturations 97% on air
- Temperature 38.2 ºc
- Blood pressure 150/80 mmHg
- Heart rate 72 beats/min

The nurses have already stopped the blood transfusion by the time you arrive to see the patient. On questioning the patient he feels well with no complaints of pain, itch or rashes. On examination his heart sounds are pure and chest is clear. He has no previous documented reactions to blood transfusions although the patient informs you he is allergic to penicillin.

What instructions do you give the nurses?

A. Take blood cultures and commence antibiotics
B. Dispose of the remaining blood in the bag
C. Restart the blood transfusion after giving the patient paracetemol
D. Take blood cultures, repeat chest x ray and perform urinalysis
E. Restart the blood transfusion an hour after giving the patient paracetemol

C. Restart the blood transfusion after giving the patient paracetemol

Blood transfusion reaction are common and can be serious. This patient is well and has no clinical evidence of haemodynamic compromise. He has an isolated pyrexia which is likely to be secondary to commencement of the blood transfusion. After confirming this his blood transfusion should be restarted as soon as possible and paracetemol given for symptomatic relief. Disposal of the blood should be considered if a serious adverse reaction occurs. Septic screen and antibiotics should be considered if any underlying infection is suspected. Blood transfusion should be completed within 4 hours and waiting an hour prior to restarting is a waste of valuable time.

Question 85 of 191

A 55-year-old lady presents to her general practitioner with a painful rash on her breasts. The rash has developed over the last 24 hours and although initially diffuse and red is now well demarcated and a much darker red. She was started on warfarin five days ago for a deep vein thrombosis diagnosed on ultrasound doppler. She has a past medical history of hypothyroidism, type two diabetes mellitus, thromboembolism and obesity. She remembers having been on warfarin at least once before for previous deep vein thrombosis but does not remember ever having had this rash. Her blood tests today are as follows.

Prothrombin time	21.4 seconds
INR	2.1 ratio

What is the most likely diagnosis?

A. Protein S deficiency
B. Idiosyncratic drug reaction
C. Stevens Johnson syndrome
D. Warfarin toxicity
E. Protein C deficiency

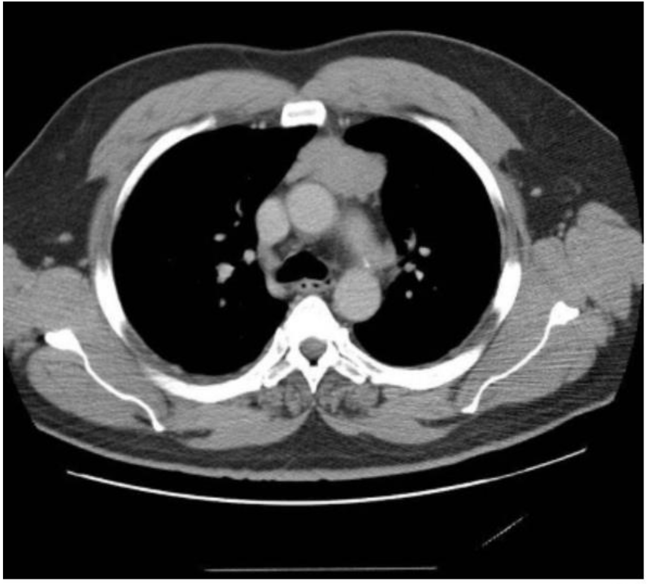
E. Protein C deficiency

Warfarin-induced skin necrosis is a symptom of the hypercoagulable state that can be seen in the first week of warfarin therapy. This is explained by the fact that warfarin inhibits both procoagulant factors (2, 7, 9, 10) as well as anti-coagulant factors (protein C). Protein C has the shortest half-life and therefore there is a short period of time during the initiation of warfarin therapy where the patient is hypercoagulable despite a therapeutic INR. Warfarin-induced skin necrosis is more likely to occur in patients with pre-existing protein C deficiency, which is a rare congenital condition associated with recurrent venous thromboembolic disease.

Question 86 of 191

A 41-year-old woman presents to her general practitioner with a three-month history of shortness of breath and cough. Her symptoms have been progressive and she is otherwise asymptomatic. She has no relevant medical comorbidities but a 30 pack-year smoking history.

Examination of the respiratory systems is unremarkable. She has a CT thorax which is displayed below:



What is the most likely cause for this woman's symptoms?

A. Bronchial cancer

B. Chronic pulmonary emboli
C. Lung fibrosis
D. Thymoma
E. Thyroid cancer

D. Thymoma

This woman's CT scan demonstrates a large mass representing a thymoma in the anterior mediastinum. Thymomas can commonly cause shortness of breath and cough, as well as retrosternal chest pain, dysphagia, and vocal changes. Although bronchial cancer could cause this woman's symptoms, there is no evidence of this pathology on the CT and therefore this answer is incorrect.

There is no evidence on this CT scan of a pulmonary embolus despite this not being a pulmonary angiogram. As the scan does identify a thymoma, chronic pulmonary emboli are therefore less likely and incorrect.

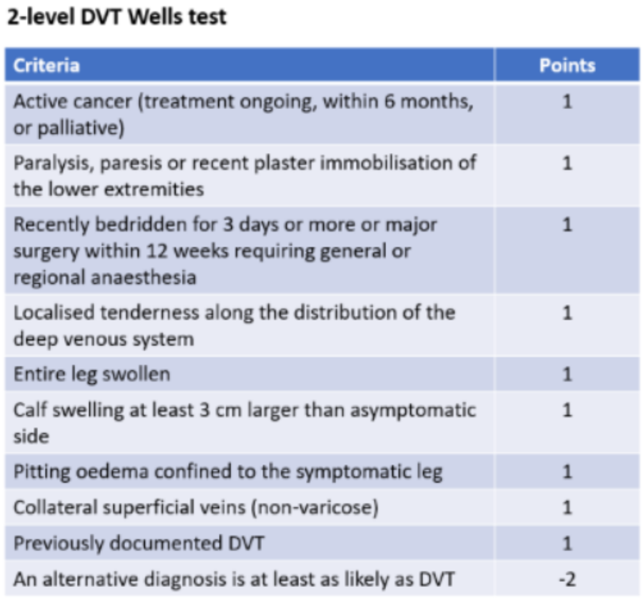
Although not a high-resolution scan, there is no evidence of fibrosis within the lung fields and therefore this answer is incorrect.

The anterior mediastinal mass shown on this CT scan is too inferior to be a thyroid cancer which would typically present as a lump in the neck. Therefore the diagnosis of thymoma is more likely and thyroid cancer is incorrect.

Question 87 of 191

A 64-year-old woman presents to her general practitioner with a one-day history of swollen left leg associated with pain. She has a past medical history of heart failure and hypertension and takes bisoprolol, ramipril, amlodipine, and furosemide. She is significantly limited in her mobility.

On examination, the left calf is red and 4cm larger than the right. There is tenderness over the deep venous system. There is bilateral, symmetrical pedal oedema. The left thigh is unremarkable.



2-level DVT Wells test

Criteria	Points
Active cancer (treatment ongoing, within 6 months, or palliative)	1
Paralysis, paresis or recent plaster immobilisation of the lower extremities	1
Recently bedridden for 3 days or more or major surgery within 12 weeks requiring general or regional anaesthesia	1
Localised tenderness along the distribution of the deep venous system	1
Entire leg swollen	1
Calf swelling at least 3 cm larger than asymptomatic side	1
Pitting oedema confined to the symptomatic leg	1
Collateral superficial veins (non-varicose)	1
Previously documented DVT	1
An alternative diagnosis is at least as likely as DVT	-2

What is the most appropriate next step?

A. CT pulmonary angiogram
B. D-dimer
C. Inferior vena cava filter
D. Lower limb venous ultrasound
E. VQ scan

D. Lower limb venous ultrasound

This woman has a 2-level Wells' score of 2 (calf swelling, tenderness over the deep venous system) for deep vein thrombosis (DVT). NICE guidance states that a score of 2 or above should be investigated by **lower limb venous ultrasound** rather than high sensitivity d-dimer testing. **CT pulmonary angiography** (CTPA) could be considered to check for a pulmonary embolus (PE) once a DVT is confirmed however this is not indicated at this stage.

High-sensitivity **d-dimer** testing is the first-line investigation for patients who have a 2-level Wells' score of less than 2. As this patient scores 2, a lower limb ultrasound should be performed and therefore this is an incorrect answer.

An **inferior vena cava filter** is a treatment for DVT, particularly in cases where anticoagulation is not an option. As a DVT has not been confirmed, this would not be the most appropriate next step.

A **VQ scan** is an alternative imaging modality to CTPA for PE however as previously discussed, prior to confirmation of a DVT and with no clinical indication, this test would not be indicated at this stage.

Question 88 of 191

A 19-year-old man presents to your clinic. For as long as he can remember, he has had a dusky discolouration that is not in keeping with the skin colour of any of his relatives, and he feels that it is becoming gradually duskier with time. He tells you that. The only medication he is taking is oral isotretinoin, for acne, which he has been taking on-and-off for four years. Auscultation of his chest was unremarkable. Oxygen saturation by pulse oximetry showed saturation of 91% and an arterial blood gas was done which showed a pO2 of 10.6kPa, FO2Hb of 65% and metHb of 30%.

What is the most appropriate treatment for this man?

A. Ascorbic acid
B. Corticosteroids
C. Methylene blue
D. Nocturnal CPAP
E. Vitamin A

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A 24 year-old medical student of Italian extraction returns from elective in India. One week after his return he presents with fever, headache, and myalgia.

Investigations are as follows:

Hb	10.1 g/dl
MCV	101.2 fl
Platelets	43 x10^9/l
WCC	6.1 x10^9/l
Na	134mmol/l
K	4.6 mmol/l
Urea	3.8 mmol/l
Creatinine	80 mol/l

ALT	44 IU/l
ALP	78 IU/l
Bilirubin	33 mol/l
Albumin	38 g/l
Thick and thin blood films	Plasmodium ovale parasites with red cells

What other blood test will be essential for the management of this condition?

A. Coomb's test
B. G6PD enzyme assay
C. Chloroquine resistance test
D. Ham's test
E. Reticuloctye count

B. G6PD enzyme assay

Primaquine can precipitate haemolysis in individuals with G6PD (glucose-6-phosphate dehydrogenase) deficiency. G6PD activity should be tested before commencing treatment. If deficient, expert advice should be sought.

Coomb's test, also known as the direct antiglobulin test (DAT), detects antibody bound to the surface of red cells and is a test for autoimmune haemolytic anaemia. A variable degree of haemolysis occurs in malaria, but is not typically autoimmune. Rather it is due predominantly to destruction of infected red cells during parasite replication and breakdown in the spleen.

Chloroquine is the first-line treatment for non-falciparum malaria. Chloroquine resistance in *Plasmodium ovale* and *malariae* is extremely rare, but has been reported for vivax. Chloroquine is inadequate for the treatment of falciparum malaria as resistance is widespread.

Ham's test was previously used for the diagnosis of paroxysmal nocturnal haemoglobinuria, a condition which causes intravascular haemolysis.

Reticulocytes are red cell precursors, which are prematurely released from the marrow in response to haemolysis. Their large size accounts for the macrocytosis which often accompanies haemolytic anaemia. An elevated reticulocyte count is an appropriate response to haemolysis in the presence of an adequately functioning bone marrow.

Question 90 of 191

A 61-year-old woman comes for review. Around one year ago she finished a 6 month course of warfarin after being diagnosed with an unprovoked, proximal deep vein thrombosis. For the past few weeks she has been experiencing 'heaviness' and 'aching' in the the same leg. This is associated with an itch and some swelling, although this seems to go down each night. Past medical history of note includes osteoarthritis and type 2 diabetes mellitus.

On examination prominent varicose veins are seen on the affected leg with some brown discolouration of the skin above the medial malleolus. There is no difference in the circumference of the calves. Her temperature is 36.9ºC, pulse 78/min and blood pressure 108/82 mmHg.

What is the most likely diagnosis?

A. Recurrence of deep vein thrombosis
B. Post-thrombotic syndrome
C. Cellulitis
D. Ruptured Baker's cyst
E. Necrobiosis lipoidica

B. Post-thrombotic syndrome

Post-thrombotic syndrome is the correct diagnosis. This chronic condition develops in approximately 20-50% of patients following deep vein thrombosis (DVT) and typically presents with symptoms including leg heaviness, aching, pruritis, and oedema that improves with elevation (such as overnight). The presence of varicose veins and skin changes (particularly brown discolouration or hyperpigmentation) above the medial malleolus are classical signs. The pathophysiology involves damage to venous valves during the initial DVT, leading to chronic venous insufficiency.

Recurrence of DVT would be less likely in this case. While unprovoked DVT does carry a higher risk of recurrence, the clinical features are different. Acute DVT typically presents with more significant unilateral leg swelling, warmth, and tenderness. The improvement in symptoms overnight and chronic skin changes point away from acute DVT.

Cellulitis can be excluded based on the presentation. Cellulitis typically presents with acute onset erythema, warmth, tenderness, and often systemic symptoms such as fever. The patient's normal temperature and chronic nature of symptoms make this diagnosis unlikely.

Ruptured Baker's cyst typically presents with acute calf pain and swelling, often following activity. The pain is usually localised to the popliteal fossa and posterior calf. The chronic nature of symptoms and venous changes make this diagnosis unlikely.

Necrobiosis lipoidica is a rare condition associated with diabetes mellitus that presents with welldemarcated, waxy, atrophic plaques, typically on the anterior shin. While this patient does have diabetes, her presentation with venous symptoms and characteristic post-thrombotic changes makes necrobiosis lipoidica an unlikely diagnosis.

Question 91 of 191

A 62-year-old gentleman with a background of Rheumatoid Arthritis, on maintenance sulfasalazine, is referred to the medical take from his GP with severe unremitting flu-like symptoms and deranged blood tests.

On examination, his temperature is 38.3 degrees celsius, heart rate 104bpm, respiratory rate 31/min and oxygen saturations 92% on room air.

His blood test reveals the following:

Hb	82 g/l
Platelets	52 * 10 /l9
WBC	18 * 10 /l9
Ferritin	50,000ng/ml
EBV Monospot test +ve	

He is initially treated as neutropaenic sepsis, with broad spectrum antimicrobials, and transferred to the intensive care unit for organ support. Unfortunately, he does not make any improvement. He is then seen by haematology who organise a bone marrow aspirate, revealing haemophagocytosis

. What is the most likely underlying diagnosis?

A. Macrophage activation syndrome
B. Drug-induced pancytopaenia
C. Atypical infection
D. Parvovirus infection
E. Felty syndrome

A. Macrophage activation syndrome

Macrophage activation syndrome is a condition characterised by excessive immune stimulation and cytokine storm in a rheumatological patient with an intercurrent EBV infection.

There is no suggestion in the question that there is a parvovirus infection and Felty syndrome would be characterised by a neutropaenia and splenomegaly. Drug-induced pancytopaenia and atypical infections would not commonly show haemophagocytosis in the bone marrow.

Question 92 of 191

A 45-year-old woman is reviewed shortly after being diagnosed with having a pulmonary embolism. Around two weeks ago she was admitted with a severe community-acquired pneumonia which resulted in her being ventilated and admitted to ITU. She responded well to intravenous antibiotics but shortly before discharge became more short-of-breath again. A CTPA was requested which showed a pulmonary embolism. She is started immediately on dalteparin.

What is the most appropriate next step?

A. Stop dalteparin. Start a direct oral anticoagulant for 6 weeks
B. Stop dalteparin. Start a direct oral anticoagulant for 3 months
C. Stop dalteparin. Start a direct oral anticoagulant for 6 months
D. Keep on dalteparin for 6 weeks
E. Keep on dalteparin for 3 months

B. Stop dalteparin. Start a direct oral anticoagulant for 3 months

As this patient developed a pulmonary embolism secondary to something (in this case severe illness with associated immobility) an anticoagulation period of 3 months is generally recommended

Question 93 of 191

A 68-year-old gentleman with known chronic lymphocytic leukaemia (CLL) is reviewed in the Haematology Clinic.

He is normally an active gentleman who enjoys playing golf 3 times per week, but he complains that he has been feeling increasingly fatigued since his last appointment 6 months previously. He states that he has not been able to play golf for several weeks and his wife tells you that he has started napping during the afternoons. His past medical history is otherwise unremarkable and he takes no regular medications.

Examination reveals a tired gentleman with marked axillary and inguinal lymphadenopathy. His abdomen is soft with mild upper abdominal tenderness. A liver edge is palpable 3cm below the costal margin and his spleen is markedly enlarged.

His full blood count today is as follows:

Hb	114 g/l
Platelets	173 * 10 /l9
WBC	30.4 * 10 /l9
Neutrophils	5.8 * 10 /l9
Lymphcytes	23.1 * 10 /l9

His lymphocyte count 2 months ago was 15.3 * 10 /l and a decision to start the patient on fludarabine, cyclophosphamide, and rituximab (FCR) chemotherapy is taken.

Given the proposed treatment strategy, which of the following prophylactic medications is it most important to start?

A. Co-trimoxazole
B. Aciclovir
C. Entecavir
D. Fluconazole
E. Penicillin V

A. Co-trimoxazole

Fludarabine is a purine analogue that prevents DNA synthesis through the inhibition of ribonucleotide reductase and DNA polymerase. It is associated with profound lymphopenia, and significantly increases the risk of opportunistic infections. Patients treated with fludarabine are at particular risk of morbidity and mortality secondary to pneumocystis pneumonia. It is essential that these patients receive regular prophylactic co-trimoxazole.

Purine analogues can lead to herpes simplex, herpes zoster, and cytomegalovirus reactivation and aciclovir is often given as prophylaxis against this. Fluconazole is also frequently given as fungal prophylaxis.

Entecavir is given to patients who are hepatitis B surface antigen (HBsAg) positive. Penicillin V is given to patients with asplenia and is not routinely used in this setting.

Pneumocystis pneumonia is the most severe complication described. Co-trimoxazole is, therefore, the most essential of the medications listed above.

Question 94 of 191

A 77-year-old lady was seen in the haematology outpatient clinic for her monthly follow-up. She has been seen in the haematology clinic for the last six months having been referred by her GP following an anomaly on her blood investigations. Overall she has been feeling well though for the last three weeks she has been feeling increasingly tired. She has noted that her appetite has been reduced for the last three months though she has not lost any weight during this time. In spite of the tiredness she is still able to lead an active life, regularly enjoying long distance rambling and gardening. She denied the presence of other symptoms, including the absence of fever or night sweats. She has a past medical history comprising asthma, hypertension, type 2 diabetes mellitus and hypercholesterolaemia for which she has been prescribed felodipine 5mg M/R OD, atorvastatin 20mg ON, Clenil modulite 2 puffs BD and metformin 500mg TDS.

Examination revealed the presence of a well elderly lady who was independently mobile. Her blood pressure was 122/82 mmHg, heart rate 82bpm and temperature 36.9 Celsius. Examination of her cardiovascular and respiratory systems revealed the presence of normal heart and breath sounds and a JVP of 3cm. Examination of her gastrointestinal and lymphatic systems revealed the presence of a smooth edge 2 fingerbreadths below the left subcostal margin and bilateral small

cervical lymphadenopathy, with the maximum node size less than 1cm. Investigations reveal the following:

Results from clinic three months ago:

Hb	112 g/l
Platelets	242 * 10 /l
WBC	22.6 * 10 /l
Neutrophils	2.1 * 10 /l
Lymphocytes	19.9 * 10 /l
Eosinophils	0.4 * 10 /l
Monocytes	0.2 * 10 /l

Renal and liver function tests were normal.

Blood film: lymphocytosis with atypical lymphocytes Bone marrow aspirate: infiltration with 25% lymphocytes

Peripheral blood flow cytometry: presence of circulating clonal B-lymphocytes expressing CD5, CD19, CD20, CD 23, and an absence of FMC-7 staining

Results from clinic on this occasion:

Hb	106 g/l
Platelets	162 * 10 /l9
WBC	34.2 * 10 /l9
Neutrophils	2.3 * 10 /l9
Lymphocytes	31.5 * 10 /l9
Eosinophils	0.3 * 10 /l9
Monocytes	0.1 * 10 /l9

What is the single next best step management step?

A. Commence prednisolone therapy
B. Commence chlorambucil therapy
C. Commence fludarabine therapy
D. Commence rituximab therapy
E. Observe in clinic and repeat tests in one month

A. Commence prednisolone therapy

This lady has chronic lymphocytic leukaemia (CLL) as manifested by the high lymphocyte count, lymphocyte marrow infiltration and evidence clinically of splenomegaly and lymphadenopathy. 70% of patients are asymptomatic and patients with stage A leukaemia as with this lady [no anaemia or thrombocytopaenia and less than 3 areas of lymphoid enlargement] have a median survival of 8 years. Treatment is therefore not indicated.

Accepted indications for treatment include:

The development of anaemia +/ thrombocytopaenia

Progressive or massive splenomegaly (6cm) +/ lymphadenopathy (10cm)

Progressive lymphocytosis [50% increase in 2 months or doubling time of less than 6 months] Systemic symptoms [fever, night sweats, weight loss >10% in 6 months, extreme fatigue]

Although this lady has been complaining of tiredness, this does not in itself constitute a 'systemic symptom' given the absence of other symptoms and her strong performance status. Her lymphocyte count is rising but does not meet the criteria for treatment at present. Should treatment be indicated, this comprises chemotherapeutic agents such as chlorambucil and fludarabine, immunological agents such as rituximab and steroids in advanced disease.

Question 95 of 191

A 32-year-old patient who is 10 weeks pregnant attends for her antenatal booking clinic. This is her first pregnancy, and she tells you she has been feeling lethargic and has a poor appetite. She has no past medical history of note.

On examination, she looks pale and appears comfortable at rest. Her chest is clear, heart sounds I&II are present and her abdomen is soft non-tender. She has some oedema of both ankles bilaterally. Her blood pressure is 111/75 mmHg and her heart rate is 79/min.

Investigations in clinics are as follows:

Hb	94 g/l
MCV	69 fl
Platelets	168 * 10 /l9
WBC	9.1 * 10 /l9
Hb electrophoresis	positive HbA2
Rhesus	negative
Blood Group	AB
Na +	139 mmol/l
K +	3.9 mmol/l
Urea	7.6 mmol/l
Creatinine	99 µmol/l

What is the single most likely underlying condition?

A. Alpha thalassaemia trait
B. Beta thalassaemia trait
C. Beta thalassemia major
D. Iron deficiency anaemia
E. Sideroblastic anaemia

B. Beta thalassaemia trait

The two main differentials in this patient who has been identified as having a symptomatic microcytic anaemia during pregnancy are iron deficiency anaemia and beta thalassaemia trait. The clue that this is beta thalassaemia trait rather than iron deficiency anaemia is that the MCV is surprisingly low for the level of haemoglobin. In iron deficiency anaemia you would expect the MCV to be much higher for a fairly modest decrease in haemoglobin.

In addition, the Hb electrophoresis shows +ve HbA2 which supports the diagnosis of beta thalassaemia trait. Of course, it would be wise to send off iron studies in this patient to assess for a coexistent iron deficiency anaemia.

Question 96 of 191

A 54-year-old gentleman is reviewed 48 hours after being admitted to his local hospital with neutropaenic sepsis. He measured his temperature at home found it to be 38.2ºC. Because of this, he called the chemotherapy helpline and he was advised to attend the emergency department who promptly admitted him under the care of the medical team.

He has a background of metastatic colorectal cancer and he has had chemotherapy ten days ago. He was started on piperacillin with tazobactam on admission. His temperature settled within 12 hours and investigations, including blood cultures, a chest X-ray, urine testing and a thorough examination did not find any source of infection. His initial neutrophil count was 0.4* 10 /l. Recent blood tests demonstrate a neutrophil count of 0.5* 10 /l. His vital parameters have all been normal since his temperature settled and he has not noticed any symptoms at any point.

What is the most appropriate management plan?

A. Convert patient to oral antibiotics and discharge
B. Keep on IV antibiotics in hospital until full course completed
C. Monitor neutrophil count in hospital and discharge when greater than 1* 109/l
D. Arrange for outpatient IV antibiotics
E. Change piperacillin with tazobactam to meropenem

A. Convert patient to oral antibiotics and discharge

The correct answer is to convert patient to oral antibiotics and discharge. This is a patient who was admitted with a fever and a low neutrophil count and therefore he was managed with empirical IV antibiotics for neutropenic sepsis. Many of these patients will not look or feel significantly unwell and in the majority of them identifying a source of the temperature can be impossible. If neutropenic they should be treated with urgent IV antibiotics and waiting for a full blood count to confirm the low neutrophil count would be inappropriate. At 48 hours the antibiotic should be reviewed and if still febrile then escalation of antibiotics should be considered If they have improved and the temperature has settled then there can be a consideration of an oral switch or if completely well can stop antibiotics altogether. NICE does not recommend keeping patients in hospital whilst waiting for their neutrophil count to improve.

Question 97 of 191

A 67-year-old gentleman presents to the emergency department following a fall. He tripped on the carpet and landed on his back. Following this he has been complaining of lower back pain, but this pain was present prior to his fall and only slightly worsened with the accident. He undergoes a CT scan which unfortunately demonstrates lytic lesions in his lumbar vertebrae. He is suspected of having multiple myeloma. He undergoes blood and urine tests which unfortunately raises further suspicion of the diagnosis. He is due to undergo a bone marrow biopsy.

What investigation prior to the biopsy can give prognostic information?

A. Serum immunofixation
B. B2 microglobulin
C. Serum corrected calcium
D. Protein electrophoresis
E. Urine electrophoresis

B. B2 microglobulin

The correct answer is B2 microglobulin. This is a patient who has unfortunately found to have multiple myeloma and is awaiting further investigation to obtain histology. B2 microglobulin elevation and fall in albumin are associated with a poor prognosis. Serum immunofixation and electrophoresis studies are useful diagnostic tools but are not as useful in terms of prognosis.

Question 98 of 191

A 45-year-old man undergoing chemotherapy mentions to the nurses that he has been feeling unwell for the last two days. He has currently having treatment for metastatic lung cancer. The nurses find that his temperature is 38.2ºC. His other observations are stable. He has been having chemotherapy via a peripherally inserted central catheter (PICC). On examination his line site appears normal, his chest his clear and his abdomen is soft and non-tender.

What is the most appropriate next step in his management plan?

A. Send a full blood count to tests neutrophil levels
B. Remove the PICC line and then observe if temperature settles
C. Remove the PICC line then discharge with IV antibiotics
D. Take blood cultures from the PICC line and peripherally then treat with IV antibiotics
E. Discharge with oral antibiotics

D. Take blood cultures from the PICC line and peripherally then treat with IV antibiotics

The correct answer is to take blood cultures from the PICC line and peripherally then treat with IV antibiotics. This is a patient on chemotherapy who has developed an asymptomatic fever at 38.2ºC. As he may be neutropenic, he should be admitted to hospital and given IV piperacillin and tazobactam, but blood cultures should be taken from any central lines that he has before doing so. If a line has more than one port then blood cultures should be taken from each port. Peripherally inserted lines should only be removed if they are shown to be the cause of infection, which is why blood cultures from these lines are important.

Question 99 of 191

A 50-year-old man is diagnosed with high-grade non-Hodgkin's lymphoma and starts his regimen of R-CHOP chemotherapy. Two days after his chemotherapy he complains of feeling increasingly weak, lethargic and generally unwell. He has developed persistent vomiting and is unable to tolerate oral fluids.

He has a history of recurrent gout but has been unable to tolerate allopurinol.

On examination, he looks unwell and pale. He seemed short of breath with a respiratory rate of 28 per minute. His temperature is 36.5ºC, heart rate 110 bpm, blood pressure 100/60 mmHg.

His heart sounds were normal. His JVP was raised by 4cm lying at 45 degrees in the bed. Examination of the chest revealed fine bibasal inspiratory crepitations. Pitting oedema was present to mid-shins bilaterally.

Abdominal examination was unremarkable.

On neurological examination, there was normal tone and sensation to all limbs. General weakness was noted.

The house officer has taken bloods:

Na+	137 mmol/L
K+	6.2 mmol/L
Urea	15 mmol/L (previously 8)
Creatinine	240 µmol/L (previously 100)
Hb	100 g/L
WBC	10.0x10^9/L
Corrected Calcium	1.95 mmol/L
Phosphate	2.3 mmol/L
Uric acid	640 mmol/L
LFTs	Normal

Chest x-ray shows congested lung fields.

ECG demonstrates tall T waves.

In view of the diagnosis which of the following is most likely to treat the hyperuricaemia? IV calcium gluconate, insulin and

A. dextrose infusion
B. Allopurinol
C. Rasburicase
D. IV fluids
E. IV electrolyte replacement

C. Rasburicase

This man has lymphoma and has presented with physical and biochemical markers suggesting established tumour lysis syndrome. He has severe electrolyte disturbance: hyperkalaemia, hypocalcaemia, hyperphosphataemia. The chest x-ray suggests pulmonary oedema and ECG indicates a degree of cardiac toxicity from the hyperkalaemia. There is also acute renal failure and hyperuricaemia.

The question asks what treatment should be used to treat the underlying condition of tumour lysis syndrome. The answer is rasburicase as he has previously been intolerant of allopurinol in the context of gout prophylaxis. All the other answers are entirely reasonable and are part of the treatment of tumour lysis syndrome but do not answer this question specifically

Question 100 of 191

An 85-year-old male is referred by the anaesthetic registrar after abnormal blood test results were noted during pre-assessment for an elective hip replacement. He is otherwise fit and well, independent with all activities of daily living and continues to drive. His past medical history includes diet controlled type 2 diabetes mellitus and hypertension. On examination, he is alert and well, reports no discomfort, pain, or non-specific malaise. No skin bruises or conjunctival pallor are noted. You note a rubbery, non-tender and firm 3cm lymph node in the left cervical chain and non-tender splenomegaly at 8cm below the costal margin. His chest is clear and normal heart sounds are noted. His blood tests are as follows, with blood tests from his GP 6 months ago in brackets:

Hb	89 (95) g/l
Platelets	78 (76) * 10 /l9
WBC	67 (32) * 10 /l9
Blood film	mature lymphocytes and smudge cells

What is the most appropriate treatment?

A. Monitor and repeat blood count in 6 months
B. Fludarabine, cyclophosphamide and rituximab treatment immediately
C. Delayed chlorambucil treatment in 6 months
D. Platelet transfusion
E. Intravenous immunoglobulin

B. Fludarabine, cyclophosphamide and rituximab treatment immediately

This patient is on the cusp of requiring immediate treatment for chronic lymphocytic leukaemia. The latest guidelines are provided by the British Committee for Standards in Haematology (BCSH) 2012 , recommending immediate treatment to commence if the patient demonstrates signs of progressive marrow failure, massive or symptomatic splenomegaly greater than 6cm below the costal margin, massive or symptomatic nodes greater than 10cm in longest diameter, progressive lymphocytosis with doubling in 6 months, autoimmune thrombocytopaenia or anaemia, or significant constitutional symptoms within the previous 6 months. It is important to note that doubling of lymphocytosis is calculated only if the initial count is greater than 30 x 109/l.

Asymptomatic patients not meeting these criteria do not benefit in long term survival when receiving immediate chlorambucil therapy versus delayed treatment . Regular blood test monitoring is more appropriate for this group of patients. Be aware that despite increased white cell counts, the mature lymphocytes are non-functional and patients are hence at increased risk of infections. Intravenous immunoglobulin may be appropriate if the patient shows features of a significant infection. Similarly, significant progressive marrow failure, demonstrated by symptomatic anaemia or thrombocytopaenia may require replacement. In this case, the patient fits criteria for immediate treatment based on his lymphocytosis doubling time and splenomegaly.

1. British Committee for Standards in Haematology (BCSH) 2012

2. Chemotherapeutic options in chronic lymphocytic leukaemia: a meta-analysis of the randomised

trials. CLL Trialists' Collaborative Group. J Natl Cancer Inst. 1999;91(10):861

Question 101 of 191

A 77-year-old female is referred to the hospitals ambulatory care clinic by her GP after 2 months of increasing generalised malaise and 'lack of energy' over the past two months. She lives with her husband and until 9 weeks ago, continued to drive and go for walks in the countryside with no limitations to her exercise tolerance. Now, she feels 'tired all the time' but denies any problems with her mood. She has no history of psychiatric disorders. Her past medical history includes hypertension (well controlled on ramipril alone), hypercholesterolaemia (well controlled on simvastatin) and chronic lymphocytic leukaemia, diagnosed 3 years ago and not requiring treatment.

On examination, she has warm peripheries with bilateral conjunctival pallor. She is alert and comfortable at rest. Non-tender lymphadenopathy in bilateral cervical chains. Her cardiovascular, respiratory, abdominal and neurological examinations are otherwise unremarkable. Her blood results are as follows:

Hb	37 g/l
MCV	87 fl
Platelets	17 * 10 /l9
WBC	44.0 * 10 /l9
Blood film	myeloblasts with elongated, needle-like cytoplasmic inclusions

Hb	72 g/l
MCV	101 fl
Platelets	70 * 10 /l9
WBC	67.0 * 10 /l9
Neut	4.0 * 10 /l9
WBC	62.0 * 10 /l9
Reticulocytes	14%
Blood film and direct agglutination test	lymphocytosis, smudge cells, reticulocytes, red cell agglutination at physiological temperature

What is the most likely cause of this patient's anaemia?

A. Cold autoimmune haemolytic anaemia 14%
B. Warm autoimmune haemolytic anaemia 75%
C. B12 deficiency anaemia 4%
D. Iron deficiency anaemia 1%
E. Microangiopathic haemolytic anaemia 6

B. Warm autoimmune haemolytic anaemia 75%

The patient presents with a borderline macrocytic/normocytic anaemia with a positive Coombs test at 37 degree temperature, suggesting an autoimmune haemolytic anaemia. The presence of red cell agglutination at a warm temperature suggests the presence of IgG on red blood cells, which in vivo leads to phagocytosis by a granulocyte: this is warm autoimmune haemolytic anaemia. Conversely, red cell agglutination at cold temperatures, typically between 0 and 4 degrees, suggests the presence of IgM and C3 component of complement, most commonly leading to direct cell lysis by the complement system: this is cold autoimmune haemolytic anaemia. A mildly raised or borderline MCV does not necessarily guarantee a macrocytic cause in this context: the high proportion of reticulocytes in the blood demonstrates increased red cell production from marrow, increasing the release of immature cells with higher corpuscular volume than mature red cells, hence the increased MCV in blood assays. Iron deficiency anaemias typically produce microcytic anaemias with target cells. Microangiopathic haemolytic anaemia (MAHA) is caused by the mechanical destruction of red cells in conditions such as thrombotic thrombocytopenic purpura, leading to fragmented red cells (schistocytes) and a typically normocytic picture with associated thrombocytopenia.

Both warm and cold autoimmune haemolytic anaemias can be idiopathic or caused by lymphoproliferative disorders. Warm agglutinins are also more commonly produced by systemic autoimmune conditions such as SLE, rheumatoid arthritis and systemic sclerosis, while cold agglutinins are also classically triggered by mycoplasma and viral infections such as infectious mononucleosis.

Question 102 of 191

A 57-year-old female presents to pre-assessment surgical clinic prior to an elective arthroscopy of her left knee that she injured while playing tennis. She is otherwise asymptomatic, has no other medical history and is a lifelong non-smoker. She drinks 10 units of alcohol per week. Recently, she has experienced hot flushes and irregular periods, which she puts down to undergoing the menopause. Examination of her cardiovascular, respiratory and abdominal systems are unremarkable.

Her blood results are as follows:

Hb	95 g/l
MCV	59 fl
Platelets	389 * 10 /l9
WBC	4.5 * 10 /l9
Red cell distribution width	13% (normal range 11.5-14.5%)
Blood film	anisocytosis, hypochromia, target cells

Which investigation is most likely to reveal the diagnosis?

A. Serum ferritin
B. Total iron binding capacity
C. Serum iron
D. Haemoglobin electrophoresis
E. Bone marrow biopsy

D. Haemoglobin electrophoresis

The patient is asymptomatic with a microcytic anaemia. Note the disproportionately lower MCV compared to the level of haemoglobin and the normal red cell distribution width: the first is a distinctive feature of thalassaemia beta minor (trait) while the second suggests that all red cells made by the marrow of similar haemoglobin quality, such as in an underlying genetic trait such as thalassaemia beta minor. In contrast, iron deficiency normally demonstrates a significant haemoglobin drop by the time MCV is at such a low value while increasing the RDW during early to mid stages of iron deficiency, as some red cells are produced normally while some are profoundly microcytic.

Diagnosis of thalassaemia beta minor is by haemoglobin electrophoresis. However, diagnosis of iron deficiency is reliant on a combination of indicators: serum iron alone is not necessarily diagnostic as it also appears in anaemia of chronic disease. Serum ferritin is perhaps the most reliable indicator of plasma and marrow iron stores but be wary that it may be increased in inflammatory states, hence mask an underlying iron deficiency anaemia. Total iron binding capacity (TIBC) is a measure of transferrin, to which iron is bound to in plasma. Again, it is a useful indicator of iron stores but can be altered by pregnancy or the oral contraceptive pill. An iron deficiency anaemia picture is generally diagnosed by a combination of all three: low serum iron, high TIBC, low ferritin.

Question 1 of 89

A 57-year-old presents with recurrent episodes of bleeding gums, nosebleeds and intermittent haematuria over the past 6 weeks. He works as an accountant, does not have any past medical history but is an active smoker of 20 pack years. He drinks occasional alcohol.

On examination, scabs and dried blood are noted on mucous membranes. No arthritis or cutaneous abnormalities are noted. The nasal bridge is unremarkable. His conjunctiva appeared pale, respiratory, abdominal and cardiovascular examination was unremarkable. His blood tests are as follows:

Na +	147 mmol/l
K +	3.2 mmol/l
Urea	7.8 mmol/l
Creatinine	70 µmol/l

What is the most likely underlying diagnosis?

A. Acute myeloid leukaemia
B. Acute lymphocytic leukaemia
C. Chronic myeloid leukaemia
D. Chronic lymphoid leukaemia
E. Myelofibrosis

A. Acute myeloid leukaemia

The diagnosis is made on blood film description of needle-like elongated cytoplasmic inclusions, known as Auer rods that are pathognomonic of acute myeloid leukaemia, with myeloblasts typical of myeloid leukaemias. Clinical features are frequently typical of cell line abnormalities secondary to marrow infiltration (shortness of breath on exertion, pallor caused by anaemia; bruising or bleeding caused by thrombocytopaenia) and infections secondary to dysfunctional white cells. Diagnosis is confirmed by bone marrow biopsy, with blast cells accounting for greater than 20% of marrow cellularity. Immunophenotyping, cytogenetic and morphological analysis guides subsequent treatment options.

Question 2 of 89

A 60-year-old female with known chronic lymphocytic leukaemia (CLL) presents with coryzal symptoms. Examination findings are unremarkable. Her blood tests are as follows:

8 months previously	Two months previously	Today	
Haemoglobin	113 g/l	108 g/l	106 g/l
White cell count	32.0 *10^9/l	50.0 *10^9/l	58.0 *10^9/l
Neutrophils	7.0 *10^9/l	4.8 *10^9/l	4.0 *10^9/l
Lymphocytes	25.0 *10^9/l	45.0 *10^9/l	54.0 *10^9/l
Platelets	358 *10^9/l	280 *10^9/l	268 *10^9/l

What is the most appropriate treatment option?

A. Chlorambucil
B. Fludarabine and chlorambucil
C. Observation
D. Prednisolone
E. Fludarabine

C. Observation

CLL is typically an indolent disease which is often managed conservatively in the first instance. Indications for treatment are multiple but include constitutional symptoms, bone marrow failure and massive lymphadenopathy. Additionally, a lymphocyte doubling time of less than 6 months is an indication for treatment, but this is not met here. Chlorambucil, fludarabine and high dose corticosteroids are all possible therapeutic options in CLL.

Question 3 of 89

A 21-year-old male is admitted to hospital after his GP identified a pancytopenia following a workup for recent fatigue and weight loss. An hour after arrival he develops a sustained nosebleed. On review, you notice oozing from his cannula sites and non-blanching purpura over his arms and legs.

What is the most likely cause of this patient's presentation?

A. Henoch-Schonlein purpura
B. Aplastic anaemia
C. Acute promyelocytic leukaemia
D. Meningococcal septicaemia
E. Idiopathic thrombocytopenic purpura

C. Acute promyelocytic leukaemia

DIC is a severe complication of acute promyelocytic leukaemia

This patient has developed disseminated intravascular coagulopathy (DIC) secondary to acute promyelocytic leukaemia (APML). This is a potentially life-threatening complication of APML that is important to be aware of in patients presenting with features of leukaemia.

Henoch-Schonlein purpura does not result in a pancytopenia or bleeding. The purpuric rash present is typically on the lower limbs and secondary to a vasculitic process. Aplastic anaemia also presents with a pancytopenia, but the features suggestive of DIC in this patient point against this as the underlying diagnosis. Meningococcal septicaemia is an important differential to consider though would not be expected to result in complete marrow suppression. Finally, idiopathic thrombocytopenic purpura causes an isolated platelet drop.

Question 4 of 89

A 25 year old man is admitted to the acute medical unit and is treated for a left lobar pneumonia. Looking through his past medical history you note he has had 3 previous admissions with similar presentations in the last 5 years. He has no other past medical history of note, apart from what he describes as 'a few' episodes of sinusitis. His blood differential, after treatment of his pneumonia, is normal. His HIV test is also negative.

What is the possible underlying diagnosis?

A. Cystic fibrosis
B. Selective IgA deficiency
C. Wiskott-Aldrich syndrome
D. Acute lymphoblastic leukemia
E. Digeorges syndrome

B. Selective IgA deficiency

Selective immunoglobulin A deficiency:

- The most common primary antibody deficiency
- Affected patients may develop recurrent chest infections, bronchitis, sinusitis or even otitis media.
- Associated with atopic disorders such as asthma and atopic dermatitis.
- Some patients may also be at risk of severe transfusion reactions.

Wiskott-Aldrich syndrome:

- Characterised by a triad of recurrent chest infections/recurrent sinus infections
- Atopic dermatitis
- Thrombocytopenia and platelet dysfunction

- Inherited as an X linked recessive condition.

Question 5 of 89

A 57-year-old man presents to his General Practitioner with persistent headaches and blurred vision. The symptoms have been present over the past six months but have worsened in recent weeks. On close questioning, the patient also reported a feeling of general fatigue and intermittent muscle aches.

One year previously, the patient had been diagnosed with obstructive sleep apnoea secondary to morbid obesity and had been provided with non-invasive ventilation to use at night. However, the patient admitted that he rarely used this equipment due to a dislike for the tight face mask. Despite dietary and lifestyle advice the patient had gained 6 kg over the past year and had a BMI of 41 kg / m².

Neurological examination including fundoscopy was unremarkable. There were no tender or inflamed joints. Blood tests requested by the GP are detailed below.

Haemoglobin	195 g/L
White cell count	7.5 * 10 /l9
Neutrophils	5.7 * 10 /l9
Lymphocytes	0.9 * 10 /l9
Platelets	195 * 10 /l9
Packed cell volume	0.60
Urea	5.9 mmol / L
Creatinine	110 µmol / L
Sodium	135 mmol / L
Potassium	4.1 mmol / L
eGFR	68 ml / min

Review of a full blood count performed 4 months previously was remarkable for previously unnoticed elevated PCV of 0.56. The patient was urgently referred to haematology for further management.

What is the most appropriate treatment for this patient's erythrocytosis?

A. Aspirin
B. Venesection
C. Hydroxyurea
D. Improved compliance with nocturnal non-invasive ventilation
E. Referral to weight management service

B. Venesection

This patient has a secondary erythrocytosis secondary to hypoxia associated with his obstructive sleep apnoea. He is presenting with significant symptoms of hyperviscosity and should be considered to be at significant risk of thrombotic complications.

Evidence from small case series suggests that erythrocytosis secondary to OSA should be treated with venesection in the presence of hyperviscosity symptoms or a PCV > 0.56. A target PCV of 0.50-0.52 has been shown to increase exercise tolerance.

Aspirin is the mainstay of treatment in polycythaemia vera and cytoreductive treatments such as hydroxyurea are used in high-risk cases.

While weight-loss and improved compliance with OSA treatment may improve erythrocytosis these interventions are likely to require significant time prior to yielding any symptomatic benefit.

Question 6 of 89

You are working in haematology. A 72-year-old man has been referred to you by the general surgeons. He recently had a CT scan for weight loss and a two month change in bowel habit. This showed no lesions within the bowel but did demonstrate some mesenteric lymphadenopathy. The largest lymph node was 4 cm in diameter. He is otherwise well and has a background of hypertension and diet controlled diabetes. He subsequently underwent a CT guided biopsy (histology report below).

The lymph node biopsied lacks a mantle zone and is made up of a predominant population of centrocytes with few tangible body macrophages. Immunohistochemistry confirms strong positivity for CD20 as well as CD70a, CD10, BCL2 and BCL6. The proliferation index (Ki-67) is low, no more than 20%.

What is the most likely diagnosis?

A. Nodular sclerosing lymphoma
B. Lymphoplasmacytic lymphoma
C. Follicular lymphoma
D. Adenocarcinoma of the large bowel
E. Diffuse large B cell lymphoma

C. Follicular lymphoma

The correct answer here can be worked out based on the Ki-67 index. This is the proliferation index and is a marker (antigen) of the cancer cells metabolic rate. In high grade lymphomas such as diffuse large B cell lymphoma the Ki-67 will be high (>45%) therefore answer 5 is wrong. In low grade lymphomas such as follicular lymphoma the Ki-67 is low (<40%).

Follicular lymphoma (a form of non Hodgkin's lymphoma) often presents as incidentally found enlarged lymph nodes and may present without the B symptoms often seen in Hodgkin's lymphoma. It can undergo a transformation at any stage into a high grade lymphoma.

Answer 1 is wrong because as it is a form of Hodgkin's lymphoma the histology report would have mentioned the presence of reed-sternberg cells. Answer 2 is Waldenstrom's macroglobulinemia and presents with an IgM gammopathy. Answer 4 is wrong because an adenocarcinoma of the large bowel would have most likely been seen on a CT scan.

Question 7 of 89

A 72-year-old woman presents to the emergency department following a fall. On examination, her right leg is shortened and externally rotated and she is unable to weight bear. A hip X-ray demonstrates a fractured neck of femur which appears pathological. She has no history of cancer. Following a dynamic hip screw insertion, she recovers well. She has blood tests sent for FBC, U&E, LDH, calcium, albumin, uric acid, serum electrophoresis, immunoglobulins and ESR. She has had a chest X-ray which was normal.

What additional tests should be requested to complete the initial investigations of the pathological nature of the fracture?

A. MRI whole spine
B. MRI hip
C. Urinary electrophoresis

D. Trephine biopsy
E. Bone marrow biopsy

C. Urinary electrophoresis

The correct answer answer is urinary eletrophoresis. This is a patient with a pathological fracture and at high risk of myeloma and needs to be fully investigated.

The following tests are needed to complete a myeloma screen:

FBC, ESR, U&E, calcium, albumin, uric acid

Serum protein electrophoresis Urine protein electrophoresis Immunoglobulin levels

Plain X-ray of symptomatic areas

If initial investigations are suggestive of myeloma then bone marrow aspirate and trephine biopsies are indicated, as well as immunofixation of the serum and urine to demonstrate the nature of the paraprotein.

MRI would be contra-indicated due to the recent operation

Question 8 of 89

A 30-year-old gentleman presents with acute lethargy and shortness of breath. He reports cutting his leg on a wooden splinter in a bench with a red area that arose in the leg and spread rapidly. Two days later his leg remained painful and he started to develop chills and was brought into the hospital by concerned family.

On examination, he is pale and clammy with a capillary refill time of 4 seconds. His chest is clear but his breathing is shallow and rapid. His heart rate is 123 beats/min and is regular while his blood pressure is 85/65 mmHg. His leg shows the cut which is now raised and red-purple with a collection of pus under the skin. His cannula site is oozing blood. He receives three litres of intravenous fluid and is started on vancomycin and gentamicin intravenously.

Hb	130 g/l
Platelets	110 * 10 /l9
WBC	34.0 * 10 /l9
CRP	459 ng/ml
PT	18 seconds
APTT	45 seconds
Fibrinogen	0.1
Chest x-ray	patchy bilateral infiltrates
Wound swab	Streptococcus pyogenes

What is the explanation of the coagulation?

A. Disseminated intravascular coagulopathy (DIC)
B. Activation of thrombin by insect venom
C. Haemodilution by intravenous fluids
D. Haemophilia A
E. Von Willebrand disease

A. Disseminated intravascular coagulopathy (DIC)

Deranged coagulation in sepsis -> DIC

This patient is acutely septic and has deranged coagulation. Haemodilution would not deplete fibrinogen severely. Haemophilia A and Von Willebrand disease have a mildly raised APTT only but normal fibrinogen level. There is no history of an insect bite making it unlikely.

DIC is a consequence of severe sepsis where the clotting cascade is activated throughout the body forming small thrombus throughout the body but depleting the clotting factors. Specifically, there is a low fibrinogen. There are rare cases of snake venom activating the clotting cascade in a manner similar to DIC but there is no recall of a snake and is rare in the UK.

Question 9 of 89

A patient has been admitted with left lower limb deep vein thrombosis. He was diagnosed by the FY1 who has efficiently started him on warfarin.

Two days after initiation you are asked to see this gentleman who has developed skin necrosis over his right thigh.

What is the most likely cause of his skin necrosis?

A. Antiphospholipid syndrome
B. Heparin induced thrombocytopenia (HIT) type II
C. Excessive serum Antithrombin III
D. Acquired haemophilia
E. Protein C deficiency

A. Protein C deficiency

Warfarin induced skin necrosis is rare but very severe. Be reminded that warfarin inhibits production of the vitamin K dependent coagulation factors including factors II, VII, IX and X but also the natural anticoagulants, proteins C and S. The first factors affected by warfarin are the anticoagulants, making warfarin temporarily pro-thrombotic during induction, in its initial few days of use and that is why it is advised to start heparin concurrently with Warfarin. Various theories have been postulated as to the cause of this problem one of which is a background of congenital or acquired protein C, S or Antithrombin III deficiency.

Question 10 of 89

A 38-year-old female presents to the emergency department with pain in her right leg, which she attributes to tripping up and landing on the floor when she went hiking with her boyfriend approximately 1 week ago. She does not recall any insect bites but has become increasingly concerned as her leg has become more swollen and tender in the calf region.

She has no significant medical history but has been recovering from an episode of pneumonia for which her GP prescribed her clarithromycin. Her only other medication is the oral contraceptive pill which she started about 1 month ago. She consumes alcohol socially at the weekends and smokes approximately 10-15 cigarettes a day. On physical exam she is afebrile and her other vital signs are within normal limits. She is able to ambulate in the emergency department without significant issues, and there are no obvious skin breakages or significant bony deformities. You note that her left leg is significantly swollen compared to the right leg and there is evidence of pitting oedema and erythema. Passive dorsiflexion of her left foot leads to significant pain.

What is the next best step in the management of this patient?

A. Obtain a doppler ultrasound scan of her left lower limb
B. D-dimer test
C. Start her empirically on oral amoxicillin for probable lower limb cellulitis
D. X-ray her left tibial and fibula
E. Reassure her and advise her to take regular paracetamol

A. Obtain a doppler ultrasound scan of her left lower limb

If a 2-level DVT Wells score is ≥ 2 points then arrange a proximal leg vein ultrasound scan within 4 hours

This patient most likely has a left lower limb deep vein thrombosis. Her important risk factors include the fact that she is taking the oral contraceptive pill and is a smoker, which combined leads to a significantly increased risk for venous thromboembolic complications. In fact, smoking is a contraindication to starting the oral contraceptive pill in females greater than the age of 35 years. In addition, physical exam findings are also concerning for a deep vein thrombosis. Although we could start investigating her by doing a D-dimer test, if this is normal she still has a high pretest probability of deep vein thrombosis and we could therefore not confidently exclude this diagnosis. Consequently, the best way to confirm or refute a diagnosis of deep vein thrombosis, in this case, would be obtaining a Doppler ultrasound scan of her leg. A D-dimer would not necessarily be wrong, but even if this returned as normal she is at considerable risk of deep vein thrombosis and should have a doppler ultrasound scan done anyway. In addition, she is being treated for pneumonia which is likely to confound the results of the D-dimer test anyway.

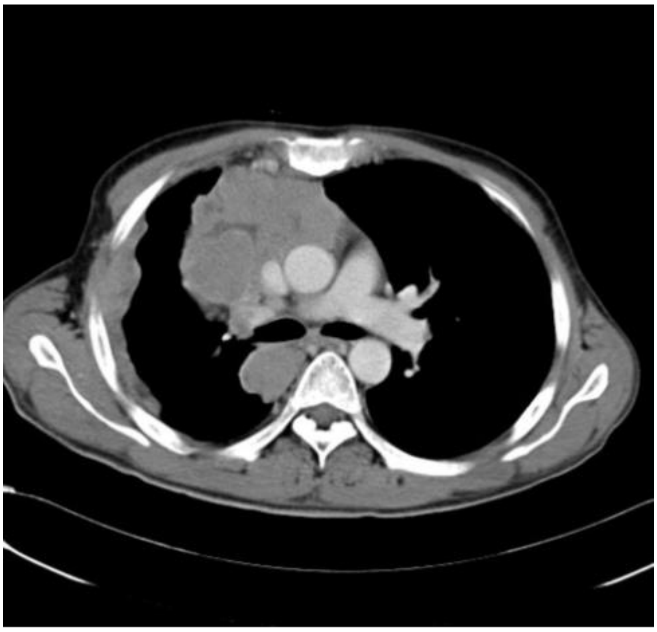
Although she does have swelling and erythema of her lower limb, she is afebrile and her clinical picture does not fit that of cellulitis.

She is ambulant and is unlikely to have sustained any significant bony injuries to her tibia and fibula, particularly as the mechanism of her injury was not very severe. Fractures of the tibia/fibula typically require a more significant mechanism of injury such as motor vehicle accidents.

It would be inappropriate to reassure her and advised her to take regular analgesia, as she is at significant risk of deep vein thrombosis and this needs to be excluded first.

Question 11 of 89

A 51-year-old man is investigated for a chronic cough, chest pain and dyspnoea. He also reports some difficult swallowing bread and meat for the past two weeks. He has no past medical history of note although has smoked for the past 30 years. A CT scan is requested:



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What is the most likely diagnosis? Sarcoidosis

A. Lung cancer
B. Cardiac myxoma
C. Oesophageal cancer
D. Thymoma

A. Thymoma

The CT slice at the bifurcation of the main bronchus shows an invasive thymoma presenting as an anterior mediastinal mass.

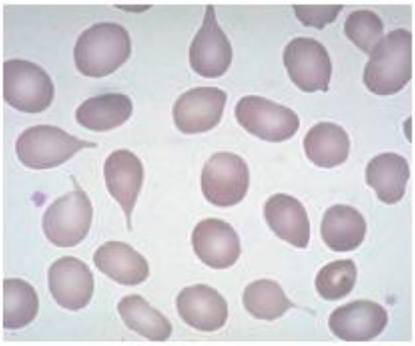
The differential diagnosis of an anterior mediastinal mass includes:

- thymoma
- lymphoma
- thyroid and parathyroid malignancies
- germ cell tumours e.g. teratomas
- thoracic aortic aneurysm

Question 12 of 89

A 77-year-old man is referred to haematology for investigation of anaemia. For several months he has been complaining of fatigue and weight loss. His GP has already arranged upper and lower gastrointestinal (GI) endoscopy which has been reported as normal.

His blood film is shown below:



What is the most likely diagnosis?

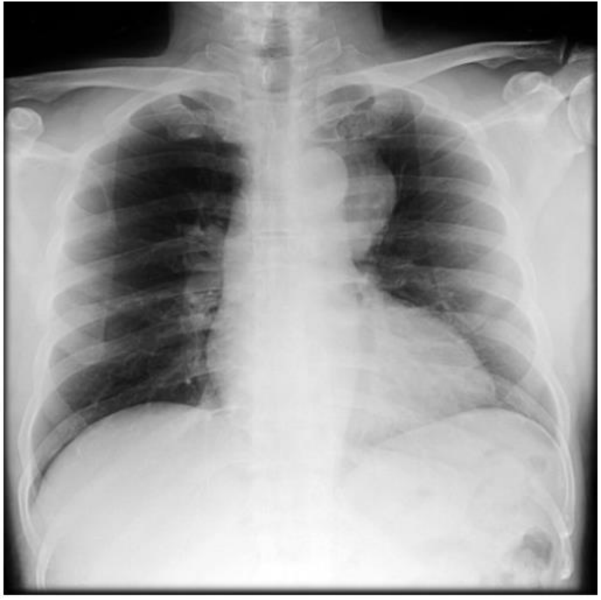
A. Chronic lymphocytic leukaemia
B. Myelofibrosis
C. Iron deficiency anaemia (bleeding from non-GI source)
D. Hyposplenism
E. Autoimmune hemolytic anaemia

B. Myelofibrosis

The blood film shows the typical 'tear-drop' poikilocytes of myelofibrosis.

Question 13 of 89

A 47-year-old man is referred to the respiratory clinic with a 3 month history of cough associated with retrosternal chest pain. A chest x-ray requested by his GP has been reported as abnormal. The chest x-ray is shown below



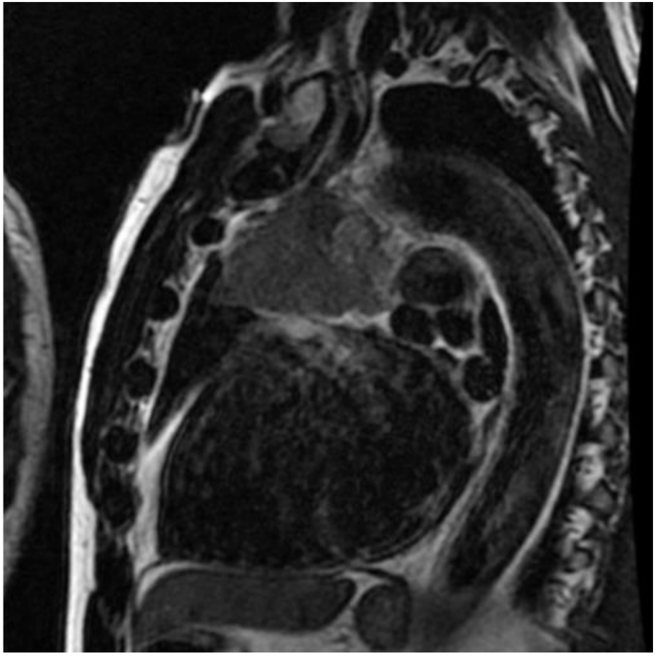
What is the most likely diagnosis?

A. Oesophageal cancer
B. Cardiac myxoma
C. Thymoma
D. Lung cancer
E. Sarcoidosis

C. Thymoma

The chest x-ray demonstrates a soft tissue density projecting to the left of the mediastinum. The distal part of the arch and proximal descending aorta can be seen as separate suggesting it is located anterior to these structures.

A MRI scan from the same patient is shown below:



MRI confirms the presence of an anterior mediastinal mass without convincing macroscopic invasion

Question 14 of 89

A 62-year-old woman presents to the haematology clinic. She has had lower back pain, which has been progressive in nature. She has also been noted to develop unexplained anaemia. Her initial protein electrophoresis and serum-free light chain assays raised suspicion of multiple myeloma. She has a past medical history of transient ischaemic attacks and hypertension. She takes clopidogrel, amlodipine and ramipril.

What imaging should be offered to further assess her?

A. Lumbosacral X-rays
B. Skeletal survey
C. Bone marrow ultrasound
D. Whole body MRI
E. PET scan

D. Whole body MRI

The correct answer is whole body MRI. NICE advises that all patients suspected to have a diagnosis of myeloma should be offered whole body MRI as first-line imaging, and only consider whole body CT if the patient declines MRI or is unable to have it. Skeletal survey should only be considered if CT and MRI are both not possible. Fluorodeoxyglucose positron emission tomography CT (FDG PET CT) can be considered once a diagnosis is confirmed

Question 15 of 89

A 69-year-old gentleman presented for routine follow-up in the oncology clinic. He has metastatic poorly differentiated adenocarcinoma of unknown primary. He commenced palliative chemotherapy with oxaliplatin and fluorouracil two months ago. The most recent CT scan demonstrated stable disease.

Ten days ago he was admitted to the local emergency department with fever and diagnosed with neutropenic sepsis, of which the cause was not clear. He was admitted for IV Tazocin for five days then discharged with co-amoxiclav and filgrastim (G-CSF). He currently feels well. On examination, there are no abnormalities.

Observations:

Saturations	95%
Respiratory rate	14/min
Blood pressure	152/83mmHg
Heart rate	69/min
Temperature	37.3°C

Blood tests:

14 days ago	Today	
Hb	135g/l	124g/l
Platelets	322* 10 /l 9	285* 10 /l9
WBC	0.2* 10 /l 9	23.6* 10 /l9

What is the most appropriate course of action?

A. Restart oral co-amoxiclav
B. Arrange admission for IV tazocin
C. Arrange admission for IV meropenem
D. Start prednisolone
E. Stop filgrastim (G-CSF)

A. Stop filgrastim (G-CSF)

The correct answer is to stop filgrastim. Filgrastim stimulates a white cell count which can increase far above the normal range, and the white cell count will return to normal once it is stopped. The key here is that the patient is clinically well, and further antibiotics are unnecessary

Question 16 of 89

A 67-year-old female presents with 4-month history of increasing lethargy and malaise. She has no past medical history and travels widely, last visiting the Middle East one week prior to this admission, returning with a respiratory tract infection that appears to be resolving. She is a lifelong non-smoker and does not drink alcohol to excess. Over the past two weeks, she reports increasing bilateral persistent headache associated with binocular visual blurring. In addition, she describes a non-specific abdominal discomfort without any changes in bowel habit.

On examination, you note bilateral axillary lymphadenopathy and conjunctival pallor. Cardiovascular and respiratory system examinations were unremarkable. Neurological examination is unremarkable. Fundoscopy reveals dilated tortuous retinal veins. Abdominal examination reveals hepatosplenomegaly. Lastly, you note areas of purpura around her left anterior shin and her right upper arm. A chest radiograph is unremarkable.

Her blood results are as follows:

Hb	87 g/l
MCV	79 fl
Platelets	190 * 10 /l9
WBC	3.4 * 10 /l9
Na +	142 mmol/l
K +	4.5 mmol/l
Urea	7.6 mmol/l
Creatinine	89 µmol/l
Adj Calcium	2.47 mmol/l
Phosphate	1.34 mmol/l
LDH	1890 (normal range 140-280 units/L)
Serum electrophoresis	IgM paraprotein band at 5.4 g/L

A bone marrow biopsy demonstrates 14% infiltration of lymphoplasmacytic cells

What is the most likely diagnosis?

A. Waldenstrom's macroglobulinaemia
B. Multiple myeloma
C. Monoclonal gammopathy of unknown significance (MGUS)
D. Chronic lymphocytic leukaemia (CLL)
E. Upper respiratory tract infection (URTI)

A. Waldenstrom's macroglobulinaemia

There is an enormous IgM paraprotein band on serum electrophoresis, immediately suggesting the differentials to narrow to Waldenstrom's macroglobulinaemia, multiple myeloma and MGUS. The patient is symptomatic, has bone marrow involvement of greater than 10% with IgM band greater than 3g/L, ruling out MGUS. The key to this diagnosis is differentiating between myeloma and Waldenstrom's: it is particularly rare (but not impossible) for an IgM-secreting plasma cell clone in myeloma but this accounts for only 0.5% of all multiple myelomas. Secondly, clinically, the patient demonstrates signs of hyperviscosity syndrome and splenomegaly, both of which are much more common in Waldenstrom's than myeloma. Thirdly, there is a lack of bone symptoms and renal involvement with normal serum calcium: bone lesions are significantly more common in multiple myeloma. Fourthly, fundoscopic tortuous and dilated veins are classical in hyperviscous patient with Waldenstrom's macroglobulinaemia. The diagnosis is clinched on bone marrow biopsy, demonstrating lymphoplasmacytic cells instead of plasma cells, confirmed by cell immunophenotyping.

CLL should not result in a paraprotein band, URTI may result in a rise in IgA but should not result in bone marrow involvement.

Question 17 of 89

A 34-year-old male presents with a 4 day history of bloody diarrhoea and vomiting, fevers and associated with occasional abdominal cramps. He reports no other symptoms. He reports no previous history of gastrointestinal disease; there is no family history of inflammatory bowel disease. He has no past medical history except for a left knee arthroscopy following an injury playing football 7 months ago. He is a lifelong non-smoker, drinks 14 units of alcohol a month, has not travelled abroad in the past year and last ate outside of his home a week ago during a barbecue at his brother's house.

On examination, he appears dehydrated. There is mild generalised abdominal tenderness with increased bowel sounds. Respiratory and cardiovascular examinations were unremarkable. His blood tests are as follows:

Hb	92 g/l
MCV	90fl
Platelets	49 * 10 /l9
WBC	14.2 * 10 /l9
Neutrophils	12.8 * 10 /l9
Blood film	schistocytes, reticulocytosis

Direct antiglobulin test	Negative
Urea	14.9 mmol/l
Creatinine	159 µmol/l
CRP	82 mg/l

What is the cause of this patient's blood abnormalities?

A. Microangiopathic haemolytic anaemia
B. Cold autoimmune haemolytic anaemia
C. Warm autoimmune haemolytic anaemia
D. Iron deficiency anaemia
E. Acute myeloid leukaemia

A. Microangiopathic haemolytic anaemia

The first recognition in this patient is the underlying syndrome: blood film with fragment red cells (schistocytes), thrombocytopaenia acute kidney injury, pyrexia and bloody diarrhoea, should strongly suggest haemolytic uraemia syndrome-thrombotic thrombocytopaenia purpura spectrum (HUS-TTP) in the absence of alternative unifying causes. In this case, gastrointestinal infection by Campylobacter from the recent barbecue, producing Shiga toxins and resulting in endothelial damage, is a strong possible culprit. The patient has a normocytic anaemia with red cell fragmentation and increased reticulocyte production, suggestive of rapid mechanical destruction and the bone marrow releasing immature red cells in (unsuccessful) compensation. This represents microangiopathic haemolytic anaemia (MAHA) in the context of HUS-TTP.

Both cold and warm autoimmune haemolytic anaemias should produce a positive direct antiglobulin test (Coombs): addition of a Coombs reagent containing antihuman globulin should agglutinate red cells IgM and IgG antibodies bound to in cold and warm autoimmune haemolytic anaemias respectively. Iron deficiency anaemia typically produces microcytic anaemias with target cells; there are no blast cells suggestive of leukaemia.

Question 18 of 89

A 23-year-old medical student went to Uganda on his elective but has had to return to the UK early due to illness. He had been careful to take malaria prophylaxis and slept under a mosquito net. He was using primaquine due to previous intolerable side effects with doxycycline. He was complaining of central abdominal pain and had noticed jaundiced sclera in the few days prior to returning to the UK. There is no relevant past medical history and he takes no regular medication. He is a non-smoker and drinks 2-4 units of alcohol weekly.

Observations show a blood pressure of 110/73 mmHg and heart rate of 98 beats per minute. He is apyrexial, has a respiratory rate of 16 per minute and oxygen saturations of 94% on room air.

On examination, he is pale and jaundiced with yellow sclera. There is no cyanosis. His chest sounds clear and heart sounds are normal with nil added. The abdomen is soft, generally tender but with no guarding or peritonism. Bowel sounds are normal.

Bloods show the following:

Haemoglobin	86 g/L	Sodium	139 mmol/L
Platelets	188 x10^9/L	Potassium	3.6 mmol/L
White cell count	11.0 x10^9/L	Urea	3.0 mmol/L
Neutrophils	8.5 x10^9/L	Creatinine	62 micromol/L
Reticulocytes	11%	Albumin	34 g/L
CRP	7 mg/L	Bilirubin	67 micromol/L
ALT	21 iu/L		
Alkaline Phosphatase	40 iu/L		

Peripheral blood film:

Heinz bodies seen with methyl violet staining.

Bite and blister cells also present.

What is the most likely diagnosis?

A. G6PD deficiency
B. Plasmodium falciparum infection
C. Mycoplasma pneumoniae
D. Hereditary spherocytosis
E. Lead poisoning

A. G6PD deficiency

This student has acute haemolytic anaemia (anaemia, reticulocytosis and raised bilirubin with normal ALT). Answers A, C, D and E are all causes of haemolytic anaemia.

The blood film is the main diagnostic tool to differentiate between these answers. Heinz bodies, bite and blister cells are all seen in acute haemolysis associated with G6PD deficiency. The acute haemolysis has been triggered by antimalarial treatment (usually primaquine).

A blood film of hereditary spherocytosis shows spherocytes and reticulocytes. In lead poisoning, there should be dimorphic cells with ring granules on Perl's staining.

Plasmodium falciparum is a common form of malaria that multiplies in the liver. Infection with this can cause anaemia with abnormal liver function tests (LFTs). Persons with G6PD deficiency are somewhat protected from infection with *Plasmodium falciparum* and *Plasmodium vivax* malaria.

G6PD deficiency is X-linked and is more prevalent in Mediterranean populations.

Question 19 of 89

A 51-year-old man presents with dysuria and a low grade fever. He is prescribed a course of Nitrofurantoin for a suspected urinary tract infection. He has a history of ischaemic heart disease for which he takes aspirin and atorvastatin. The following day, he notices that his urine has become very dark and he feels breathless and more unwell.

Bloods show:

Hb	78 g/L
WCC	14.1 x 10^9/L
Neutrophils	13 x 10^9/L
Platelets	280 x 10^9/L
Bilirubin	87 mg/dL
ALT	45 IU/L
Alkaline phosphatase	73 IU/L
Urea	5 mmol/L
Creatinine	80 µmol/L

Blood film microscopy comments on the presence of Heinz bodies.

What is the underlying diagnosis?

A. Hereditary spherocytosis
B. Paroxysmal nocturnal haemoglobinuria
C. Haemolytic uraemic syndrome
D. Urinary sepsis secondary to E. coli
E. Glucose-6-phosphate dehydrogenase deficiency (G6PD)

A. Glucose-6-phosphate dehydrogenase deficiency (G6PD)

The blood tests reveal anaemia with a bilirubin rise, which indicates haemotlyic anaemia. Heinz bodies are red cell inclusions consisting of denatured Hb; they are a feature of oxidative stress. Nitrofurantoin is one of many drugs that can cause haemolytic crisis in G6PD deficiency. Heinz bodies would not usually be seen on the blood films of the other conditions.

Question 20 of 89

A 26 year old female who is 14 days post-partum (spontaneous vaginal delivery) presents to your medical admissions unit with her partner who is worried that she is confused. She is unable to provide any history but her partner tells you that she has gradually gotten worse over the past few days. Her observations are as follows: temperature 38ºC, pulse 90/min, blood pressure 154/88 mmHg, respiratory rate 16/min, sats 99% on room air.

On examination, she looks pale. Her chest is clear and abdomen is soft, non-tender. She has multiple bruises on her arms. Her blood results are pending.

What is the most likely diagnosis?

A. Haemolytic uraemic syndrome
B. Thrombotic thrombocytopenic purpura
C. Idiopathic thrombocytopenic purpura
D. Retained products of placenta
E. Disseminated intravascular coagulation (DIC)

B. Thrombotic thrombocytopenic purpura

Thrombotic thrombocytopenic purpura is associated with fever, anaemia, thrombocytopenia, renal failure and confusion. Pregnancy and the post-partum state account for about 20% of cases seen.

Haemolytic uraemic syndrome is not associated with fever and confusion. Retained products of placental is more unlikely as her abdomen is soft, non-tender. DIC would be a differential diagnosis but is not the most likely diagnosis here. Idiopathic thrombocytopenic purpura would not cause confusion.

Question 21 of 89

A 52-year-old female with acute lymphoblastic leukaemia in on her third cycle of chemotherapy. She is admitted to the ward after developing a temperature of 38.7ºC before her fourth cycle. She feels well in herself but has ongoing trouble with diarrhoea and mucositis. At present, her stools are type four on the Bristol stool chart and her mouth ulcers are being treated with a lidocaine/nystatin topical solution. She denies any cough, sore throat or urinary symptoms.

On examination, her abdomen is soft and non-tender with normal bowel sounds. Her chest is clear with air entry heard throughout. She has no murmurs, joint effusions or areas of cellulitis. Her mouth contains multiple ulcers with areas of straw colored exudate overlying them.

Hb	110 g/l
Platelets	60 * 10 /l9
WBC	1.1 * 10 /l9
Neuts	0.5 * 10 /l9
Blood culture (1st)	Staphylococcus epidermidis
Blood culture (2nd)	no growth
Chest X-ray	clear lung fields, normal cardiac contour
Nasopharyngeal PCR	negative
Urine dip	negative for leucocytes and nitrites

Which investigation is most likely to elicit the cause of the fever?

A. Sputum culture
B. Urine culture
C. Blood culture
D. Stool sample
E. Swab mouth ulcer

A. Swab mouth ulcer

Mucositis can be a source of neutropenic sepsis

This patient has neutropenic sepsis of unknown source. In neutropenic patients, almost any site

can be the source. From the history there is diarrhoea but her stools are currently normal. There

are no urinary symptoms and the dipstick is normal making the urine unlikely to culture an

organism. Ideally, three blood cultures should be completed in pyrexia of unknown origin so this is

a useful suggestion. Sputum culture is impossible if there is no productive cough and would need

to be induced by bronchoscopy.

Importantly, we are told there are mouth ulcers and this represents a break in the protective lining

through which oral commensal bacteria can enter the blood stream and trigger sepsis. Therefore,

a swab should be taken for bacterial culture and for viral PCR from the ulcer site.

Question 22 of 89

A 45-year-old lady presents to the Emergency Department progressive shortness of breath for the last three days. It is worse on activity but is not associated with any cough or wheeze. She has a past medical history of asthma and HIV, for which takes antiretroviral medication regularly. At her last clinic appointment two weeks ago, she was found to have oral candida and so was given a 2 week course of nystatin and started on dapsone for prophylaxis of pneumocystis jirovecci pneumonia. She is a non-smoker.

On examination, her lips and nail beds have a bluish tinge and she is visibly breathless. Her respiratory rate is 26 per minute and on pulse oximetry her saturations are 91% on air both at rest and on exercise. Her temperature is 36.5ºC and she has not felt feverish. On auscultation she has vesicular breath sounds with minimal wheeze and normal heart sounds with no murmurs. She has no ankle oedema and JVP is not raised. There is no evidence of oral candidiasis and no lymphadenopathy. Her calves are soft and non-tender.

A chest x-ray shows clear lung fields with no focal consolidation or lymphadenopathy. ECG is sinus rhythm at 90 beats per minute with normal complexes throughout.

Arterial blood gas on air:

pH	7.51
PaO2	13.7 kPa
PaCO2	3.34 kPa
HCO3-	22.1 mmol/l
BE	-3.3 mmol/l
sO2	97%
Hb	113 g/l
Na+	143 mmol/l
K+	3.7 mmol/l
Glu	5.2 mmol/l
Lac	1.9 mmol/l

What is the most likely diagnosis?

A. Acute asthma
B. Carbon monoxide poisoning
C. Methemoglobinemia
D. Pneumocystis jirovecii pneumonia
E. Pulmonary embolus

C. Methemoglobinemia

This lady has shortness of breath with low saturations on pulse oximetry but normal PaO2 and saturations on arterial blood gas. This combined with a bluish discolouration and normal chest examination point towards a diagnosis of methemoglobinaemia, a known side effect of the dapsone on which she has been started.

In exacerbation of asthma, one would expect wheeze and a low PaO2. Carbon monoxide poisoning typically results in cherry red appearance. This lady does not have any history of suggest pulmonary embolus and one would again expect a low PaO2, associated with a tachycardia. Pneumocystis pneumonia would be unlikely on prophylaxis, and there may be x-ray changes and drop in saturations on exercise.

Question 23 of 89

A 44 year old female patient is admitted to the oncology ward to undergo chemotherapy for Diffuse Large B-Cell lymphoma stage IVb. She had originlly presented to her GP with intermittent abdominal bloating and constipation and occasional shortness of breath. She also reported having to often get up in the middle of the night to change the bed clothes due to drenching sweats. Her GP had been concerned by these symptoms and had organised an immediate chest X-ray to be undertaken at the nearby hospital.

Chest X-ray

large mediastinal mass with clear lung fields.

On receiving this report the GP arranged an urgent appointment with a local haematologist as he suspected that the patient was suffering from lymphoma. She was seen only 4 days later. Given the clinical history the team at the hospital arranged some urgent investigations.

Hb	9.5 g/dl
Platelets	140 * 10 /l9
WBC	36.5 * 10 /l9
Lactate Dehydrogenase	2540IU/l

CT-guided Lymph node biopsy

cells are large, with prominent nucleoli and abundant cytoplasm and many mitoses expressing

CD19 and CD20 markers

PET scan

large extra-nodal disease bulks most notable in the ileo-caecal area and in the mediastinum. Overall bulky disease in keeping with the diagnosis of advanced stage lymphoma.

The patient is admitted to receive cycle one of R-CHOP chemotherapy under close monitoring.

What electrolyte abnormalities would suggest tumour lysis syndrome?

A. High Potassium, high Calcium, low Phosphate
B. Low Potassium, high Calcium, low Phosphate
C. High Potassium, low Calcium, high Phosphate
D. Low Potassium, low Calcium, high Phosphate
E. Low Potassium, low Calcium, low Phosphate

C. High Potassium, low Calcium, high Phosphate

Question 24 of 89

A previously healthy 68-year-old male patient is referred by his GP to the general medical clinic. He has mixed symptoms of pain in multiple areas, including his upper arm, neck and legs. This has worsened over a period of months and seems not to have been helped with multiple analgesics, including paracetamol, codeine phosphate and ibuprofen. In this time the patient has also become increasingly short of breath.

A full work up is undertaken and the results are shown -

Hb	9.4 g/dl
Platelets	174 * 10 /l9
WBC	8.4 * 10 /l9
Na +	136 mmol/l
K +	4.7 mmol/l
Urea	8.4 mmol/l
Creatinine	125 µmol/l
Corrected calcium	2.9mmol/l
Albumin	34g/L

Kappa light chains detected Lambda light chains absent

IgG elevated IgA normal IgM normal

Urine Bence Jones proteins detected

Skeletal survey multiple osteolytic lesions seen throughtout axial skeleton including on the right humerus, thoracic spine and both femurs.

Given the presumed diagnosis, what tests are most relevant for staging of the condition?

A. Calcium, x-rays and creatinine
B. Free light chain level
C. Renal function
D. B2-microglobulin and albumin
E. Haemaglobin and albumin

D. B2-microglobulin and albumin

The diagnosis here is multiple myeloma. Myeloma is a plasma cell dyscrasia that is more common in men than women. It is most common in elderly populations. The affects of myeloma can easily be remembered by the tetrad of high calcium (c), renal disease (r), anaemia (a) and bone pain (b) (crab). The original staging system was known as the Durie-Salmon Staging system which was developed in 1975. It is still commonly used as a supplementary measure but it's role in staging has been replaced by the International Staging System (ISS) that was implemented by the International Myeloma Working Group in 2005.

Question 25 of 89

A 70 year-old woman presents with severe back pain which has been worsening over the last month. Prior to this she has never suffered from back pain. She has been lethargic, and her husband notes some intermittent confusion. A systemic enquiry reveals long standing exertional breathlessness, and constipation. She has no other bowel or bladder disturbance.

Her background includes chronic obstructive pulmonary disease, which is managed by her GP. She gave up smoking two years ago. Her well woman check up 12 months ago was entirely normal, aside from a slightly raised cholesterol which is being management with diet.

On examination, she has a normal gait. There is some mild tenderness over L3/L4 vertebra with no lower limb neurological deficit. Cardiorespiratory examination reveals an ejection systolic murmur, with a normal second heart sound.

Hb	90 g/l	Na +	135 mmol/l	Bilirubin	5 µmol/l
Platelets	200* 10 /l 9	K +	5.5 mmol/l	ALP	101 u/l
WBC	10 * 10 /l 9	Urea	15 mmol/l	ALT	40 u/l
Neuts	8 * 10 /l 9	Creatinine	230 µmol/l	corrected calcium	2.7 u/l
ESR	40 mm/hr				

What is the most likely diagnosis?

A. Multiple myeloma
B. Monoclonal gammopathy of undetermined significance
C. Non-Hodgkin's, lymphoma
D. Paget's disease
E. Renal cell carcinoma with spinal metastases

A. Multiple myeloma

Multiple myeloma is malignant proliferation of plasma cells, producing a monoclonal protein detected in blood and/or urine; this causes organ or tissue damage. The median age of presentation is 70 years old.

Presenting clinical features include symptoms of:

Impaired renal function- from light chain deposition from plasma cells, other causes include amyloid deposition, dehydration, hypercalcaemia, hyperviscosity, and nephrotoxic drugs Anaemia

Hypercalcaemia- myeloma cells cause an increased production of osteoclast activating factors and cytokines that inhibit osteoblast differentiation

Recurrent infections- decreased humoral immunity

Hyperviscosity symptoms (headaches, epistaxis, blurred vision, and confusion)- high paraprotein levels

Bone pain

Monoclonal gammopathy of undetermined significance (MGUS) is an asymptomatic condition thought to precede multiple myeloma.

Question 26 of 89

Matthew is an 18-year-old man presenting with diarrhoea. The diarrhoea has been ongoing for the past 7 days and his stool is described as a type 6-7 stool on the Bristol stool charts. He did not note any blood or mucous accompanying his diarrhoea. There is accompanying crampy abdominal pains and he noted a fever of 38.2ºC yesterday. He did not note any accompanying weight loss but has been feeling tired over this period.

The abdominal examination was unremarkable. He is not sexually active at present.

He has been hospitalized approximately 5-6 times in the last 3 years with recurrent infections. From his previous medical notes, his last admission related to a chest infection. He had a dry cough and shortness of breath during this time. Oxygen saturation measured on admission was 96% but quickly decreased to 89% on exertion with bilateral opacification noted on chest X-rays.

Stool cultures had been taken and on Ziehl-Neelsen staining, red oocysts are visible within the stool culture. Blood tests taken reveals:

Hb	150 g/L	Male: (135-180)Female: (115 - 160)
Platelets	250 * 10 /L 9	(150 - 400)
WBC	4.5 * 10 /L 9	(4.0 - 11.0)
Neuts	1.1 * 10 /L 9	(2.0 - 7.0)
Lymphs	2.5 * 10 /L 9	(1.0 - 3.5)
Mono	0.6 * 10 /L 9	(0.2 - 0.8)
Eosin	0.3 * 10 /L 9	(0.0 - 0.4)

Immunoglobulin studies were carried out and this revealed reduced levels of IgG and IgA but IgM levels were normal.

What is the most likely unifying diagnosis for his symptoms?

A. Brunton's agammaglobulinemia
B. Chediak-Higashi syndrome
C. Selective IgA deficiency
D. Hyper IgM syndrome
E. Wiskott-Aldrich syndrome

D. Hyper IgM syndrome

Hyper IgM syndrome characteristically presents with infections e.g. *Pneumocystis* pneumonia, hepatitis, diarrhoea

Hyper IgM syndrome is a rare X-linked inherited immunodeficiency that presents with multiple infections throughout their life and typically presents in infancy. It is common to see opportunistic infections as described above as cryptosporidiosis and *Pneumocystis jirovecii* pneumonia. Definitive testing requires flow cytometry to identify the lack of expression of CD40 on T-cells or molecular genetic testing to identify a pathogenic variant on the CD40LG gene. However, findings of neutropenia and reduced IgG and IgA with normal or elevated IgM levels in patients presenting with recurrent, chronic infection highly suggests this diagnosis.

Chediak-Higashi syndrome presents with recurrent infections but this tends to be caused by more common pathogens with recurrent *Staph aureus* being the most common. It is also accompanied by other findings including albinism, photophobia and peripheral neuropathy during teenage years.

Selective IgA deficiency patients tend to be asymptomatic. However, patients can present with recurrent sinopulmonary infections and gastrointestinal infections. It is unlikely in this case due to low IgG levels as selective IgA deficiency will typically have normal IgG levels.

Brunton's agammaglobulinemia will also present with recurrent bacterial infections but investigations will reveal a generalized reduction in all immunoglobulin levels. Definitive tests include genetic testing for mutations within the BTK gene.

Wiskott-Aldrich syndrome typically presents with symptoms bruising and petechiae with eczema during the 1st month of life. Investigations will show thrombocytopenia and immunoglobulin tests will show reduced IgM but elevated IgA and IgE with normal IgG levels. Note that these patients are at increased risk of autoimmune conditions and haematology malignancy.

Question 27 of 89

A 54-year-old woman presents to the emergency department. She has noticed a sore throat over the last 24 hours and checked her temperature and found it to be 38.2ºC. Her other observations are all normal. She has had no other symptoms and specifically denies coughing, chest pain, dysuria and diarrhoea. She is currently undergoing chemotherapy for breast cancer having last had treatment six days ago.

What is the most appropriate treatment?

A. Confirm neutropenia before treatment
B. IV co-amoxiclav and oral clarithromycin
C. Oral metronidazole
D. IV piperacillin with tazobactam
E. IV gentamicin

D. IV piperacillin with tazobactam

Piperacillin with tazobactam (Tazocin) is the empirical antibiotic of choice for neutropenic sepsis

The correct answer is IV piperacillin with tazobactam. This is a patient who is likely to have a low neutrophil count as she has recently had chemotherapy. Such patients should be treated as neutropenic sepsis if there is a temperature above 38ºC or any other sign of sepsis, and need immediate IV piperacillin with tazobactam. Some units use vancomycin as well at this point. This should be given before confirming the neutropenia as that would cause an unnecessary delay. IV gentamicin would be generally used for pyelonephritis, whilst IV co-amoxiclav and oral clarithromycin would be used for a severe community acquired pneumonia.

Question 28 of 89

A 62-year-old man had a routine set of blood tests performed by his General Practitioner. These demonstrated an erythrocytosis (Packed Cell Volume 0.56) but no other abnormality. Further questioning by the GP found that the patient had no symptoms of hyperviscosity, was a nonsmoker with no symptoms of daytime somnolence and took no regular medications. Past medical history included only a left knee hemiarthroplasty performed due to osteoarthritis.

Two weeks after the initial blood test, the patient's bloods were repeated and showed the persistence of the erythrocytosis. A referral to haematology clinic was made for further investigation. Details of further investigations arranged through haematology clinic are listed below.

Haemoglobin	188 g / L
White cell count	6.7 * 10 /l
Neutrophils	3.2 * 10 /l
Lymphocytes	2.1 * 10 /l
Monocytes	0.8 * 10 /l
Eosinophils	0.3 * 10 /l
Basophils	0.3 * 10 /l
Platelets	202 * 10 /l
Packed cell volume	0.59
Urea	4.5 mmol / L
Creatinine	97 micromol / L
Sodium	140 mmol / L
Potassium	3.9 mmol / L
eGFR	85 ml / min
Ferritin	80 ng / ml
Albumin	38 g / L
Alkaline phosphatase	89 U / L
ALT	25 U / L
Bilirubin	20 micromol / L
JAK 2 V617F mutation	Negative
Serum erythropoietin	0 U / L (reference 0-19)

Blood film: no abnormality detected; no features of myeloproliferative disease Abdominal ultrasound: liver, hepatic duct system and gallbladder unremarkable; mild-moderate splenomegaly; kidneys and renal tract unremarkable

What is the most appropriate next investigation?

A. JAK2 exon 12 mutation testing
B. Bone marrow aspiration and trephine biopsy
C. Measurement of red cell mass
D. CT brain
E. Erythropoietin receptor gene analysis

A. JAK2 exon 12 mutation testing

Erythrocytosis is defined as a haemoglobin > 18.5 g / dL or PCV > 0.52 (male) / 0.48 (female). Patients with a persistent erythrocytosis without a clear cause (i.e. chronic hypoxia or drug causes) should be referred to haematology for further investigation. An urgent referral should be made if symptoms of hyperviscosity or polycythaemia (raised PCV, white cells and platelets) are present.

More than 95 % of individuals with polycythaemia vera test positive for the JAK 2 V617F mutation. Other baseline investigations include a blood film to exclude myeloproliferative disease, renal and liver profiles and serum ferritin (as iron deficiency can mask the degree of erythrocytosis). Abdominal ultrasound is performed in patients with high suspicion for polycythaemia vera as this condition is associated with radiographical splenomegaly in two-thirds of cases.

A low serum erythropoietin is suggestive of primary bone marrow disease even in the absence of JAK 2 mutation and should prompt testing for the rarer exon 12 mutation of JAK 2. This test should be performed before the more invasive bone marrow biopsy.

Raised serum erythropoietin should prompt investigation for an exogenous source, for example, CT brain to look for a cerebellar haemangioma or meningioma.

Question 29 of 89

A 78-year-old man presents to the acute medical unit with a 3-day history of lower limb weakness. He has a 6-month history of progressive back pain. He denies any bowel or bladder dysfunction, and saddle anaesthesia.

Blood results are as follows:

Hb	110 g/L	Male: (135-180) Female: (115 - 160)
Platelets	182 * 10 /L 9	(150 - 400)
WBC	6.8 * 10 /L 9	(4.0 - 11.0)
Na +	134 mmol/L	(135 - 145)
K +	5.1 mmol/L	(3.5 - 5.0)
Urea	16.2 mmol/L	(2.0 - 7.0)
Creatinine	288 µmol/L	(55 - 120)
Paraprotein	No paraprotein detected	
Kappa free light chains	12,450 mg/L	(3.3 - 19.4)
Lambda free light chains	125 mg/L	(5.7 - 26.3)
Kappa to Lambda ratio	99.6	(0.26 - 1.65)

An MRI spine confirms cauda equina syndrome due to a large paravertebral soft tissue mass. In addition, there is widespread abnormal marrow involvement throughout the spine.

What is the most likely diagnosis?

A. High grade B cell lymphoma
B. Multiple myeloma
C. Prostate carcinoma
D. Solitary bone plasmacytoma
E. Solitary extra medullary plasmacytoma

B. Multiple myeloma

In approximately 1-2% of myeloma cases, the myeloma cells produce very low amounts of paraprotein, which can only be detected using the serum free light chain test. This is called oligosecretory myeloma

The diagnosis is most certainly **multiple myeloma** due to evidence of a monoclonal plasma cell population (markedly raised kappa to lambda ratio) and myelomatous end-organ dysfunction (e.g. anaemia, renal dysfunction and diffuse bone involvement). Although the paraprotein is negative, in approximately 1-2% of myeloma cases, the myeloma cells produce very low amounts of paraprotein, which can only be detected using the highly sensitive serum free light chain test.

Light chains are secreted by B-cells and plasma cells. Individual B- or plasma cells possess either kappa or lambda light chains, but never both together. Interpretation of serum free light chains (sFLC) can be confusing. It is important to remember that it is the ratio that is the most important aspect of the analysis. Several diseases can give rise to increased absolute levels of sFLC. For example due to decreased excretion as seen in renal failure, or due to increased production (e.g. infections, or inflammatory conditions). These 'reactive' causes will result in a broadly similar increase in both kappa and lambda light chains thereby maintaining a normal ratio. However, in monoclonal conditions, the B-cell or plasma cell clone will be either lambda or kappa light chain restricted subsequently resulting in an abnormal ratio.

The large soft tissue mass likely represents an extramedullary plasmacytoma. However, the diagnosis of a **solitary extramedullary plasmacytoma** is excluded given that the patient has myelomatous end-organ dysfunction and involvement of the axial skeleton.

Similarly, a **solitary bone plasmacytoma** is excluded due to the absence of a bone plasmacytoma and the presence of myelomatous end-organ dysfunction.

Although lymphomas can present with soft tissue masses and produce light chains, the clinical features (e.g. anaemia and renal impairment) are more in keeping with myeloma, making a **highgrade B cell lymphoma** less likely.

Prostate carcinoma classically presents with sclerotic bone metastases, and would not cause an abnormal serum free light chain ratio.

Question 30 of 89

A 72-year-old man attends the emergency department with a headache and blurred vision which has progressively worsened over the past 2 weeks. On examination, his observations are within normal limits, and he has no focal neurology. On fundoscopy, there is bilateral retinal vein dilation and tortuosity with visible retinal haemorrhages.

Blood results are as follows:

Hb	90 g/L	Male: (135-180)Female: (115 - 160)
Platelets	110 * 10 /L 9	(150 - 400)
WBC	4.8 * 10 /L 9	(4.0 - 11.0)
Na +	138 mmol/L	(135 - 145)
K +	4.2 mmol/L	(3.5 - 5.0)
Urea	10.8 mmol/L	(2.0 - 7.0)
Creatinine	184 µmol/L	(55 - 120)
CRP	4 mg/L	(< 5)

Plasma viscosity	4.2mPas	(1.50 -1.72)
IgG	1.2 g/L	(6-16)
IgM	42 g/L	(0.4 - 2.5)
IgA	0.2 g/L	(0.8 - 3.0)

Given the likely diagnosis, what treatment is urgently indicated?

A. Chemotherapy
B. Ibrutinib
C. Plasma exchange
D. Red cell transfusion
E. Steroids

C. Plasma exchange

Patients with Waldenstrom's macroglobulinaemia often present with issues secondary to hyperviscosity

The patient has clinical features of hyperviscosity (e.g. headache, blurred vision, and renal impairment). This is confirmed biochemically with the raised plasma viscosity. The fundoscopic appearances (bilateral retinal vein dilation and tortuosity with retinal haemorrhages) are also classic for hyperviscosity within the retinal vasculature.

The markedly raised IgM level is suggestive of Waldenstrom's macroglobulinaemia.

The presence of anaemia and thrombocytopenia are most likely due to bone marrow infiltration. However alternative causes should also be considered (e.g. autoimmune haemolytic anaemia, and immune thrombocytopenia).

Plasma exchange is correct. Hyperviscosity is a haematological emergency and the patient thus needs emergency plasma exchange. Plasma exchange is an extra-corporeal technique able to remove macromolecules (e.g. IgM) from blood.

Chemotherapy is incorrect. Although the patient will most certainly need chemotherapy to treat their underlying disorder, the first priority is to treat the hyperviscosity by reducing the IgM level with plasma exchange.

Ibrutinib is incorrect. Ibrutinib is a type of tyrosine kinase inhibitor (TKI) that is highly efficacious in a variety of haematological malignancies. It is less toxic compared to traditional chemotherapies and is therefore often used as a first-line agent in frail elderly patients. It can also be used in Waldenstrom's macroglobulinaemia however at the current time the priority is for plasma exchange.

Red cell transfusion is incorrect. Although the patient is anaemic, they most certainly should not receive a red cell transfusion at the current time as this could worsen the hyperviscosity.

Steroids is incorrect. Steroids are often used as chemotherapy adjuncts to treat Waldenstrom's macroglobulinaemia, however at the current time the priority is for plasma exchange.

Question 31 of 89

A 35-year-old woman who was diagnosed with hereditary angioedema is about to undergo an elective meniscal repair for her left knee. She has been well otherwise with no recent changes to her health or medications.

Which is the drug of choice for prophylaxis for her hereditary angioedema prior to her procedure?

A. Prednisolone
B. Hydrocortisone
C. Conestat alfa
D. Tranexamic acid
E. Icatibant

D. Tranexamic acid

The best answer here is tranexamic acid.

In hereditary angioedema, the relevant medications can be used as follows:

A C1-esterase inhibitor can be used for short-term prophylaxis before procedures or to terminate acute attacks of hereditary angioedema. Conestat alfa and icatibant are licensed for the treatment of acute attacks of hereditary angioedema in adults with C1-esterase inhibitor deficiency. Tranexamic acid and danazol are used for short-term and long-term prophylaxis.

There is no role of glucocorticoids in this case

Question 32 of 89

A 65-year-old woman presents to the emergency department with a painful leg. She had noticed that her calf started to become tender when she was getting dressed in the morning and found it to be swollen. She was concerned and called her GP surgery who gave her an emergency appointment. She was seen by her GP earlier in the day who suspected a deep vein thrombosis and advise her to attend her local emergency department.

She has a past medical history of breast cancer which was operated on three months ago with a wide local excision, and she has been told that the operation was successful in removing the cancer. Her observations are stable. On examination, she has a swollen left calf which is mildly tender without erythema. A D-dimer sent by the emergency department team was positive but all other blood tests are normal. She undergoes a doppler ultrasound scan which shows no thrombus.

How should she be further managed?

A. Start warfarin
B. Repeat ultrasound scan within 6-8 days
C. CT pulmonary angiogram
D. Start treatment dose low-molecular weight heparin
E. Repeat D-dimer

B. Repeat ultrasound scan within 6-8 days

The correct answer is to repeat the ultrasound scan within 6-8 days. This is a patient with a major risk factor for deep vein thrombosis (DVT) and pulmonary embolus (PE) who presents with typical features of a DVT; unilateral tender and swollen calf. She would likely have a Well's score at 2 and be considered at high risk.

Since the ultrasound is negative, a repeat ultrasound is advised, and there as of yet no indications to start treatment with either warfarin or heparin. In patients with cancer warfarin is not recommended as treatment for DVT or PE. A CT pulmonary angiogram is not indicated as there are no symptoms or signs suggestive of a pulmonary embolus. Repeating the D-dimer is unlikely to give any additional information.

'Venous thromboembolic diseases: diagnosis, management and thrombophilia testing' Clinical guideline [CG144]. The National Institute for Health and Care Excellence, June 2012.

Question 33 of 89

A 44-year-old female is referred to haematology outpatients with fatigue, shortness of breath and abnormal blood results organized by her general practitioner. She has no past medical history and is on no medications. She does not smoke or drink alcohol. She works in a bakery.

Clinical examination is unremarkable. Blood tests:

Hb	101 g/L	Male: (135-180)Female: (115 - 160)
Platelets	111 * 10 /L 9	(150 - 400)
WBC	14.2 * 10 /L 9	(4.0 - 11.0)
Na +	138 mmol/L	(135 - 145)
K +	4.1 mmol/L	(3.5 - 5.0)
Urea	5.2 mmol/L	(2.0 - 7.0)
Creatinine	88 µmol/L	(55 - 120)
CRP	4 mg/L	(< 5)

A blood film is organized, which demonstrates Auer rods. What is the most likely diagnosis?

A. Acute lymphocytic leukaemia
B. Acute promyelocytic leukaemia
C. Chronic lymphocytic leukaemia
D. Chronic myeloid leukaemia
E. Thrombotic thrombocytopenic purpura

B. Acute promyelocytic leukaemia

Auer rods on blood film strongly suggests acute promyelocytic leukaemia

Acute lymphocytic leukaemia is incorrect. Auer rods are not typical of the peripheral blood film in this condition. It may show blast cells in this case or it may be normal if the cells are confined to the bone marrow.

Chronic myeloid leukaemia is incorrect. The blood film in this case would not demonstrate Auer rods but rather a significant leucocytosis consisting of the whole spectrum of mature granulocytes. Organomegaly and lymphadenopathy are common.

Chronic lymphocytic leukaemia is incorrect. This typically affects an older age range with a significant lymphocytosis. The blood film may show 'smear' cells.

Thrombotic thrombocytopenic purpura is incorrect. Clinically this is often characterized by confusion, headache, fever and bruising. It is a medical emergency. The blood film will show schistocytes.

Question 34 of 89

A 58-year-old male is referred to the haematology outpatient department with reports of widespread bruising. He does not have any significant past medical history and does not take any regular medications. He does not smoke or drink alcohol.

On examination, there are widespread ecchymoses affecting all of his limbs, abdomen and thorax. Blood tests:

Hb	111 g/L	Male: (135-180)Female: (115 - 160)
Platelets	167 * 10 /L 9	(150 - 400)
WBC	4.2 * 10 /L 9	(4.0 - 11.0)
Na +	138 mmol/L	(135 - 145)
K +	4.2 mmol/L	(3.5 - 5.0)
Urea	5.2 mmol/L	(2.0 - 7.0)
Creatinine	88 µmol/L	(55 - 120)
CRP	4 mg/L	(< 5)
Activated partial thromboplastin time (APTT)	61 seconds	(30-40)
Prothrombin time	11 seconds	(11 - 12.5)
Factor VIII	8 IU/dl	(50-150)
Bleeding time	5 minutes	(3-10)
Lupus anticoagulant	absent	(present)

A mixing study is abnormal and further testing demonstrates the presence of factor VIII inhibitor.

What is the most likely diagnosis?

A. Acquired haemophilia A
B. Haemophilia B
C. Hereditary haemophilia A
D. Vitamin K deficiency
E. Von Willebrand's disease

A. Acquired haemophilia A

Acquired haemophilia A is diagnosed in patients without previous or familial histories of bleeding who have isolated prolongation of the activated partial thromboplastin time (aPTT), reduced FVIII levels as well as evidence of FVIII inhibitor activity

Acquired haemophilia A is the correct answer. This acquired haemophilia presents in later life without a preceding history of coagulopathy or family history. It is characterised by an isolated prolongation of the activated partial thromboplastin time (aPTT), reduced FVIII levels as well as evidence of FVIII inhibitor activity. It typically presents with bleeding into the skin or muscles and occasionally haematuria or melena. Haemarthrosis is less likely than with an inherited haemophilia. It is autoimmune disorder with the formation of antibodies that attack clotting factors. A mixing study is crucial in differentiating inherited factor deficiencies from acquired factor deficiencies. In inherited disorders that are characterized by a factor deficiency, the addition of normal plasma to blood restores the clotting parameters. In individuals with a factor inhibitor, the coagulation test remains abnormal.

Haemophilia B is incorrect. This is inherited bleeding disorder characterised by deficiency of factor IX.

Hereditary haemophilia A is incorrect. The absence of a family history, bleeding from a young age and normal mixing study, make this an unlikely diagnosis.

Vitamin K deficiency is incorrect. This will cause an abnormal PT and APTT.

Von Willebrand's disease is incorrect. This will cause an abnormal bleeding time.

Question 35 of 89

A 55-year-old woman is referred to the haematology outpatient department with unexplained eosinophilia. She has a past medical history of systemic lupus erythematosus and recurrent urinary tract infections. Her regular medications include azathioprine and nitrofurantoin.

On examination, there is a rash across the malar region that spares the nasolabial folds but is otherwise unremarkable.

Plain radiography of the chest is unremarkable.

Hb	136 g/L	Male: (135-180)Female: (115 - 160)
Platelets	189 * 10 /L 9	(150 - 400)
WBC	8.2 * 10 /L 9	(4.0 - 11.0)
Neuts	5.2 * 10 /L 9	(2.0 - 7.0)
Lymphs	2.0 * 10 /L 9	(1.0 - 3.5)
Mono	0.2 * 10 /L 9	(0.2 - 0.8)
Eosin	0.8 * 10 /L 9	(0.0 - 0.4)

What is the most likely explanation for her eosinophilia?

A. Azathioprine
B. Eosinophilic granulomatosis with polyangiitis
C. Hypereosinophilic syndrome
D. Nitrofurantoin
E. Systemic lupus erythematosus

D. Nitrofurantoin

Nitrofurantoin can cause eosinophilia

Nitrofurantoin is correct. This is an antibiotic used for urinary-tract infections, sometimes given as a prophylactic long-term dose to prevent recurrent infections. It is associated with eosinophilia as a recognised side effect and is the most likely explanation in this case.

Azathioprine is incorrect. Eosinophilia is not a common side effect of this medication. Indeed, it may be used as a steroid-sparing agent in some hyper-eosinophilic syndromes. It is more likely to cause cytopaenias as a side effect.

Eosinophilic granulomatosis with polyangiitis (EGPA) is incorrect. A malar rash is not typical of EGPA but rather of lupus. EGPA typically causes a vasculitic rash, which would be non-blanching and not likely to be located in the malar region. Additionally, there is no history of asthma or sinus problems, making the diagnosis unlikely. Furthermore, the eosinophil count in this disorder tends to be >1.5 * 10 /L.

Hypereosinophilic syndrome is incorrect. This is a myeloproliferative disorder associated with persistent eosinophilia and organ damage. The eosinophil count must be at least 1.5 * 10 /L for this diagnosis to be considered.

Systemic lupus erythematosus (SLE) is incorrect. This disorder is associated with haematological abnormalities. However, active SLE is typically associated with cytopaenias (anaemia, thrombocytopaenia, leucopaenia) rather than the converse. Eosinophilia is not typical

Question 36 of 89

A 74-year-old patient is referred to the haematology outpatient clinic with a lump in her neck. She has no past medical history.

On examination, widespread lymphadenopathy is detectable in the cervical, inguinal and axillary regions.

Hb	121 g/L	Male: (135-180)Female: (115 - 160)
Platelets	189 * 10 /L 9	(150 - 400)
WBC	95 * 10 /L 9	(4.0 - 11.0)
Neuts	2.0 * 10 /L 9	(2.0 - 7.0)
Lymphs	92.7 * 10 /L 9	(1.0 - 3.5)
Mono	0.2 * 10 /L 9	(0.2 - 0.8)
Eosin	0.1 * 10 /L 9	(0.0 - 0.4)

Given the presentation, what is the most important investigation to confirm the diagnosis?

A. Bone marrow aspiration
B. Bone marrow trephine biopsy
C. Cytogenetics
D. Immunophenotyping
E. Labelled white blood cell scan

D. Immunophenotyping

CLL - immunophenotyping is investigation of choice

Immunophenotyping is incorrect. Immunophenotyping by flow cytometry is a method used to detect the presence or absence of white blood cell antigens. These antigens are protein structures associated with white blood cells. Specific groupings of these antigens are normally present on or within white blood cells (WBCs) and are unique to specific cell types and stages of cell maturation. Specific patterns of antigens are present on abnormal cells seen in specific lymphomas like chronic lymphocytic leukaemia (CLL). On flow cytometry, CLL cells classically demonstrate CD5, CD19, CD23, weak monotypic surface immunoglobulin and weak or absent CD79B, CD22 and FMC7.

Bone marrow aspiration is incorrect. A bone marrow aspirate contains a liquid sample from the bone marrow for analysis. Bone marrow (BM) aspirate and trephine biopsy is not typically necessary at diagnosis but is used in cases with atypical features or to rule out differential diagnoses in the case of anaemia/thrombocytopenia.

Bone marrow trephine biopsy is incorrect. A trephine biopsy removes a small piece of bone with the marrow inside. Again, this is not required at diagnosis. Bone marrow trephine biopsies may show interstitial, nodular or diffuse infiltration if carried out.

Cytogenetics is incorrect. This may be used to inform prognosis (as a complex karyotype is associated with poor survival). However, it is not typically required at diagnosis.

Labelled white blood cell scan is incorrect. This scan is typically used to localise a site of unknown infection rather than diagnose CLL

Question 37 of 89

A 44-year-old male presents with a 2-month history of increasing lethargy. His wife reports him to be not quite himself for the past 2 months now, with poor oral intake and poor appetite. He is a self-employed software engineer but currently is unable to work due to his lethargy. He was previously treated for Hodgkin's lymphoma 5 years ago and has since been in remission.

On examination, he has no conjunctival pallor, is dry on his mucous membranes and extremely lethargic. Firm, rubbery lymph nodes are noted in the right axilla. Cardiovascular examination reveals a soft systolic murmur, respiratory and abdominal examinations are unremarkable. He is a non-smoker and drinks alcohol only occasionally. His blood tests are as follows:

Hb	88 g/l
MCV	104 fl
Platelets	94 * 10 /l9
WBC	12.8 * 10 /l9
Red cell distribution	9% (normal 11.5-14.5%)
Blood film	leucoerythroblastic with myeloblasts

What is the most likely cause of this patient's anaemia?

A. B12 and folate deficiency
B. Iron deficiency
C. Anaemia of chronic disease
D. Myelodysplasia post-chemotherapy
E. Marrow infiltration

A. Marrow infiltration

This patient has a symptomatic macrocytic anaemia, of which B12 and folate deficiency, myelodysplasia post-chemotherapy and marrow infiltration could all be causes. Iron deficiency results in a microcytic anaemia while anaemia of chronic disease typically causes a microcytic or normocytic anaemia. Blood cells in B12 and folate deficiency are typically megaloblastic, large immature red cells caused by impaired DNA synthesis. Myelodysplasia typically results in red cells of abnormal shapes (poikilocytosis) and sizes (anisocytosis) with Pappenheimer bodies (abnormal granules of iron). Chemotherapy is a possible cause of myelodysplasia but typically after recent treatment only. Sadly, in this case, the blood film of myeloblasts and erythroblasts suggests the transformation of Hodgkin's lymphoma into acute myeloid leukaemia, a well-recognised complication of Hodgkin's lymphoma chemotherapy with older alkylating agents, typically 5 to 10 years after treatment. Blast cells have now subsequently infiltrated the bone marrow, resulting in macrocytic anaemia.

Question 38 of 89

A 78-year-old gentleman with chronic lymphocytic leukaemia (CLL) presents with spontaneous bruising. He recalls no trauma and is otherwise well although he takes aspirin, simvastatin and ramipril for a previous myocardial infarction.

On examination, he looks well. His conjunctiva are pale but his chest is clear and he is warm and well perfused. A number of purpura are noted across the arms, back and chest. Palpable lymph nodes are felt in the left inguinal region. There is no hepatosplenomegaly.

Hb	104 g/l	Na +	135 mmol/l
Platelets	54 * 10 /l 9	K +	4.0 mmol/l
WBC	10.3 * 10 /l 9	Urea	3.4 mmol/l
Neuts	6.7 * 10 /l 9	Creatinine	87 µmol/l
Lymphs	3.4 * 10 /l 9	CRP	14 mg/l
Eosin	0.1 * 10 /l9		

What is the most appropriate management option?

A. Suspend aspirin
B. Start chlorambucil
C. Start dexamethasone
D. Platelet transfusion
E. Splenectomy

B. Start chlorambucil

Thrombocytopenia -> indication to start treatment in CLL

This gentleman has developed thrombocytopenia on a background of CLL. This is an indication to start chemotherapy. In some cases, these may also respond to steroids but typically chemotherapy would be started and chlorambucil is a commonly used agent. Suspending aspirin will not fix thrombocytopenia but may be advised. There are no signs of hypersplenism as the cause of the thrombocytopenia. Platelet transfusion will not fix the underlying problem and is not urgent given there is no acute bleeding.

Question 39 of 89

A 30-year-old previously fit and well gentleman is injured following a road traffic accident after being thrown off his motorcycle. He was blue-lighted to the emergency department, where he was found to have multiple, profusely bleeding, lacerations of his extremities.

He was transfused 2 units of cross-matched blood, with no reactions detected in blood bank. Ten minutes after the transfusion, the patient developed severe urticaria.

Which of the following syndromes would contribute to the patient's picture?

A. Adenosine deaminase deficiency
B. Ataxia telangiectasia
C. DiGeorge syndrome
D. Selective IgA deficiency
E. Wiskott-Aldrich syndrome

D. Selective IgA deficiency

IgA deficiency is associated with anaphylactic reaction to blood products

The majority of selective IgA deficiency patients are asymptomatic; with about 5% suffering from recurrent respiratory tract infections. The condition becomes clinically significant when blood transfusion is required, as there is the potential for anaphylaxis to occur on exposure to IgA containing blood products.

Although rare, anaphylaxis can happen from the first transfusion if the patient has been previously exposed to IgA i.e. in consumed animal meat products. If ever the patient needs a future blood transfusion, the transfused blood must be IgA free.

Question 40 of 89

A 16-year-old female was admitted with new onset facial swelling. The facial swelling started 45 minutes ago and initially involved her lips. She complained of a sensation of choking and a feeling of being unable to speak with hoarseness of her voice. She had been investigated on multiple occasions for abdominal pain and was diagnosed with non-specific abdominal pain. She was not taking any medication and was otherwise healthy prior to the admission.

She was given prednisolone 40mg PO and chlorpheniramine 10mg PO and admitted for observation. Whilst in the department she developed profound shortness of breath with associated stridor. Her swelling around her lips worsened and involved the whole of her face. On examination, she was in respiratory distress with severe biphasic stridor. Her respiratory rate was 32/min with an oxygen saturation of 88% on air. Auscultation of her chest also revealed the presence of a widespread polyphonic wheeze. Examination of her cardiovascular system revealed the presence of flushed peripheries with a bounding peripheral pulse. Her pulse was 102bpm and her blood pressure was 92/68 mmHg. Her GCS was 15 and neurological and abdominal examinations were unremarkable. She was cannulated and commenced on stat intravenous colloid solution. She was given adrenaline 0.5mg IM on three separate occasions within 10 minutes with no improvement. She was transferred immediately to the Intensive Care Unit and an anaesthetist fast bleeped to secure her airway.

What is the best immediate management step pending definitive airway management?

A. Commence IV adrenaline infusion
B. Commence danazol
C. Commence IV dopamine
D. Commence fresh frozen plasma infusion
E. Commence C1 esterase inhibitor concentrate infusion

E. Commence C1 esterase inhibitor concentrate infusion

Hereditary angioedema: the acute management is IV C1-inhibitor concentrate or fresh frozen plasma if this is not available

This patient has hereditary angioedema. This condition is characterised by a lack of C1 esterase inhibitor and may present with laryngeal oedema, recurrent abdominal pain and localized subcutaneous swelling. Laryngeal oedema may be fatal, does not respond to glucocorticoids or antihistamines, and has an only modest response to adrenaline. The immediate management is to commence C1 esterase inhibitor concentrate infusion. Fresh frozen plasma infusion may be administered if the concentrate is not available. Danazol is more suitable for subcutaneous oedema or as prophylaxis in certain situations eg prior to surgery.

Question 41 of 89

An 18-year-old woman presents to the emergency department with two weeks of right upper quadrant pain. She also reported longstanding pain in her right femur. She has recently returned from a holiday in Tanzania.

On examination, there was mild tenderness in the right upper quadrant of the abdomen. There was hepatomegaly and splenomegaly detectable along with conjunctival pallor. She is afebrile. There is tenderness over the right femur.

Hb	91 g/L	Male: (135-180)Female: (115 - 160)
Platelets	112 * 10 /L 9	(150 - 400)
WBC	4.2 * 10 /L 9	(4.0 - 11.0)
Na +	138 mmol/L	(135 - 145)
K +	4.2 mmol/L	(3.5 - 5.0)
Urea	5.3 mmol/L	(2.0 - 7.0)
Creatinine	88 µmol/L	(55 - 120)
CRP	4 mg/L	(< 5)
Bilirubin	12 µmol/L	(3 - 17)
ALP	88 u/L	(30 - 100)
ALT	32 u/L	(3 - 40)
Î³GT	44 u/L	(8 - 60)
Albumin	36 g/L	(35 - 50)
LDH	512 U/L	(200-400)
Recticulocytes	43 * 10 /L 3	(5-15)
Direct antiglobulin test	negative	(negative)
Haptoglobin	101 mg/dl	(50-220)

An ultrasound abdomen demonstrates massive splenomegaly (24 x 8 cm) and hepatomegaly. An x-ray of the right femur demonstrates an Erlenmeyer flask deformity.

What is the likely diagnosis?

A. Autoimmune haemolytic anaemia
B. Chronic myeloid leukaemia
C. Gaucher's disease
D. Malaria
E. Myelofibrosis

C. Gaucher's disease

Gaucher disease causes splenomegaly

Gaucher's disease is the correct answer. The patient has hepatomegaly and massive splenomegaly along with anaemia and thrombocytopenia. While all of the options listed can cause splenomegaly, only Gaucher's disease is associated with constriction of the diaphysis and flaring of the metaphysis of the femur, resulting in a deformity known as the Erlenmeyer flask deformity (named because it resembles the conical flask used by chemists).

Autoimmune haemolytic anaemia is incorrect. This is a cause of mild rather than massive splenomegaly. Additionally, the negative direct antiglobulin test and haptoglobin make haemolysis unlikely.

Chronic myeloid leukaemia is incorrect. While this can cause hepatosplenomegaly and cytopenias, it is not associated with characteristic bone abnormalities like Gaucher disease. Additionally, it tends to present in older age rather than childhood/adolescence, like Gaucher's disease.

Malaria is incorrect. While she has travelled recently to Africa, she is afebrile and therefore this diagnosis is very unlikely.

Myelofibrosis is incorrect. This is another cause of massive splenomegaly. However, the characteristic bone abnormality in this condition is osteosclerosis. Additionally, it tends to present after the age of 60.

Question 42 of 89

A 72-year-old lady is on her third cycle of chemotherapy following mastectomy and axillary clearance of her left breast. She has a PICC line in situ through which she receives her chemotherapy. She developed chills and lethargy two days prior which led her to seek her GP who arranged admission to hospital. She has had one episode of diarrhoea and is currently being barrier nursed. Her swallowing is a problem due to odynophagia and she is only managing to take oral fluids.

She is currently taking G-CSF, omeprazole and dexamethasone. She has not travelled abroad and reports that she is excluding soft cheeses and pates from her diet.

On exam, she has a clear chest with no murmurs. There is *Candida* in the mouth and a temperature of 38.5ºC recorded on admission. There are no wounds or rashes to be seen. She was started on broad-spectrum intravenous antibiotics and oral nystatin and admitted. You are asked to review her as she has spiked a fever of 39.8ºC on her third day after admission and she is struggling to swallow fluids due to retrosternal pain.

Hb	96 g/l	Na	139 mmol/l
Platelets	123 * 10 /l	K	5.1 mmol/l
WBC	1.6 * 10 /l	Urea	4.5 mmol/l
Neuts	0.5 * 10 /l	Creatinine	56 µmol/l
Lymphs	0.1 * 10 /l	CRP	96 mg/l
Eosin	0.1 * 10 /l		

Chest X-ray	clear lung fields, no effusion, no air under the diaphragm
Bloods cultures (1st peripheral)	no growth
Blood cultures (PICC line)	no growth
Blood cultures (2nd peripheral)	coagulase negative *staphylococcus*
Urine microscopy	no pyuria, no growth
Skin swab (PICC site)	awaited

What of the following is the most appropriate action?

A. Start metronidazole
B. Start fluconazole
C. Take a further set of blood cultures
D. Increase dose of G-CSF
E. Start paracetamol

B. Start fluconazole

Neutropenic sepsis with no response to antibiotics at 48 hrs - > possible fungal infection

This lady has neutropenic sepsis of unknown source and has failed to improve despite broadspectrum antibiotics. This prompts thoughts of what the cause could be. Increasing G-CSF may help her white cells increase but it does not treat the infection present. Paracetamol may reduce fevers but it does not treat the infection. Adding metronidazole is a reasonable thought since it covers anaerobic organisms and she has had a single episode of diarrhoea but the source of the sepsis is not clearly abdominal. Taking two sets of further blood cultures will be part of the workup needed but will not improve the patient's situation. Starting fluconazole is the answer. In any neutropenic sepsis, fungal causes should be considered especially if the patient does not improve on broad spectrum antibiotics. She has oral candida with odynophagia suggests possible oesophageal candidiasis. We should consider this especially given the *Candida* seen.

Question 43 of 89

A 50-year-old woman presents to the emergency department with a 3-day history of headache and blurred vision. Despite taking co-codamol and ibuprofen, her headache has not been relieved and she has since started vomiting. She describes feeling generally unwell for the last 2-3 weeks with general malaise and body aches but denies fever and has no past medical history.

On examination, her visual acuity is reduced with noticeable nystagmus more pronounced on the left side. The remainder of her neurological examination is unremarkable.

Laboratory tests:

Hb	99 g/L	(115 - 160)
Platelets	150 * 10 /L 9	(150 - 400)
WBC	38.3 * 10 /L 9	(4.0 - 11.0)
Lymphs	3.2 * 10 /L 9	(1.0 - 3.5)
Eosin	0.2 * 10 /L 9	(0.0 - 0.4)
Na +	136 mmol/L	(135 - 145)
K +	3.8 mmol/L	(3.5 - 5.0)
Urea	6.2 mmol/L	(2.0 - 7.0)
Creatinine	90 µmol/L	(55 - 120)
Bilirubin	20 µmol/L	(3 - 17)
ALP	87 u/L	(30 - 100)
ALT	39 u/L	(3 - 40)
Albumin	38 g/L	(35 - 50)

What is the most likely diagnosis?

A. Acute myeloid leukaemia
B. Idiopathic intracranial hypertension
C. Meningitis
D. Multiple sclerosis
E. Non-Hodgkin's lymphoma

A. Acute myeloid leukaemia

Acute myeloid leukaemia can present acutely with hyperviscosity syndrome

This patient is presenting with hyperviscosity syndrome. Hyperviscosity syndrome is the increased viscosity of the blood caused by hyperproliferative states (e.g. leukaemia and polycythaemia), increased cell components (e.g. RBCs and WBCs) or increased immunoglobulins (e.g. multiple myeloma). Different bodily systems can be affected, but commonly the central nervous can be affected causing headaches, nystagmus and visual disturbance. This patient's blood results demonstrate a raised WBC count and a normal lymphocyte count, indicating that a large portion of the WBC is made of neutrophils, in keeping with a diagnosis of **acute myeloid leukaemia**. Additional symptoms of acute myeloid leukaemia include lethargy and malaise that are also described by this patient.

Idiopathic intracranial hypertension (IIH) is most common in women and is associated with obesity. It can present with a pressure-type headache with blurred vision. However, eye signs include 6th nerve palsy and papilloedema on fundoscopy. Furthermore, IIH does not explain this patient's raised WBC.

Meningitis can be secondary to a viral or bacterial infection. Presenting features typically include headache, fever and meningism. Vomiting and photophobia may also be seen. Although this patient has a raised WBC that could represent an underlying infection, meningitis does not typically cause nystagmus and the absence of fever and meningism also suggest an alternative diagnosis.

Multiple sclerosis is a demyelinating disease of the central nervous system. Common presenting symptoms include optic neuritis. Although a patient with multiple sclerosis could present with headache and nystagmus, it does not explain this patient's raised WBC.

Non-Hodgkin's lymphoma is a malignant proliferation of lymphocytes. It can present with malaise and hyperviscosity syndrome as described by this patient. However, additional symptoms include painless lymphadenopathy and constitutional symptoms such as night sweats and weight loss which are not described in this question. Furthermore, the presence of a normal lymphocyte count points towards an alternative diagnosis.

Question 44 of 89

A 44-year-old woman visits her general practitioner with a 10-day history of intermittent epistaxis. She has never had nosebleeds before but now says that even after blowing her nose, it will bleed for 2-3 minutes at a time. There is a past medical history of hypothyroidism for which she takes levothyroxine 75 micrograms daily. Other than recovering from influenza 3 weeks ago, she has had no recent illnesses.

On examination, she is afebrile. Her heart rate is 71bpm and regular with a blood pressure of 125/65mmHg. On auscultation, she has a clear chest with normal heart sounds. Her abdomen is soft and non-tender with no palpable organomegaly. There are multiple bruises and petechiae over her lower limbs and trunk.

Investigations:

Hb	116 g/L	Male: (135-180)Female: (115 - 160)
Platelets	16 * 10 /L 9	(150 - 400)
WBC	5.1 * 10 /L 9	(4.0 - 11.0)
Na +	136 mmol/L	(135 - 145)
K +	3.5 mmol/L	(3.5 - 5.0)
Urea	5.0 mmol/L	(2.0 - 7.0)
Creatinine	61 µmol/L	(55 - 120)
Prothrombin time (PT)	12 secs	(10-14 secs)
Activated partial thromboplastin time (APTT)	28 secs	(25-35 secs)
Fibrinogen	2.6 g/L	(2 - 4)
D-Dimer	254 ng/mL	(< 400)

What is the most appropriate next step in this patient's management?

A. Desmopressin
B. Eltrombopag
C. IV immunoglobulin
D. Platelet transfusion
E. Prednisolone

A. Prednisolone

First-line treatment for ITP is oral prednisolone

This patient has immune thrombocytopenic purpura (ITP) the immune-mediated destruction of platelets. In ITP, autoantibodies are directed against glycoprotein IIb/IIIa or Ib-V-IX complexes and can be detected in up to 60-70%. The disease can develop acutely in children following a viral infection or vaccination and usually presents with mucocutaneous bleeding. There is also an association with autoimmune disease. ITP in children is often self-limiting compared to adults which tends to be more of a chronic condition with a relapsing-remitting course. This patient has a history of hypothyroidism and is recovering from a recent viral infection making ITP more likely. Features include easy bruising, epistaxis, purpura and menorrhagia. Massive haemorrhage is a rare complication. The treatment of choice is oral prednisolone at a dose of 1-2mg/kg. Approximately 80% respond to initial treatment.

Desmopressin is used in the treatment of Von-Willebrand's disease (VWD). VWD is a common inherited bleeding disorder that may first present as spontaneous mucocutaneous bleeding. It is caused by a deficiency in the von-Willebrand factor, a protein molecule that encourages platelet adhesion and binds to clotting factor VIII to prevent it from being broken down. A deficiency in this factor predisposes patients to increased bleeding. Blood tests in patients with VWD may demonstrate a microcytic anaemia, thrombocytopenia and a prolonged APTT (especially if factor VIII levels are reduced). However, this patient has never encountered issues with bleeding prior to a recent viral illness. This history combined with an isolated thrombocytopenia makes ITP the more likely diagnosis.

Eltrombopag is a thrombopoietin receptor agonist which acts to increase the platelet count. It is used as a 3rd-line treatment of ITP and is typically prescribed for chronic or refractory ITP.

IV immunoglobulin (IVIG) is another potential treatment option for ITP. It is commonly used in patients who fail to respond to corticosteroids or in patients for which corticosteroids are contraindicated. Another indication for IVIG is if a rapid rise in platelets is needed, for example prior to surgery.

Platelet transfusions are not the treatment of choice in ITP. They are reserved for extreme haemorrhage including intracranial haemorrhage.

Question 45 of 89

A 39-year-old man is referred to the haematology clinic by his GP due to abnormal blood results.

In the clinic, he reports a 2-week history of fatigue, malaise and intermittent abdominal pain. His past medical history includes anxiety and depression for which he takes citalopram. There is no history of smoking or recreational drug use although he drinks 6 cans of cider most evenings. He returned from a trip to India last month.

On examination, he appears lethargic. Chest auscultation is clear and his abdomen is soft with generalised tenderness.

Repeat blood tests are requested:

Hb	100 g/L	(135-180)
Mean Cell Volume	105 fL	(80 - 100)
Platelets	100 * 10 /L 9	(150 - 400)
WBC	7.1 * 10 /L 9	(4.0 - 11.0)
Bilirubin	80 µmol/L	(3 - 17)
ALP	118 u/L	(30 - 100)
ALT	40 u/L	(0 - 40)
AST	65 u/L	(< 35)

Blood glucose	6.1 mmol/L	(4 - 7)
Total cholesterol	7.3 mmol/L	(< 5)

Further investigations are sent:

Blood film	Schistocytes
Coombs test	Negative

What is the most appropriate management for this patient?

A. Alcohol cessation
B. Artesunate
C. Blood transfusion
D. Intravenous immunoglobulin (IVIg)
E. Prednisolone

A. Alcohol cessation

Zieve syndrome usually resolves with abstinence from alcohol

Alcohol cessation is correct. This patient has a likely diagnosis of Zieve syndrome given the triad of non-autoimmune haemolysis, jaundice and hyperlipidaemia on a background of alcohol excess. The negative Coombs test indicates non-autoimmune haemolytic anaemia. In Zieve syndrome, jaundice can be cholestatic in nature but also secondary to haemolysis. The raised ALP supports a cholestatic picture. Zieve syndrome is a rare syndrome and generally resolves following the cessation of alcohol use.

Artesunate is incorrect. Another differential diagnosis for haemolytic anaemia includes infective causes such as malaria. Although this patient has recently travelled to India, the majority of India is relatively malaria-free in comparison to other parts of the world. It also does not account for the raised triglyceride level and cholestatic jaundice.

Blood transfusion is incorrect. Generally, the threshold for blood transfusion is a Hb < 70g/dL or < 80g/dL if the patient has ischaemic heart disease. It would not treat this patient's underlying diagnosis.

Intravenous immunoglobulin (IVIg) is incorrect. This can be used to treat autoimmune haemolytic anaemia and other haematological conditions such as immune thrombocytopenic purpura. However, the negative Coombs test makes autoimmune haemolytic anaemia highly unlikely.

Prednisolone is incorrect. Like IVIg, prednisolone is used in the treatment of autoimmune haemolytic anaemia. This is unlikely to be the underlying diagnosis since the Coombs test is negative. Therefore, other causes of haemolytic anaemia should be considered and investigated

Question 46 of 89

A 59-year-old man suddenly becomes unwell on the oncology unit. He is receiving an intravenous infusion of a new chemotherapy agent as part of a clinic trial. He says he feels very lightheaded. On examination his blood pressure is 80/50 mmHg, heart rate is 120/min, oxygen saturations are 94% on air and bilateral wheeze is heard on auscultation of the chest. An ECG shows a sinus tachycardia of 120 beats per minute. The infusion is stopped, the patient is lay flat with their legs raised and an arrest call has been put out.

What should you administer immediately?

A. 0.5mL of 1:1000 intramuscular adrenaline
B. 0.5mL of 1:10000 intramuscular adrenaline
C. 0.5mL of 1:1000 intravenous adrenaline
D. 0.5mL of 1:10000 intravenous adrenaline
E. 1mL of 1:10000 intravenous adrenaline

A. 0.5mL of 1:1000 intramuscular adrenaline

Anaphylaxis - adult adrenaline dose = 500 mcg (0.5 ml of 1 in 1,000)

This patient is having an anaphylactic reaction to the infusion. Essential immediate management of anaphylaxis comprises: recognition that the patient is seriously unwell, removal of the trigger (if possible), an early call for help, assessment via the ABCDE (airway, breathing, circulation, disability, exposure) approach and administration of adrenaline.

The correct dose of adrenaline in anaphylaxis via the intramuscular route is 0.5mL of 1:1000 (1mg in 1mL/ 0.5mg in 0.5mL). Adrenaline should usually be administered via the intramuscular route as there is a lower risk of inappropriate administration, the incidence of adverse effects is rare, it is an easy method to learn and IV access is not required. Inappropriate intravenous administration risks precipitating arrhythmias, which may result in cardiac arrest. Specialists such as anaesthetists may use the intravenous route, but for the majority of practitioners the intramuscular route is the best option.

As soon as possible after or concurrently with the administration of adrenaline; high-flow oxygen and a rapid intravenous fluid challenge should be delivered. Subsequent to this, an intravenous or intramuscular antihistamine (e.g. chlorphenamine) and steroid (e.g. hydrocortisone) may be given.

Question 47 of 89

A 24-year-old male is admitted with shortness of breath. On examination his respiratory rate is 34 breaths per minute and heart rate is 125 beats per minute. His blood pressure is 120/85 mmHg. An SpO2 reading is 84%. He looks cyanotic. He has a past medical history of coeliac disease, dermatitis herpetiformis, rheumatoid arthritis and asthma. His regular medicines include dapsone, prednisolone, paracetamol and ibuprofen. He just recently completed a course of amoxicillin for a chest infection.

An arterial blood gas is performed (on 15 litres of O2):

Hb	125 g/l
pH	7.33
PaO2	58.11 kPa
PaCO2	2.48 kPa
SO2	99.7%
HCO3	12 mmol/l

What drugs is most likely implicated?

A. Dapsone
B. Amoxicillin
C. Prednisolone
D. Ibuprofen
E. Paracetamol

A. Dapsone

Dapsone + dyspnoea → methaemoglobinaemia

Several inferences should be made with regards to the blood gas. The PaO2 is appropriately high given the patient is on 15L of O2. A simple way of estimating an appropriate PaO2 is to multiple the % of inspired O2 (FiO2) by 2/3. For example, 15L of O2 driven by a non-rebreather mask delivers approximately 90% of O2. Therefore we would expect the PaO2 to be is approximately 2/3 * 90 = 60 kPa. The patient in this example has a PaO2 of 62.22 kPa. Therefore we can confidently state that gas exchange at the level of the alveoli is not the cause of the cyanosis. It is important toremember that the PaO2 is a measure of dissolved O2 in blood. Oxygen is carried in the blood in two forms: dissolved in plasma (about 2% of the total) and reversibly bound to haemoglobin (about 98% of the total). Therefore alternative causes for the cyanosis should be sought e.g. anaemia, shock, and toxic causes (e.g. methemoglobinemia).

The saturation of Hb on the ABG (SaO2) is 99.7%. This is strange as there is a large discrepancy between this and the peripheral pulse oximeter result (86%). This should alert you to consider two differentials - carbon monoxide poisoning and methemoglobinemia. The SaO2 on the blood gas machine is broken down to several components - O2Hb (the 'true' saturation of Hb with O2), COHb (carboxyhemoglobin), HHb, and MetHb. The sum of all these components produces the total saturation SaO2. In this case, the patient has a significantly high MetHb confirming the diagnosis of methemoglobinemia. The true oxygen saturation is 55.3%. This is the cause of the hypoxia and the resultant lactic acidosis.

Methemoglobinemia describes haemoglobin which has been oxidised from Fe2+ to Fe3+. Fe3+ cannot bind oxygen, and hence the oxidation dissociation curve is moved to the left. This results in tissue hypoxia. Drugs (e.g. sulphonamides, nitrates, dapsone, sodium nitroprusside, primaquine) are common causes. Dapsone is a common cause of methemoglobinemia.

None of the other agents are known to cause methemoglobinemia.

Question 48 of 89

A 64-year-old man presents to the haematology clinic with a history of back pain over the past 6 months. On systematic enquiry and examination, there are no features of spinal cord compression or cauda equina syndrome.

Blood results are as follows:

Hb	110 g/L	Male: (135-180)Female: (115 - 160)
Platelets	165 * 10 /L $_9$	(150 - 400)
WBC	6.2 * 10 /L $_9$	(4.0 - 11.0)
Na $^+$	135 mmol/L	(135 - 145)
K $^+$	4.8 mmol/L	(3.5 - 5.0)
Urea	10.8 mmol/L	(2.0 - 7.0)
Creatinine	190 µmol/L	(55 - 120)

Protein electrophoresis	Monoclonal IgG 38g/l

Given the likely diagnosis, what is the imaging modality of choice?

A. CT PET
B. No imaging required
C. Plain film skeletal survey
D. Whole body MRI
E. Whole body low-dose CT

D. Whole body MRI

Whole body MRI 1st line imaging in suspected multiple myeloma

The clinical features (e.g. back pain, anaemia, renal dysfunction), and the presence of a paraprotein make multiple myeloma the most likely diagnosis.

Whole body MRI is recommended as the first-line imaging modality in multiple myeloma. MRI is the most sensitive tool available for detecting marrow infiltration at an early stage, before bone destruction occurs, as well as offering improved detection of lesions, particularly in the axial skeleton.

Skeletal survey is incorrect. The major disadvantage of plain X-rays is the significantly lower sensitivity compared to advanced imaging. Lytic lesions are only demonstrated when at least 30-50% of the trabecular bone has been lost and around 20% of myeloma patients have no abnormal findings by plain X-ray. Plain X-rays cannot distinguish osteopenia or vertebral collapse caused by myeloma from more common causes such as early osteoporosis or corticosteroid use. For these reasons - skeletal surveys with plain X-rays are no longer recommended.

CT skeletal survey is incorrect. From a practical point of view, CT has the advantage of being quick to perform and allows patients to lie on their back, without the need to change position. CT is also helpful for visualising soft tissue involvement, assessing spinal fracture stability, depicting spinal cord and cauda equina compression (although MRI is superior for this), guiding needle biopsies and surgical interventions, and planning radiotherapy. However, MRI remains the gold standard.

FDG PET/CT is incorrect. PET may be considered for patients with newly diagnosed nonsecretory or oligosecretory myeloma and for evaluation of extramedullary disease. Although FDG PET/CT has some prognostic value when used in the initial diagnosis of myeloma, there is currently insufficient evidence to justify the routine use of FDG PET/CT in all cases of newly diagnosed myeloma.

No imaging required is incorrect. The patient most certainly has a new diagnosis of multiple myeloma and thus cross-sectional imaging is required.

Question 49 of 89

A 22-year-old man is seen in the general medical clinic. He has had several episodes of gallstones over the last three months. He is due to see the surgical team for consideration of laparoscopic cholecystectomy but he was also referred to the medical team due to anaemia. He has needed blood transfusions in the past when he has become tired and found to be anaemic. He was referred for an appointment with a haematologist but was unable to attend and therefore discharged from the clinic without seeing the consultant. He takes no regular tablets. He is unaware of any family history of significant medical problems, but does think that his mother used to turn yellow when slightly unwell, but thinks that this has never been a serious medical problem. He also knows that his father, to whom he is estranged, had some medical problem with his blood. His observations are normal as is his examination apart from conjunctival pallor and 2cm of a palpable spleen.

Blood tests:

Hb	110 g/l
MCV	84.1 fl
MCHC (mean corpuscular haemoglobin concentration)	391 g/l
Platelets	389 * 10 /l9
WBC	8.2 * 10 /l9
Na +	138 mmol/l
K +	4.8 mmol/l
Urea	5.2 mmol/l
Creatinine	85 µmol/l
Bilirubin	22 µmol/l
ALP	42 u/l
ALT	15 u/l

What is the most likely underlying diagnosis?

A. Sickle cell disease
B. Pernicious anaemia
C. Glucose-6-phosphate dehydrogenase (G6PD) deficiency
D. Thalassaemia
E. Hereditary spherocytosis

A. Hereditary spherocytosis

Hereditary spherocytosis is an explanation for anaemia in recurrent gallstones

This young man has a history of anaemia with recurrent gallstones. This implies that there has been destruction of red blood cells and the subsequent rise in bilirubin has provoked the formation of gallstones. The presence of normocytic anaemia with elevated MCHC further supports a diagnosis of spherocytosis. The family history of potentially Gilbert's syndrome would raise the possibility of the patient having this diagnosis as well, which would increase the possibility of gallstones in spherocytosis. The presence of splenomegaly supports this diagnosis. Diagnosis would be confirmed with microscopy and osmotic fragility testing. Pernicious anaemia would be macrocytic and would not be associated with haemolysis. Sickle cell disease would clinically have severe haemolytic normocytic anaemia, recurrent infections and vaso-occlusive phenomena. The absence of such episodes and age of presentation makes sickle cell disease unlikely. In addition, it is screened for in the UK and routinely diagnosed in the neonatal setting. G6PD is a X-linked cause of haemolytic anaemia triggered commonly by drugs causing acute episodes of haemolysis. The absence of a neonatal history of jaundice and demographic risk factors, as well as no trigger makes this less likely, as does the presence of splenomegaly. Thalassaemia would not cause splenomegaly or gallstones

Question 50 of 89

A 32-year-old woman is on her second cycle of chemotherapy for breast cancer. She has developed two days of watery diarrhoea and vomiting. She has occasional chills and feels generally weak. She has not travelled abroad and there are no ill contacts. Her husband is a farmer and together they attended a farm foods show which hosted local cheese and meats in the local region.

On examination, she has a soft abdomen that is mildly tender in the right iliac fossa. Her mucosa is dry and she is achy in muscle groups throughout her body.

Hb	110 g/l	Na +	138 mmol/l
Platelets	348 * 10 /l 9	K +	3.9 mmol/l
WBC	2.4 * 10 /l 9	Urea	4.3 mmol/l
Neuts	0.7 * 10 /l 9	Creatinine	76 µmol/l
Lymphs	1.4 * 10 /l 9	CRP	96 mg/l

Stool specimen	gram-positive bacillus

What is the causative organism in this case?

A. Listeriosis
B. Salmonellosis
C. Shigellosis
D. Clostridium perfringens poisoning
E. Staphylococcus aureus poisoning

A. Listeriosis

Neutropenic patients should avoid soft cheese due to risk of listeriosis

This is a case of neutropenic sepsis. Patients are at high risk to many organisms and it is suggested they avoid cold meats and dairy products. She had gastrointestinal symptoms and cultured a gram-positive bacillus. This immediately rules out salmonellosis and shigellosis. The gram-positive is a bacillus so cannot be *staphylococcus*. Of the two remaining, only *listeria* is found in dairy products and therefore is the likely cause of her illness

Question 51 of 89

A 22-year-old woman awaiting stem cell transplant for acute myeloid leukaemia has become unwell over the past three days and arrives with a Glasgow Coma Scale of 3 in the emergency department. Her boyfriend who is present with her reports that she has had severe headaches and been sitting in bed but gradually became more confused. She was struggling to breathe and began to seize causing him to call the ambulance.

On examination, she is intubated and has bilateral coarse crepitations. Her pupils are reactive but she is sedated and does not respond to pain. There is papilloedema bilaterally and haemorrhages are seen in the left eye. She is warm and well perfused but her left arm is cold and pale. She is transferred to the intensive care unit.

Hb	120 g/l
Platelets	350 * 10 /l9
WBC	105 * 10 /l9
Troponin	10,876 (normal range <20)
CRP	56 mg/l
Chest x-ray	bilateral pulmonary infiltrates, small left sided effusion

What medical treatment will correct her underlying pathology should be initiated?

A. Intravenous antibiotics
B. High dose corticosteroids
C. Leukapheresis
D. High dose chemotherapy
E. Venesection

C. Leukapheresis

Leukapheresis can be used to acutely lower the white cell count in AML

This lady presents with pulmonary oedema and neurological dysfunction on a background of hyperleukocytosis. This is leukostasis where the blood is thick in white cells. The treatment is to rapidly remove white cells. This can be done by leukapheresis of the blood and removing the white cells which is called leukapheresis and this is the most appropriate answer. Venesection and high dose chemotherapy are less effective ways of achieving a lower white cell count.

Corticosteroids and antibiotics will not help.

Question 52 of 89

A 64-year-old woman is referred by her GP to the haematology clinic with an incidental lymphocytosis noted first four months prior. On questioning, she reports that she has been feeling more fatigued over the past few months and has begun to experience episodes of early satiety, both of which she attributed to looking after her two grandchildren. She is otherwise well and denies any fevers, night sweats or unusual bruising.

On examination, she is slim with an unremarkable cardiovascular and respiratory exam. There is a fullness in the left upper quadrant which moves with inspiration but no lymphadenopathy.

Her blood tests are shown below:

Hb	140 g/L	Male: (135-180)Female: (115 - 160)
Platelets	200 * 10 /L 9	(150 - 400)
WBC	30.1 * 10 /L 9	(4.0 - 11.0)
Neuts	4.0 * 10 /L 9	(2.0 - 7.0)
Lymphs	25.0 * 10 /L 9	(1.0 - 3.5)
Mono	0.8 * 10 /L 9	(0.2 - 0.8)
Eosin	0.3 * 10 /L 9	(0.0 - 0.4)
Blood Film	lymphocytosis with smudge cells	

Ultrasound abdomen confirms splenomegaly.

Chromosomal analysis is performed to guide further management.

Given the likely diagnosis, which of the following chromosomal findings would suggest a poor prognosis?

A. >2% mutation immunoglobulin heavy chain variable region
B. Deletion of 13q
C. Deletion of 17p
D. < 20% CD38 expression
E. TP53 - wild-type

C. Deletion of 17p

del 17p is associated with a poor prognosis in CLL

This patient has chronic lymphocytic leukaemia (CLL) demonstrated by a history of significant lymphocytosis for > 3 months and splenomegaly with a blood firm showing smudge cells. A bone marrow aspirate is not required in all CLL patients but may be indicated in patients with anaemia and/thrombocytopenia to confirm whether these may be attributed to marrow infiltration or splenic destruction.

The deletion of 17p is associated with a poor prognosis in CLL. All other options are markers of good prognosis. Note that a wild type TP53 refers to an unmutated form, however, a mutated TP53 is, by contrast, a bad prognostic marker.

Question 53 of 89

A 54-year-old alcoholic man with chronic hepatitis C is taken to the emergency department by the police. There it was noted that the man had blisters and crusted lesions on his face and lower arms.

Laboratory tests showed elevated plasma porphyrins and elevated uroporphyrin I in the urine, and isocoproporphyrin in the faeces. Biopsy of the skin lesion showed subepidermal blisters with minimal inflammation, marked solar elastosis, thickening of the vessel wall in the papillary dermis and 'caterpillar bodies' in the roof of the blister.

Which of the following is the most likely diagnosis?

A. Acute intermittent porphyria
B. Delta-aminolevulinic acid dehydrase deficiency
C. Erythropoietic protoporphyria
D. Hereditary coproporphyria
E. Porphyria cutanea tarda

A. Porphyria cutanea tarda

Porphyria cutanea tarda causes chronic blistering and crusting skin lesions on sun-exposed skin. Precipitating factors: iron (even if normal amounts), oestrogen and alcohol use, and chronic hepatitis C infection.

The findings on skin biopsy can help with the diagnosis but are not very specific (the 'caterpillar bodies' are essentially clumps of basement membrane material). The key to the diagnosis is the presentation and porphyrin analysis

Question 54 of 89

A 68-year-old female, presents with lethargy and anorexia. She underwent a partial gastrectomy 3 years ago for bleeding gastric ulcer. Her blood results showed:

Hb	90 g/l
MCV	109 fL
Platelets	60 * 10 /l9
WBC	3.5 * 10 /l9
Blood film	Oval erythrocytes, macrocytic erythrocytes, hypersegmented neutrophils, low platelets and basophilic stippling

What is the underlying diagnosis?

A. Sideroblast anaemia
B. Spur cell haemolysis
C. Vitamin-B12 deficiency
D. Thalassaemia
E. Myelodysplasia

C. Vitamin-B12 deficiency

Gastrectomy is defined as partial when a part of the stomach is removed surgically and as total when the entire stomach is removed. Typical indications are gastric cancer, recurrent gastric ulcers, large duodenal perforations, bleeding gastric ulcers and gastrointestinal stromal tumours. Malnutrition is less common after partial than after total gastrectomy, but the key nutritional deficiencies are iron-deficiency anaemia, calcium deficiency, and vitamin B12 deficiency. However, the hypersegmented neutrophils are strongly suggestive of B12 deficiency.

Hypersegmented neutrophils are hallmarks of B12 deficiency in multiple choice questions

Question 55 of 89

A 19-year-old man is referred to see you as he has been suffering from recurrent epistaxis and he tells you that if he accidentally injures himself the wound bleeds for a long time.

Initial blood tests are as follows:

Hb	101 g/l
MCV	79 fl
Platelets	298 * 10 /l9
WBC	9.2 * 10 /l9
Bleeding time	Prolonged
PT	14 seconds
APTT	28 seconds
LDH	290 u/l (240 - 480 u/L)
Factor VIIIc	low
Ristocetin platelet aggregation test	Impaired aggregation

What is the single most likely diagnosis?

A. Haemophilia A
B. Haemophilia B
C. Haemophilia C
D. Idiopathic Thrombocytopenic Purpura (ITP)
E. von Willebrand's disease

A. von Willebrand's disease

This patient has a coagulopathy and normal platelets, which immediately excludes ITP. Factor VIIIc is low, meaning that the diagnosis must either be haemophilia A or von Willebrand's disease. In this patient two things favour the diagnosis of von Willebrand's over haemophilia A. Firstly, the bleeding time is prolonged and secondly, the platelet aggregation is impaired in response to ristocetin. Both of these would be normal in haemophilia.

question 56 of 89

A 21-year-old girl complains of easy bruising. She has menorrhagia for which she is being investigated by the gynaecology team. She takes no regular medications. Her father had prolonged bleeding after a tooth extraction.

Blood tests show:

Hb	110 g/L
MCV	74 fL
WBC	4.2 x 10^9/L
Platelets	135 x 10^9/L
APTT	45 seconds
INR	1.0

What is the most likely diagnosis?

A. Haemophilia B
B. Anti-thrombin III deficiency
C. Von Willebrand's disease
D. Immune thrombocytopenia
E. Haemophilia A carrier

C. Von Willebrand's disease

Von-Willebrand's disease is an autosomal dominant condition (chromosome 12) that leads to a mild-moderate bleeding tendency. The prevalence of clinically significant cases is 1 in 10,000. Factor VIII is bound to vWF, which protects it from breakdown, so deficiency of vWF can lead to low factor VIII levels and a prolonged APTT. In this case the patient also has a microcytic anaemia, which would be in keeping with iron deficiency anaemia secondary to menorrhagia. Haemophilia B (Factor IX deficiency) is X-linked so does not affect females. Haemophilia A (Factor VIII deficiency) is also X-linked. Most carriers are asymptomatic. The APTT is normal in ITP. Anti thrombin III deficiency is a pro thrombotic condition.

Question 57 of 89

A 72-year-old woman with breast cancer presents with a swollen, painful left calf. She is known to have metastases in the vertebral bodies and is taking denosumab as prophylaxis. A Doppler ultrasound is arranged which shows a proximal deep vein thrombosis on the left side. This is her first episode of venous thromboembolism.

What is the most appropriate management?

A. Warfarin for 3 months
B. Warfarin for 6 months
C. Low-molecular weight heparin for 3-6 months
D. Direct oral anticoagulants (DOACs) for 3-6 months
E. Aspirin 300 mg for 3 months

C. Low-molecular weight heparin for 3-6 months

Cancer patients with VTE - 6 months of a DOAC

Question 58 of 89

A 23-year-old gentleman reviewed by the on-call medical team after having become unwell in the radiology department. He was undergoing a contrast-enhanced MRI of the small bowel to investigate ongoing diarrhoea. He has no other past medical history and takes no regular medications. He is not known to have any allergies.

Ten minutes ago he started feeling his throat closing up and became very anxious. This was shortly after having been given a contrast agent by IV injection. The medical emergency team arrives quickly and note him to be tachycardia at 132/min and hypotensive with a blood pressure of 82/35mmHg. He also has a widespread erythematous rash over his body and feels itchy. He is treated for anaphylactic shock.

Which blood tests could confirm anaphylaxis?

A. Histamine levels

B. Mast cell tryptase immediately and repeat within one to two hours
C. Mast cell tryptase immediately and repeat within 12 hours
D. Mast cell tryptase immediately and repeat within 24 hours
E. Serum immunoglobulins

B. Mast cell tryptase immediately and repeat within one to two hours

Anaphylaxis - serum tryptase levels rise following an acute episode

The correct answer is mast cell tryptase immediately and repeat within one to two hours. Tryptase levels are the most specific marker for an anaphylactic reaction. Tryptase is a marker of mast cell activation. Serum concentration is normally very low and will quickly return to normal, which is why an immediate sample can be useful but should never delay treatment. Tryptase levels increase to maximum levels over 30 minutes from the onset of symptoms, then peaks at one to two hours. The half-life of tryptase is roughly two hours and may return to normal within six to eight hours. This means that a sample at 12 or 24 hours may not confirm anaphylaxis. Histamine levels can be elevated in various conditions, including simple allergic reactions and does not help to distinguish anaphylaxis. Immunoglobulins are elevated in cell-mediated immune responses.

Question 59 of 89

A 43 year old man is brought to the Emergency Department by his wife. She has become worried about him in the past six months as he has become increasingly confused, aggressive and depressed. He has also lost 4kg in weight in this time and has developed severe, intermittent abdominal pain and diarrhoea, and complains of constant headaches.

Examination reveals a normal cardiorespiratory examination but the abdomen is diffusely tender and there is tender fullness in the right upper quadrant. Neurological examination reveals minor hypotonia throughout and there are bilateral radial nerve palsies as well as a left common peroneal nerve palsy. Sensation appears intact. He is alert but confused with an abbreviated mental test score of 7/10.

Blood tests reveal:

Sodium	136mmol/L	ALP 135U/L Haemoglobin 79g/L
Potassium	5.1mmol/L	AST 265U/L MCV 101fL
Urea	7.1mmol/L	ALT 298U/L White cells 9.4x10^9/L
Creatinine	102µmol/L	GGT 197U/L Neutrophils 5.6x10^9/L
CRP	10mg/L	Bilirubin 12µmol/L Lymphocytes 3.1x10^9/L
Calcium (corr)	2.34mmol/L	Eosinophils 0.1x10^9/L
Phosphate	0.56mmol/L	Basophils 0.6x10^9/L
Magnesium	0.76mmol/L	
Glucose	3.8mmol/L	

The blood film shows anaemia with a dimorphic picture, significant reticulocytosis and high basophil numbers with cytoplasmic stippling.

The patients wife tells you all the symptoms coincided with the patient starting a new job as a loading crane driver at a scrap-yard.

What is the most likely diagnosis?

A. Chronic alcoholism
B. Acute promyelocytic leukaemia
C. Porphyria cutanea tarda
D. Chronic lead poisoning
E. Minimata disease

D. Chronic lead poisoning

Abdominal pain, constipation, neuropsychiatric features, basophilic stippling → lead poisoning

The above vignette gives a classical description of chronic lead poisoning, also known as plumbism or Devon colic. The typical signs are gastrointestinal and neuropsychiatric and include severe, colicky abdominal pain with weight loss and diarrhoea and a metallic taste in the mouth. Insidious neurological signs include headache, difficulty concentrating, subtle personality changes including aggression and difficulty sleeping, dyspraxia and cerebellar signs. The pathognomonic hallmark of chronic lead poisoning is a radial nerve palsy, or wrist drop, although many peripheral nerve palsies may be seen. Patients may also have a bluish discolouration to their skin and a blue line on the gums, known as Burtons line, is very rarely seen. Biochemically, a hepatitic picture can be seen and also occasionally renal impairment due to proximal renal tubular failure, particularly with increased excretion of phosphate and glucose and acidification of the urine. However, most notable is the haematological impact of lead poisoning with a pronounced anaemia, often with dimorphic picture and reticulocytosis due to arrest of the haem biosynthetic pathway. Basophilia with stippling is seen and bone marrow trephine may show ring sideroblasts.

Blood lead levels do not correlate well with total lead burden and the diagnosis may be missed due to incorrect assay or test selection. Since lead is concentrated in red cells, whole blood levels of lead rather than plasma must be analysed, however testing for enzymes affected by lead is more reliable than whole lead levels alone, and hence erythrocyte zinc protoporphyrin (ZPP) levels and delta aminolevulinic acid dehydratase (ALAD) are more reliable. These, coupled with skeletal analysis and blood lead levels are used to test for occupational lead poisoning. The term metal fume fever is used to describe many syndromes of heavy metal and metal oxide poisonings, often in gantry crane drivers who deposit scrap metals in furnaces from above. Many scrap metals are coated in lead based paints leading to occupational lead toxicity syndromes.

Lead poisoning is clinically similar to porphyria and pathologically an interruption in haem pathways are seen in both. The classical hallmark of porphyria cutanea tarda is a blistering photosensitive rash that is not present in this case. Chronic alcoholism may cause B12 deficiency and hence a megaloblastic anaemia and confusion but it is unlikely to cause this constellation of neuropathies and does not explain the basophilic stippling. There is no mention of the patients alcohol habits either. An acute promyelocytic leukaemia presents with non specific symptoms and infections. It may present with sudden onset neurological symptoms only if there is a spontaneous intracranial bleed, in which case the outlook is bleak. It is unlikely here. Minimata disease is an acute mercury toxicity and presents with neuropsychiatric symptoms and rapid death. It would not be expected to cause the haematological picture seen here.

Question 60 of 89

A 30-year-old man with acute myeloid leukaemia receives a bone marrow transplant from a matched unrelated donor.

7 days post-transplant, he develops a non-blanching purpuric rash over both legs. The rash is not itchy or scaly.

His heart rate 98/min and his blood pressure is 124/72 mmHg. His temperature is 37.3 ºC and he is otherwise asymptomatic.

Blood tests are as follows:

Hb	89 g/l	Na^+	138 mmol/l
Platelets	9 * 10 /l 9	K^+	3.2 mmol/l
WBC	0.8 * 10 /l 9	Urea	4 mmol/l
Neuts	0.2 * 10 /l 9	Creatinine	62 µmol/l
CRP	10 mg/ll		

What is the most likely cause for the rash?

A. Fungal infection
B. Graft vs host disease
C. Henoch Schonlein purpura
D. Meningitis
E. Thrombocytopenia

E. Thrombocytopenia

This man is on day 7 of his transplant and his blood counts are starting to drop, including his platelets. Thrombocytopenia typically causes small purpuric spots that are otherwise asymptomatic.

A fungal infection is possible in an immunosuppressed patient but would be more likely to cause an itchey or painful rash with a different appearance.

Graft vs host disease typical causes a dry scaley rash and develops when the patients immune system is starting to recover, around 2 weeks after transplant. Henoch Schonlein purpura typically affects children and is associated with a glomerulonephritis. It is immune mediated so unlikely in someone who is still profoundly immunosuppressed.

Meningitis can cause a non-blanching purpuric rash but the patient would be symptomatic with headache, fever, meningism and a CRP rise.

Question 61 of 89

A 45-year-old woman is admitted with chest pain three days after binge drinking 4 bottles of wine and 250mL of spirits.

She has a background of alcohol excess and systemic lupus erythematosus. She has a family history of G6PD deficiency on her father's side.

On examination, she is hemodynamically stable but jaundiced. She has pain on palpation over her right upper quadrant. Chest is clear and heart sounds are normal.

Hb	67 g/L	Male: (135-180)Female: (115 - 160)
Platelets	130 * 10 /L 9	(150 - 400)
WBC	9.8 * 10 /L 9	(4.0 - 11.0)
Bilirubin	72 µmol/L	(3 - 17)
ALP	360 u/L	(30 - 100)
ALT	70 u/L	(3 - 40)
Amylase	67 u/L	(40-140)
Triglycerides	5.8 mmol/L	(<1.7)
LDH	1095 IU/L	(105-333)
Direct anti-globulin test	Negative	

Spur cells and acanthocytes are present on blood film.

Given the likely diagnosis, what treatment from the following options would be most appropriate?

A. Abstinence from alcohol
B. Ascorbic acid
C. Intravenous fluids
D. Liver transplantation
E. Oral prednisolone

A. Abstinence from alcohol

Zieve syndrome usually resolves with abstinence from alcohol

This patient is presenting with jaundice, hyper-triglyceridaemia, and Coomb's negative haemolytic anaemia due to alcoholism and recent binge. The diagnosis here is Zieve syndrome - a rare complication of alcohol excess that is thought to be caused by changes in the red cell membrane due to the transient hyperlipidaemia seen following alcohol binges.

Abstinence from alcohol is the correct answer. Treatment of Zieve syndrome is with supportive management with blood transfusion and abstinence from alcohol. In rare cases, plasmapheresis may be required.

Ascorbic acid is incorrect. This can be used in the treatment of G6PD deficiency, however, this patient does not have this condition. G6PD is inherited in an X-linked recessive pattern and so a female child of a male with G6PD deficiency would be a carrier. G6PD deficiency would also not explain the cholestatic liver function tests and rise in triglycerides.

Intravenous fluids is incorrect. IV fluids would be indicated if there was evidence of pancreatitis, or if the patient was hemodynamically unstable. In this case, the patient has severe anaemia, but in the context of haemodynamic stability IV fluids would be inappropriate as they would lead to a further drop in Hb concentration via a dilutional effect.

Liver transplantation is incorrect. The King's college criteria state that for non-paracetamolinduced liver injury an INR >6.5 or three from; bilirubin of greater than 600µmol/L, an interval of >7 days between jaundice and encephalopathy, age <10 years or >40, INR >3.5, drug-induced or indeterminate cause of hepatitis, would be poor prognostic features and indicate liver transplant may be required.

Oral prednisolone is incorrect. This would be an option for alcoholic hepatitis or autoimmune haemolytic anaemia. In this case, the direct coomb's test is negative indicating that the low haemoglobin is not a result of immune-mediated haemolysis.

Question 62 of 89

A 52-year-old male was seen in the rapid access Transient Ischaemic Attack (TIA) clinic. He presented to his GP with new onset left leg and arm weakness three days ago. The weakness lasted for 90 minutes and fully resolved with no residual defect. He had a past medical history of hypertension, obstructive sleep apnoea and a left sided deep vein thrombosis eight years ago. His medication comprised ramipril 5mg OD. He smoked ten cigarettes per day and did not drink alcohol.

On examination, he had obvious truncal obesity and a flushed complexion. Blood pressure was 128/82 mmHg, heart rate 78/min, respiratory rate 16/min and oxygen saturations 99% on air. Cardiovascular examination revealed a regular pulse and nil else of note. Respiratory and gastrointestinal examination were normal, though examination of the abdomen was somewhat limited by the presence of truncal obesity. Neurological examination was unremarkable with normal cranial nerve, fundoscopy and peripheral neurological examinations.

Initial investigations revealed the following results:

Hb	191 g/l
MCV	98 fl
Hct	0.523
Platelets	502 * 10 /l
WBC	14.0 * 10 /l
Neutrophils	86%
Lymphocytes	10%
Monocytes	4%
HbA1c	43 mmol/mol
Fasting cholesterol	5.6 mmol/l

ECG: 76bpm normal sinus rhythm no other abnormality Chest x-ray: unremarkable

24 hr ECG: no arrhythmia seen

Echo: normal systolic function, mild aortic stenosis with pressure gradient of 42mmHg

CT head: normal intracranial appearances, no evidence of mass shift, space occupying lesion or haemorrhage

What is the most appropriate next investigation most likely to lead to the underlying diagnosis?

A. Cardiac catheterization
B. Testing for presence of JAK2 mutation
C. Bone marrow biopsy
D. Radioisotope scanning of circulating blood volumes
E. MRI scanning of the abdomen

B. Testing for presence of JAK2 mutation

This gentleman has polycythaemia rubra vera (PRV). Whilst all of the above investigations may facilitate the diagnostic process, this question asks *What is the most appropriate next investigation most likely to lead to the underlying diagnosis?*. Testing for JAK2 mutation has widely surpassed the use of radioisotope scanning and is diagnostic of PRV. It is, therefore, the best option

. Question 63 of 89

An 18-year-old man presents to the emergency department with headache and discolouration of his face. He has a past medical history of NADH methaemoglobinaemia reductase deficiency. He does not smoke cigarettes or drink alcohol.

His observations are as follows: heart rate 94 beats per minute, oxygen saturations 85% on room air, blood pressure 120/77 mmHg, respiratory rate 20/minute and temperature 37ºC.

Despite administration of 15L of oxygen via a non-rebreathe mask, the Sp2 remains 85%. On examination, he appears cyanotic. The cardiorespiratory examination is unremarkable.

Plain radiography of the chest is normal.

ABG results:

pH	7.36	(7.35-7.45)
pO2	11 kPa	(11-13)
pCO2	4 kPa	(4-6)
Hb	136 g/L	(135-175)
Lactate	2.0 mmol/L	(0-2)
MetHb	30%	(0.2-0.6)

What is the most appropriate treatment?

A. Antibiotic treatment
B. Ascorbic acid
C. Methylene blue
D. No specific treatment
E. Thrombolysis

B. Ascorbic acid

Ascorbic acid is the treatment of choice for NADH methaemoglobinaemia reductase deficiency

Ascorbic acid is correct. This patient has NADH methaemoglobinaemia reductase deficiency, which is a congenital form of methaemoglobinaemia. IV methylene blue is ineffective in this form of the disease. He has evidence of a deterioration in his condition requiring treatment with a methaemoglobin level >= 30%, cyanosis, headache and a low peripheral oxygen saturation level despite normal arterial oxygen content. Ascorbic acid has been shown to be effective in the treatment of this disorder.

Methylene blue is incorrect. This is not effective in this form of methaemoglobinaemia but is the typical treatment for acquired causes.

Antibiotics treatment is incorrect. The patient is apyrexial with a normal chest x-ray and there no history of cough or shortness of breath. An infection seems unlikely.

No specific treatment is incorrect. Symptoms and a methaemoglobin level >= 30% are both indications for treatment in this condition.

Thrombolysis is incorrect. While a pulmonary embolism is a plausible cause of low peripheral oxygen saturations with a normal examination and chest x-ray, the clinical context otherwise does not suggest this as the diagnosis. Furthermore, the typical indication for thrombolysis in pulmonary embolism is hypotension, which is absent in this case.

Question 64 of 89

A 34 year old man is admitted under the medics from the emergency department with central abdominal pain and vomiting not responding to IV morphine. He had been seen by the surgeons earlier in the day as an acute abdomen, but the CT scan they did of his abdomen and pelvis revealed no abnormalities, and his bloods suggested no surgical cause of the abdominal pain.

You note that the patient has presented to the emergency department five times in the past two years with similar problems, and no cause has ever been found, with the patient being discharged one or two days later with analgesia.

On examination his abdomen is generally tender with evidence of voluntary guarding. On further examination you note that he has a rash on the back of his hands, neck and cheeks. This rash consists of several small fluid filled bullae.

His past medical history includes depression - for which he is taking citalopram, and one short psychiatric inpatient stay for an episode of psychosis.

What is the most likely diagnosis?

A. Erythropoietic protoporphyria
B. Porphyria cutanea tarda
C. Hereditary coproporphyria
D. Acute intermittent porphyria
E. Congenital erythropoietic porphyria

D. Acute intermittent porphyria

The porphyrias are a group of rare inborn errors of metabolism caused by abnormalities of enzymes involved in the biosynthesis of haem, resulting in overproduction of intermediate compounds called porphyrins.

Three patterns of symptoms occur clinically with porphyrias:

1) Neurovisceral - neuropathy, epilepsy, psychiatric disorders, abdominal, vomiting, constipation

2) Photosensitive - bullous eruption in sun exposed areas

3) Haemolytic

Neurovisceral only	Photosensitive only	Mixed
Acute intermittent porphyria	Porphyria cutanea tarda	Variegate porphyria
Aminolaevulinic acid dehydrogenase porphyria	Congenital erythropoietic porphyria	Hereditary coproporphyria
-	Erythropoietic protoporphyria	-

This patient has both photosensitive symptoms; his rash, and neurovisceral symptoms; abdominal pain and previous psychiatric history. The only mixed presentation porphyria given as a possible answer is hereditary coproporhyria, hence it is the correct answer.

Question 65 of 89

A 62 year old man is attending his GP as part of regular screening. He has a past medical history significant only for hypertension for which he has been taking amlodipine for many years. He reports no specific symptoms of note and is still working full time as an accountant. His blood pressure on this visit is 132/78mmHg and his urinalysis shows no abnormalities. Blood tests were taken for routine monitoring.

Hb	13.8 g/dl
Platelets	362 * 10 /l9
WBC	6.3 * 10 /l9
Na +	142 mmol/l
K +	4.2 mmol/l
Urea	4.6 mmol/l
Creatinine	84 µmol/l
Adjusted calcium	2.41mmol/l
Lactate Dehydrogenase	300 IU/l
Albumin	36 g/l
Globulin	52 g/l

An abnormal protein band is detected during analysis and the GP arranges for further review with the Haematologist at the hospital. Further investigations are undertaken:

Monoclonal paraprotein 18g/l

Bone Marrow examination showing 6% plasma cells Skeletal Survey showing no abnormalities

What is the diagnosis?

A. Multiple Myeloma
B. Hodgkins Lymphoma
C. Waldenstrom's macroglobulinemia (lymphoplasmacytic lymphoma)
D. Asymptomatic Myleoma
E. Monoclonal gammopathy on uncertain significance (MGUS)

E. Monoclonal gammopathy on uncertain significance (MGUS)

Monoclonal gammopathy on uncertain significance (MGUS) is a condition where an abnormal paraprotein is found in the blood, often during routine testing. It is on a spectrum that includes multiple myeloma but at the MGUS stage no treatment is indicated. The level of serum paraprotein is lower than in myeloma (under 30g/l) and the number of plasma cells in the bone marrow is also lower (less than 10%). The condition is totally asymptomatic. There is an annual risk of progression to myeloma of 1-2% and as such annual surveillance is recommended.

The diagnosis of MGUS can be made in patients who fulfil the following criteria –

- A monoclonal paraprotein band lesser than 30 g/L (< 3g/dL)
- Plasma cells less than 10% on bone marrow examination
- No evidence of bone lesions,anemia, hypercalcemia, or renal insufficiency related to the paraprotein
- No evidence of another B-cell proliferative disorder

In terms of myeloma this can be classified as below, taking note of both asymptomatic and symptomatic myeloma:

Symptomatic myeloma:

- Clonal plasma cells >10% on bone marrow biopsy or (in any quantity) in a biopsy from other tissues (plasmacytoma)
- A monoclonal protein (paraprotein) in either serum or urine (except in cases of true nonsecretory myeloma)
- Evidence of end-organ damage felt related to the plasma cell disorder (related organ or tissue impairment, ROTI, commonly referred to by the acronym 'CRAB'):
- hyperCalcemia (corrected calcium >2.75mmol/L)
- Renal insufficiency attributable to myeloma
- Anemia (hemoglobin <10 g/dL)

- Bone lesions (lytic lesions or osteoporosis with compression fractures)

Asymptomatic (smoldering) myeloma:

- Serum paraprotein >30 g/L AND/OR
- Clonal plasma cells >10% on bone marrow biopsy AND
- NO myeloma-related organ or tissue impairment

Question 66 of 89

You are the medical doctor on an acute admission unit. An elderly female with recently diagnosed ovarian cancer, who is on day 8 of cisplatin chemotherapy presents with a temperature of 39°C, tachycardia at 130 bpm, blood pressure 128/68 mmHg, respiratory rate 14/min, sats 98% on room air.

She is complaining of abdominal pain and has been vomiting today. Her bloods and blood culture have been sent and you are awaiting the results. Her chest x-ray was normal and urine dipstick clear.

What is the most appropriate antibiotic therapy to start her on?

A. IV benzylpenicillin + flucloxacillin
B. IV augmentin
C. IV ciprofloxacin
D. IV piperacillin/tazobactam
E. IV piperacillin/tazobactam + gentamicin

D. IV piperacillin/tazobactam

Piperacillin with tazobactam (Tazocin) is the empirical antibiotic of choice for neutropenic sepsis

The patient has presented with a systemic inflammatory response syndrome (SIRS) - tachycardia, pyrexia. The most likely cause for this would be an infection and she should be treated as a probable neutropenic sepsis until proven otherwise. Neutropenic sepsis tend to present on day 7- 10 post-chemotherapy. Given that we are unable to determine the source of infection, she should ideally be treated with a broad-spectrum antibiotic such as IV piperacillin/tazobactam. You would also normally add a stat dose of gentamicin but given that she was on cisplatin therapy, the combination of these 2 medications would be highly nephrotoxic and should be witheld until neutropenic sepsis is confirmed and renal function results are available.

Question 67 of 89

An 82-year-old man presents with a 6-month history of 8 kg of weight loss, fevers, and night sweats. On examination, he has cervical and inguinal lymphadenopathy and a mildly enlarged spleen. Blood results are as follows:

Hb	101 g/L	Male: (135-180)Female: (115 - 160)
Platelets	142 * 10 /L 9	(150 - 400)
WBC	40.5 * 10 /L 9	(4.0 - 11.0)
Neuts	2.8 * 10 /L 9	(2.0 - 7.0)
Lymphs	34 * 10 /L 9	(1.0 - 3.5)

Given the most likely diagnosis, what would be the diagnostic investigation of choice?

A. Bone marrow aspirate / trephine
B. CT neck, chest, abdomen and pelvis
C. Flow cytometry (immunophenotyping)
D. HIV test
E. Lymph node biopsy

C. Flow cytometry (immunophenotyping)

CLL - immunophenotyping is investigation of choice

The diagnosis is most certainly CLL which is the most common cause of significant lymphocytosis in an elderly patient.

CLL has a very specific immunophenotype and can thus be diagnosed on **flow cytometry** of peripheral blood.

Although a **bone marrow examination** and **lymph node biopsy** would also likely be diagnostic of CLL, they are invasive tests and thus should only be performed if immunophenotyping was nondiagnostic. A **CT neck, chest, abdomen, and pelvis** would be useful for staging, however, it would not be particularly helpful in making the diagnosis.

HIV can cause an early mild lymphocytosis (due to a CD8 lymphocytosis) however it is most often associated with lymphopaenia. The patient's age and presence of a marked lymphocytosis favour the diagnosis of CLL.

Question 68 of 89

A 39-year-old woman presents with anaemia secondary to menorrhagia. She has a past medical history of fibroids, rhesus incompatibility in pregnancy 9 years ago, and Hodgkin's lymphoma which has been in remission for 21 years.

She has a severe penicillin allergy. Her investigations show the following:

Hb	59 g/L	Female: (115 - 160)
Platelets	350 * 10 /L 9	(150 - 400)
WBC	4.7 * 10 /L 9	(4.0 - 11.0)

Pregnancy test: negative.

The consultant asks for you to prescribe her 2 units of packed red cells.

What requirement would apply to this patient's blood transfusion?

A. Give antihistamines with packed red cells
B. No special requirements
C. Use CMV negative packed red cells
D. Use irradiated packed red cells
E. Use washed red blood cells

D. Use irradiated packed red cells

Hodgkin lymphoma is an indication for irradiated blood products

The patient would require **irradiated** packed red cells due to her history of Hodgkin's lymphoma. Irradiated blood is treated with radiation which prevents lymphocyte division which can cause transfusion-associated graft-versus-host disease (TA-GvHD). A history of Hodgkin's lymphoma increases the risk of this reaction and hence all red cell, platelet, and granulocyte concentrates should be irradiated for life.

No special requirements is incorrect because this patient requires irradiated blood as explained above.

There is no indication for **antihistamines** to be given with packed red cells for this patient. Like all patients, she should be monitored for any signs of allergic reaction regularly during her transfusion but only treated for this if this occurs.

CMV negative blood is blood collected from donors who have tested negative for CMV IgG antibodies. CMV can cause serious infections in immunocompromised patients and is the most common cause of congenital infection cause developmental abnormalities in the UK. CMV negative blood should be used for intrauterine transfusions, neonates up to 28 days post expected delivery and for transfusions during pregnancy. There is no indication for CMV negative blood in this patient.

Washed red cells are used when there are recurrent and/or severe allergic reactions to transfusion and can also be used for IgA deficient patients with anti-IgA antibodies. This patient has a penicillin allergy but not to blood products.

Question 69 of 89

A 47-year-old woman with a background of obesity is admitted with left leg cellulitis. Blood cultures are taken and she is commenced on IV flucloxacillin.

Shortly afterwards, you are asked to see her as she has become itchy and light-headed in the last 5 minutes. Observations are as follows.

Respiratory rate 30 breaths per minute Oxygen saturations 90% on air Heart rate 127 beats per minute Blood pressure 75/42mmHg Temperature 36.8°C

Chest examination reveals widespread wheeze. The rest of the examination is unremarkable, other than her cellulitis which has not spread beyond the markings.

What statement is correct concerning the suspected diagnosis?

A. An elevated tryptase that remains at the same level after 2 hours supports the diagnosis
B. High tryptase now confirms the diagnosis
C. Normal tryptase at 24 hours excludes the diagnosis
D. Normal tryptase now excludes the diagnosis
E. Normal tryptase now that becomes elevated in 2 hours supports the diagnosis

A. Normal tryptase now that becomes elevated in 2 hours supports the diagnosis

Anaphylaxis - serum tryptase levels rise following an acute episode

The correct answer is **normal tryptase now that becomes elevated in 2 hours supports the diagnosis**. The features of cardiovascular collapse, breathing difficulty and bronchospasm following IV antibiotics are suggestive of anaphylaxis. Tryptase is released into the blood in the context of mast cell degranulation. In anaphylaxis, it is expected to rise quickly in the first minutes to hours before gradually falling. NICE recommend it is measured at baseline and after 1-2 hours (but no later than 4). If measured at onset, it may be normal - the key finding is the elevation over the first few hours. If measured after 4 hours, it may have returned to normal.

An elevated tryptase that remains at the same level after 2 hours supports the diagnosis is incorrect. Some patients have elevated tryptase at baseline, for example in the context of systemic mastocytosis. It can also cross-react, for example with rheumatoid factors, leading to elevated levels in the absence of anaphylaxis. The lack of a change over 2 hours is more suggestive of a high baseline rather than anaphylaxis, where we would expect a significant change from onset to 2 hours.

High tryptase now confirms the diagnosis is incorrect. A single elevated measurement does not confirm the diagnosis for the reasons described above - a second sample must be taken within the next few hours to check for a change from baseline.

Normal tryptase at 24 hours excludes the diagnosis is incorrect. By 24 hours, we would expect the tryptase level to have returned to baseline, even in a significant anaphylactic episode. A baseline level and a 1-2 hour level would be much more helpful.

Normal tryptase now excludes the diagnosis. Tryptase level at the onset of symptoms may be normal as it takes at least 15 minutes (but longer in some patients) to mount a response, and we are told the symptoms have lasted 5 minutes here. A single measurement has limited utility, and we would have to repeat the sample at 1-2 hours for more useful information.

Question 70 of 89

A 17-year-old man presents to the acute medical unit with shortness of breath. He takes no regular medicines and denies the use of any recreational, herbal, or over-the-counter medications. He has not been exposed to any local anaesthetics or chemicals. On examination, his chest was clear on auscultation and he was markedly cyanotic. Despite the cyanosis, his SpO2 is 92%.

You start the patient on 15L O2 via a non-rebreather mask. Blood gas analysis results are as follows:

pH	7.28	(7.35 - 7.45)
PaO2	66.5 kPa	(10.3 - 13.3 kPa)
PaCO2	2.8 kPa	(4.7 - 6 kPa)
HCO3	22 mmol/L	(22 - 28 mmol/L)
SaO2	52%	(94-98)

The patient moved to the UK from India 6 weeks ago. His medical records are not available. However, he advises you that he has a 'blood disorder' that runs in his family.

Given the most likely diagnosis, what is the treatment of choice?

A. 4-Dimethylaminophenol
B. Ascorbic acid
C. C1-inhibitor concentrate
D. Hydroxocobalamin
E. Hyperbaric oxygen

B. Ascorbic acid

Ascorbic acid is the treatment of choice for NADH methaemoglobinaemia reductase Deficiency

The striking feature, in this case, is the significant discrepancy between the PaO2 and SaO2. Given the patient is on 15L O2, the PaO2 is at a level expected indicating that gas exchange at the level of the alveoli is not impaired. However, the SaO2 is markedly low at 52% indicating impaired oxygen binding to haemoglobin. The main two causes of this picture are methaemoglobinaemia and carboxyhemoglobinemia. The family history is indicative of familial NADH methaemoglobinaemia reductase deficiency.

Ascorbic acid is the treatment of choice for NADH methaemoglobinaemia reductase deficiency.

Hyperbaric oxygen is the treatment of choice for severe carbon monoxide poisoning. Although carbon monoxide poisoning presents similarly and remains within the differential diagnosis, the positive family history makes congenital methaemoglobinaemia the more likely diagnosis.

4-Dimethylaminophenol and **hydroxocobalamin** can be used for the treatment of cyanide poisoning. Cyanide binds the ferric (Fe3+) ion of cytochrome oxidase causing 'histotoxic hypoxia' and lactic acidosis. It would not cause a marked reduction of SaO2 as seen in this case unless it was associated with coexistent carbon monoxide poisoning (e.g. due to smoke inhalation). There is nothing in this case to suggest that this is the case.

C1-inhibitor concentrate remains the gold standard treatment for the acute management of hereditary angioedema. The clinical and laboratory features, in this case, are not in keeping with hereditary angioedema which presents with recurrent attacks of self-limiting oedema affecting the skin, gastrointestinal tract, and airways.

Question 71 of 89

A 36-year-old man is brought to the emergency department following a motor vehicle accident. He is drowsy but conscious. He has an open fracture on the right anterior shin which is bleeding profusely. Vital sign monitoring reveals that he has a respiratory rate of 22 bpm, heart rate of 118 bpm, blood pressure of 92/55 mmHg and temperature of 37.1 degrees Celsius. In an attempt to resuscitate the patient, the doctor decides to transfuse 2 pints of group O negative blood. A few minutes after giving the blood, the nurse notices that the patients developed a generalized rash and swelling of the lips, associated with a loud breathing sound. His medical notes reveal that he has atopy and has been treated for giardiasis multiple times previously.

Which one of the following conditions could possibly represent a predisposition to the patient's condition?

A. Von Willebrand disease
B. Hairy cell leukemia
C. Selective IgA deficiency
D. Severe combined immunodeficiency
E. Wiskott-Aldrich syndrome

C. Selective IgA deficiency

IgA deficiency is associated with anaphylactic reaction to blood products

This patient presented after a motor vehicle accident with an open fracture wound. The vital signs pointed towards a hypovolemic shock secondary to blood loss from the fracture wound and also possibly secondary to an internal injury, such as a spleen laceration. The patient was transfused with group O negative blood, which is the universal donor blood group, but developed an anaphylactic reaction. This is quite unusual given that the blood was group O negative. Anaphylactic reactions to products are common among individuals with selective IgA deficiency. Selective IgA deficiency is the most common primary immunodeficiency and individuals with this condition have low IgA levels despite normal levels of the IgG and IgM. Patients often suffer from recurrent airway infections, atopy, autoimmune diseases and are prone to have giardiasis infection. To avoid this, these patients must receive blood products without IgA.

1: Von Willebrand disease is a coagulation disorder which affects intrinsic pathway and is due to deficient von Willebrand factor. This leads to an impairment in the formation of the platelet plug. There is no increased risk of anaphylaxis to blood products in patients with this condition.

2: Hairy cell leukemia is one type of lymphoid neoplasm with the cells having hair-like projections and hence the name of the condition. The disease leads to bone marrow fibrosis. There is no increased risk of anaphylaxis to blood products in patients with this condition.

4: Severe combined immunodeficiency is a type of immunodeficiency which involves both a B and T-cell disorder. There are different types comprising IL-2R gamma chain deficiency and adenosine deaminase deficiency. Patients often present with failure to thrive, thrush and diarrhea. They have an increased susceptibility to infections from several types of microorganisms such as viruses, fungi, protozoan, and bacteria. There is no increased risk of anaphylaxis to blood products in patients with this condition.

5: Wiskott-Aldrich syndrome is a type of immunodeficiency which involves both a B and T-cell disorder. It is due to a mutation on the WASp gene causing platelets and leukocytes to be unable to carry out antigen presentation due to a defective actin cytoskeletal reorganization. Patients often present with the triad of thrombocytopenia, recurrent pyogenic infections, and eczema. There is no increased risk of anaphylaxis to blood products in patients with this condition.

Question 72 of 89

A 35-year-old patient presents to a rural emergency department with abdominal pain and facial swelling. He has a past medical history of C1 esterase inhibitor deficiency. He has recently arrived from Belarus and does not have any of his regular medications. He does not smoke cigarettes or drink alcohol.

His observations are heart rate 88 beats per minute, blood pressure 122/78mmHg, respiratory rate 18/minute, oxygen saturations 96% on room air and temperature 37ºC.

On examination, there is facial swelling without airway compromise. There is mild abdominal tenderness without any evidence of peritonism. There is no rash or wheeze.

Unfortunately, there is no possibility of locating IV C1-inhibitor concentrate in this rural setting. Given the clinical history,

what is the most appropriate medication to abort the attack?

A. Adrenaline
B. Danazol
C. Fexofenadine
D. Fresh frozen plasma
E. Prednisolone

D. Fresh frozen plasma

Hereditary angioedema: the acute management is IV C1-inhibitor concentrate or fresh frozen plasma if this is not available

Fresh frozen plasma is the correct answer. This is an available option for treating acute attacks of hereditary angioedema (HAE) if IV C1-inhibitor concentrate is not available.

Danazol is incorrect. This is a gonadotropin inhibitor used in the prophylaxis of HAE rather than in the management of acute attacks.

Fexofenadine, prednisolone and adrenaline are all incorrect. They are ineffective in managing the swelling associated with this condition. The postulated theory is that histamine-mediated angioedema responds to these interventions but bradykinin mediated angioedema is resistant. Bradykinin is the principal mediator of symptoms in HAE.

Question 73 of 89

A 73-year-old woman is reviewed routinely in haematology clinic. She has a diagnosis of chronic lymphocytic leukaemia. She is not on any treatment but remains under regular review. She has lost 5% of her body weight in the last 9 months. She reports occasional fatigue. She does not smoke cigarettes or drink alcohol. She lives alone and is independent.

On examination, there are two 0.5cm lymph nodes palpable in her neck, which were palpable at her previous appointment.

Blood tests at the time of clinic review:

Hb	122 g/L	Male: (135-180)Female: (115 - 160)
Platelets	75 * 10^9/L	(150 - 400)
WBC	14 * 10^9/L	(4.0 - 11.0)
Neuts	6.0 * 10^9/L	(2.0 - 7.0)
Lymphs	7.8 * 10^9/L	(1.0 - 3.5)
Mono	0.2 * 10^9/L	(0.2 - 0.8)
Eosin	0.0 * 10^9/L	(0.0 - 0.4)

Blood tests two months ago:

Hb	123 g/L	Male: (135-180)Female: (115 - 160)
Platelets	189 * 10^9/L	(150 - 400)
WBC	13.5 * 10^9/L	(4.0 - 11.0)
Neuts	6 * 10^9/L	(2.0 - 7.0)
Lymphs	7.3 * 10^9/L	(1.0 - 3.5)
Mono	0.2 * 10^9/L	(0.2 - 0.8)
Eosin	0.0 * 10^9/L	(0.0 - 0.4)

What patient-specific factor does this patient possess that warrants commencing treatment for her underlying malignancy?

A. Fatigue
B. Weight loss
C. Lymphadenopathy
D. Progressive lymphocytosis
E. Thrombocytopenia

A. Thrombocytopenia

Thrombocytopenia -> indication to start treatment in CLL

Thrombocytopenia is the correct answer. The development of thrombocytopenia is an indication to start treatment in CLL.

Fatigue is incorrect. While extreme fatigue may be an indication to start treatment, this patient reports only occasional fatigue and therefore treatment is not warranted on this basis.

Progressive lymphocytosis is incorrect. This patient has a < 50% increase in lymphocyte count over the course of two months and therefore does not meet criteria to start treatment on this basis.

Lymphadenopathy is incorrect. The lymphadenopathy needs to be either massive (>10cm) or progressive to warrant treatment and it is neither.

Weight loss is incorrect. This patient has lost 5% of her body weight in 9 months. She would need to have lost 10% in approximately six months to warrant treatment.

Question 74 of 89

A 67-year-old male with known chronic lymphocytic leukaemia (CLL) currently under monitoring only presents to his general practitioner. He has developed new fevers and reports his pillow being wet. He has a dry cough and has lost a few pounds in weight but otherwise feels well. He is otherwise well and has hypertension and type 2 diabetes. He was taking amlodipine, metformin and tolbutamide but has stopped these two weeks prior due to his prescription not being renewed. He is a smoker and drinks alcohol 10-14 units a week.

On examination, his temperature is 37.9ºC and his chest is clear. He is clammy but there are no visible rashes.

Hb	124 g/l
Platelets	378 * 10^9/l
WBC	13.2 * 10^9/l
Neuts	11.2* 10^9/l
CRP	9mg/L
LDH	857 U/L (normal range 180-360 U/L)

What is the likely explanation?

A. Drug withdrawal reaction
B. Richter's transformation
C. Lower respiratory tract infection (LRTI)
D. Glandular fever
E. Neutropaenic sepsis

B. Richter's transformation

New B-symptoms in CLL -> Richter's transformation

This gentleman presents with new fevers and night sweats on a background of CLL which is normally controlled. He notably has a raised lactate dehydrogenase (LDH) and these symptoms in the context of haematology are B-symptoms. He is not neutropenic. Glandular fever would not explain the night sweats and fevers and usually presents with fatigue and a sore throat. The medications would not cause isolated fever as a withdrawal reaction. A LRTI is possible but there is only a dry cough and no chest signs. We know that 5% of CLL will undergo Richter's transformation becoming a high grade lymphoma with new B-symptoms and crucially a rise in LDH which makes this the likeliest answer.

Question 75 of 89

A 53-year-old man presents with abdominal pain. He was seen six weeks ago by surgeons who diagnosed gastritis and offered omeprazole. He initially improved but the pain recurred and he has no benefit with omeprazole or gaviscon. He feels full quickly but has no dysphagia or regurgitation. He denies any heartburn. He reports general low energy and is sleeping more hours in the day and can fall asleep at any time.

On examination, a mass is palpable in the epigastrium. It has a palpable indent and you cannot get above it. There are bowel sounds and no other masses are felt. There are some isolated left axillary lymph nodes palpable. His temperature is 37.8ºC and heart rate is 78 beats per minute.

Hb	114 g/l
Platelets	289 * 10 /l9
WBC	51 * 10 /l9

What is the likeliest underlying diagnosis?

A. Gastric carcinoma
B. Visceral leishmaniasis
C. Chronic myelogenous leukaemia (CML)
D. Obstructive sleep apnoea (OSA)
E. Narcolepsy

C. Chronic myelogenous leukaemia (CML

CML may present with massive splenomegaly

This gentleman presents with abdominal pain and a new mass that based on location is likely spleen, stomach or left kidney. OSA and narcolepsy explain the sleepiness but not the abdominal symptoms. Gastric carcinoma with outlet obstruction can give a mass but usually there is vomiting or regurgitation of food contents. The mass is therefore likely to be spleen of which CML and leishmaniasis are the commonest causes. Leishmaniasis is less common making CML the likeliest diagnosis. Furthermore, the WBC is through the roof supporting CML as the cause.

Question 76 of 89

A 12-year-old boy is referred by his paediatrician to a tertiary immunology service. Since infancy, he has suffered from recurrent infections. He has required multiple courses of antibiotics for episodes of sinusitis, otitis media and pneumonia. Heel prick testing was normal perinatally. The referral was ultimately made after he had a prolonged admission to the paediatric intensive care unit with respiratory failure requiring intubation, where he was diagnosed with *Pneumocystis jirovecii* pneumonia. HIV testing was negative. In addition to recurrent infections, he has had chronic diarrhoea, for which no cause has been found. He is on no regular medications. He does not smoke or drink alcohol and is doing well in school despite his frequent illnesses. His parents attend the appointment with him.

Clinical examination revealed a thin adolescent. There was evidence of stomatitis and gingivitis. There was no organomegaly or lymphadenopathy. The cardiovascular and respiratory examination was unremarkable. There were no rashes. Neurological examination is unremarkable.

Blood tests:

Hb	124 g/L	Male: (135-180)Female: (115 - 160)
Platelets	144 * 10 /L	(150 - 400)
WBC	3.2 * 10 /L	(4.0 - 11.0)
Neuts	1.5 * 10 /L	(2.0 - 7.0)
Lymphs	0.8 * 10 /L	(1.0 - 3.5)
Mono	0.6 * 10 /L	(0.2 - 0.8)
Eosin	0.3 * 10 /L	(0.0 - 0.4)
Na	136 mmol/L	(135 - 145)
K	4.4 mmol/L	(3.5 - 5.0)
Urea	5.2 mmol/L	(2.0 - 7.0)
Creatinine	77 µmol/L	(55 - 120)
CRP	4 mg/L	(< 5)
Bilirubin	8 µmol/L	(3 - 17)
ALP	77 u/L	(30 - 100)
ALT	16 u/L	(3 - 40)
Î³GT	44 u/L	(8 - 60)
Albumin	32 g/L	(35 - 50)
Immunoglobulin A	0.4 g/L	(0.6-5.0)
Immunoglobulin M	2.6 g/L	(0.53 - 2.47)
Immunoglobulin G	5.4 g/L	(6.60 - 15.90)

What is the likely diagnosis?

A. Coeliac disease
B. Cystic fibrosis
C. Hyper IgM syndrome
D. Malnutrition
E. X-linked congenital agammaglobulinaemia

C. Hyper IgM syndrome

Hyper IgM syndrome characteristically presents with infections e.g. *Pneumocystis* pneumonia, hepatitis, diarrhoea

Hyper IgM syndrome is correct. This is usually x-linked and therefore affects males. It is characterized by recurrent bacterial infections, chronic diarrhoea and infection with atypical organisms such as Pneumocystis. It may also be associated with autoimmune anaemia, neutropenia and thrombocytopenia. Patients often have stomatitis and gingivitis. IgM may either be normal or high. IgG and IgA will be low as the condition causes an inability to class switch from IgM.

Bruton's (x-linked) congenital agammaglobulinaemia is incorrect. This causes recurrent bacterial infections but all immunoglobulin subclasses will be low including IgM.

Cystic fibrosis is incorrect. While it would cause recurrent chest infections, heel prick testing as a newborn being normal makes this diagnosis unlikely.

Coeliac disease is incorrect. This can cause a thin habitus and diarrhoea but would not explain the other findings.

Malnutrition is incorrect. While this can predispose to infection, there is no reason to believe he is not being cared for properly. We are told he is doing well in school and has supportive parents that attend his hospital appointments.

Question 77 of 89

A 74-year-old man is seen in the haematology clinic. He was diagnosed with chronic lymphocytic leukaemia 6 months ago following an incidental finding of lymphocytosis on some routine blood tests with his General Practitioner. He is currently under surveillance monitoring.

Over the last 6 months he has generally been in good health. He has occasional fatigue particularly towards the end of the day but remains to have a good appetite and reports no change in his weight. Only for the last week has he reported to be feeling more unwell with intermittent fevers and chills.

On examination he is febrile at 38.3ºC. He is mildly tachycardic with a heart rate of 114bpm and a blood pressure of 133/79mmHg. His chest is clear and heart sounds are normal. His abdomen is soft and non-tender. There is palpable 2cm splenomegaly.

Full blood count (6 months ago):

Hb	148 g/L	Male: (135-180)Female: (115 - 160)
WBC	49.3 * 10 /L 9	(4.0 - 11.0)
Lymphocytes	46.3 * 10	(0.8 - 5.0)
Neutrophils	2.7 * 10	(2.5 - 7.5)
Platelets	200 * 10 /L 9	(150 - 400)

Full blood count (today):

Hb	127 g/L	Male: (135-180)Female: (115 - 160)
WBC	75.6 * 10 /L 9	(4.0 - 11.0)
Lymphocytes	69.4 * 10	(0.8 - 5.0)
Neutrophils	3.8 * 10	(2.5 - 7.5)
Platelets	102 * 10 /L 9	(150 - 400)

Blood film: smudge cells seen.

Which of the following is an indication to start treatment in this patient?

A. Splenomegaly
B. Presence of fever for 1 week
C. White cell count > 75
D. Presence of smudge cells on peripheral blood film
E. Thrombocytopenia

A. Thrombocytopenia

Thrombocytopenia -> indication to start treatment in CLL

This patient has a new thrombocytopenia compared to his blood tests 6 months ago. A new thrombocytopenia or sign of bone marrow failure is an indication to initiate treatment. Treatment options vary depending on the patient's overall health, co-morbidities and prognostic factors.

Massive splenomegaly > 5cm is an indicator to start treatment. However, this patient has a 2cm splenomegaly which does not make it an indicator for starting treatment.

Recurrent infections or fevers persisting beyond 2 weeks are indicators for starting treatment. This patient has had fevers for 1 week. However, if this continued then that would be a second indicator for starting treatment.

A rising lymphocyte count is an indicator for starting treatment. However, a doubling of lymphocytes in 6 months is needed which is not seen here.

Smudge cells are not an indicator for starting treatment.

Other indicators for starting treatment include: massive lymphadenopathy, constitutional symptoms including fever, weight loss or night sweats and the presence of haemolysis

Question 78 of 89

A 45-year-old man is referred to the haematology clinic with a 4-month history of easy bruising and prolonged bleeding. He denies any other symptoms and is otherwise feeling well. His past medical history includes rheumatoid arthritis that he reports is not well-controlled, with frequent flares. There is no family history of bleeding disorders.

Laboratory blood tests:

Hb	126 g/L	(135-180)
Platelets	201 * 10 /L 9	(150 - 400)
WBC	5.5 * 10 /L 9	(4.0 - 11.0)
Prothrombin time (PT)	12 secs	(10-14 secs)
Activated partial thromboplastin time (APTT)	45 secs	(25-35 secs)
Fibrinogen	2.2 g/L	(2 - 4)
D-Dimer	300 ng/mL	(< 400)
Von Willebrand Factor	100 IU/dL	(50-150)
Factor VIII levels	Reduced	

What is the most likely diagnosis?

A. Acquired haemophilia A

B. Beta thalassaemia trait

C. Christmas disease

D. Factor V Leiden

E. Von Willebrand's disease

A. Acquired haemophilia A

Acquired haemophilia A is diagnosed in patients without previous or familial histories of bleeding who have isolated prolongation of the activated partial thromboplastin time (aPTT), reduced FVIII levels as well as evidence of FVIII inhibitor activity

This patient has a diagnosis of acquired haemophilia A, an autoimmune disorder secondary to antibody production against factor VIII. Unlike congenital haemophilia A, acquired haemophilia A develops later in life. Up to 50% of cases of acquired haemophilia A are associated with co-existing autoimmune conditions such as rheumatoid arthritis, systemic lupus erythematosus and inflammatory bowel disease. The investigation findings are similar to congenital haemophilia A and include a raised aPTT and reduced FVIII levels. The presence of antibodies to factor VIII helps to differentiate between congenital and acquired causes.

Beta thalassaemia trait is a disorder of haemoglobin chain production, causing microcytic anaemia. Symptoms include those associated with anaemia such as pallor, shortness of breath and fatigue. Bruising and bleeding are not common features and beta thalassaemia trait does not explain this patient's clotting abnormalities.

Christmas disease (also known as haemophilia B) is an inherited coagulation disorder associated with reduced levels of factor IX. It has no impact on factor VIII levels and cannot explain this patient's laboratory findings.

Factor V Leiden (also known as activated protein C resistance) is the most common cause of thrombophilia. Rather than increasing bleeding, increased coagulation is seen, including an increased risk of deep vein thromboses. Therefore, factor V Leiden is incorrect.

Von Willebrand's disease is the most common inherited bleeding disorder. Symptoms include epistaxis, menorrhagia and gum bleeding. Similarly to this patient, coagulation studies demonstrate a prolonged APTT and reduced factor VIII levels. However, the presence of factor VIII inhibitor activity makes a diagnosis of Von Willebrand's disease less likely and points toward an autoimmune cause of this patient's symptoms.

Question 79 of 89

A 65-year-old female, known to have metastatic breast carcinoma presents to the hospital with a 24-hour history of increasing pain, swelling and erythema of her right calf. She is otherwise relatively well. She was diagnosed with breast carcinoma four years previously and was initially treated with surgery, radiotherapy and chemotherapy. She was found to have disease recurrence with distant metastasis 12 months previously. She is known to have liver and bone metastases.

On examination, she appears pale and has some hair loss following recent chemotherapy. Apart from her swollen, erythematous, tender right calf and a fentanyl patch examination is unremarkable.

Bloods on admission show:

Hb	100 g/l
Platelets	180 * 10 /l9
WBC	7.2* 10 /l9
D-Dimer	1276 µg/L (normal <500µg/L)
Na $^+$	132 mmol/l
K $^+$	4.5 mmol/l
Urea	7.2mmol/l
Creatinine	110 µmol/l

She is diagnosed with a right leg deep vein thrombosis.

Given her current clinical condition, what is the best method of anticoagulation?

A. Low molecular weight heparin (LMWH) and warfarin until warfarin therapeutic followed bywarfarin therapy for 6 months then reassess.
B. Low molecular weight heparin (LMWH) for 3-6 months then reassess
C. Warfarin therapy starting with a slow loading regime for 6 months then reassess
D. Direct oral anticoagulants (DOACs) for 3-6 months then reassess
E. Referral for inferior vena cava filter insertion

D. Direct oral anticoagulants (DOACs) for 3-6 months then reassess

Cancer patients with VTE - 6 months of a DOAC

Question 80 of 89

A 68-year-old woman is reviewed in haematology clinic routinely. She has a past medical history of chronic lymphocytic leukaemia (CLL), characterised by a deletion of the short arm of chromosome 17 (17p).

On examination, there is cervical lymphadenopathy and splenomegaly. Blood tests:

Hb	115 g/L	Male: (135-180)Female: (115 - 160)
Platelets	189 * 10 /L $_9$	(150 - 400)
WBC	35.3 * 10 /L $_9$	(4.0 - 11.0)
Neuts	2.1 * 10 /L $_9$	(2.0 - 7.0)
Lymphs	33.0 * 10 /L $_9$	(1.0 - 3.5)
Mono	0.2 * 10 /L $_9$	(0.2 - 0.8)
Eosin	0.0 * 10 /L $_9$	(0.0 - 0.4)
Na $^+$	138 mmol/L	(135 - 145)
K $^+$	4.2 mmol/L	(3.5 - 5.0)
Urea	5.1 mmol/L	(2.0 - 7.0)
Creatinine	88 µmol/L	(55 - 120)
CRP	4 mg/L	(< 5)
Lactate dehydrogenase (LDH)	180 U/L	(140-280)

Given the clinical history and underlying diagnosis, which clinical feature is associated with the worst prognosis?

A. Female sex
B. Age > 60
C. Lymphocyte count > 30
D. Normal LDH
E. 17p deletion

A. 17p deletion

del 17p is associated with a poor prognosis in CLL

17p deletion is the correct answer. The patient has chronic lymphocytic leukaemia. This a type of cancer in which the bone marrow makes too many lymphocytes, as evidenced in this case. The cancer in this case is characterized by a genetic mutation causing deletion of 17p, which is a part of chromosome 17. This is associated with a poor prognosis in CLL. It is seen in 5-10% of patients. The absence of part of chromosome 17 results in absence of one allele of the TP53 tumour suppressor gene, predisposing to the development of CLL.

Age > 60 is incorrect. Poor prognosis is associated with those > 70 years of age.Lymphocyte count > 30 is incorrect. Poor prognosis is associated with those patients with lymphocyte counts > 50.Normal LDH is incorrect. A raised LDH implies a worse prognosis.Female sex is incorrect. Male sex implies a worse prognosis.

Question 81 of 89

A 24-year-old woman is admitted with a 1-hour history of worsening dyspnoea. On examination, you note stridor and marked angioedema of the lips and tongue. No rash is present. You immediately seek the assistance of the anaesthetic team to help secure the airway.

The patient has a history of hereditary angioedema. You are told that due to manufacturing issues, IV C1-inhibitor concentrate is not available.

What alternative treatment should be offered?

A. Danazol
B. Fresh frozen plasma
C. Intramuscular adrenaline
D. Intravenous methylprednisolone
E. Tranexamic acid

B. Fresh frozen plasma

Hereditary angioedema: the acute management is IV C1-inhibitor concentrate or fresh frozen plasma if this is not available

The correct answer is **fresh frozen plasma**. This is considered a second-line therapy where C1 inhibitor concentrate is not available as the plasma itself contains C1 inhibitor. No randomised control trials exist but a majority of case reports describe efficacy with signs of improvement described between 30 minutes and 12 hours.

Danazol is incorrect. This is a synthetic androgen that is used in prophylaxis of acute episodes in patients with hereditary angioedema. It has not been shown to provide any benefit in acute attacks.

Intramuscular adrenaline is incorrect. Similar to glucocorticoids, adrenaline is not effective in hereditary angioedema. Episodes of angioedema not responding to adrenaline may be one of the first clues to the diagnosis.

Intravenous methylprednisolone is incorrect. While glucocorticoids are used in histamine-driven angioedema, they are not effective in hereditary angioedema as the pathogenesis is driven by bradykinin rather than histamine. **Tranexamic acid** is incorrect. It has been used for prophylaxis but a minimal effect is seen when used for the treatment of acute attacks.

Question 82 of 89

A 35-year-old woman presents to the emergency department with a one day history of a fever. She has a past medical history of metastatic breast cancer and has received her most recent round of chemotherapy 8 days ago. She is not on any other regular medications.

Her observations are heart rate 111 beats per minute, blood pressure 96/58 mmHg, respiratory rate 22/minute, oxygen saturations 97% on room air and temperature 38.7ºC.

On examination, she is warm to touch with capillary refill time < 2 seconds. A bounding pulse is noted. There is no obvious localizing source of infection from the rest of the examination.

Urinalysis is unremarkable.

Plain radiography of the chest is normal. Blood tests:

Hb	121 g/L	Male: (135-180)Female: (115 - 160)
Platelets	389 * 10 /L 9	(150 - 400)
WBC	1.7 * 10 /L 9	(4.0 - 11.0)
Neuts	0.4 * 10 /L 9	(2.0 - 7.0)
Lymphs	0.9 * 10 /L 9	(1.0 - 3.5)
Mono	0.2 * 10 /L 9	(0.2 - 0.8)
Eosin	0.2 * 10 /L 9	(0.0 - 0.4)
Na +	137 mmol/L	(135 - 145)
K +	4.4 mmol/L	(3.5 - 5.0)
Bicarbonate	20 mmol/L	(22 - 29)
Urea	8.9 mmol/L	(2.0 - 7.0)
Creatinine	111 µmol/L	(55 - 120)
CRP	180mg/l	(<4)

What is the appropriate antibiotic choice?

A. Meropenem
B. Piperacillin/tazobactam
C. Piperacillin/tazobactam and gentamicin
D. Piperacillin/tazobactam and gentamicin and vancomycin
E. Piperacillin/tazobactam and vancomycin

B. Piperacillin/tazobactam

Piperacillin with tazobactam (Tazocin) is the empirical antibiotic of choice for neutropenic sepsis

Piperacillin/tazobactam is the correct answer. This patient has neutropenic sepsis secondary to chemotherapy treatment. This choice is supported by NICE guidelines and is first-line for the treatment of neutropenic sepsis.

Meropenem is incorrect. If the patient fails to respond after 48 hours then they may be escalated to meropenem.

Piperacillin/tazobactam and vancomycin is incorrect. Some units advocate giving vancomycin in addition to piperacillin/tazobactam if there is an indwelling central venous access device. This patient does not have such a device in situ and this procedure is not supported by NICE.

Piperacillin/tazobactam and gentamicin is incorrect. NICE specifically advise not adding an aminoglycoside unless local policy dictates.

Piperacillin/tazobactam and gentamicin and vancomycin is incorrect. This is not a treatment strategy supported by NICE.

Question 83 of 89

A 67-year-old woman presents to her GP with a lump in her armpit that she first noticed two months ago, and feels is getting bigger. She has not suffered from pyrexia, night sweats, weight loss or lethargy. The GP runs some blood tests, which can be seen below.

Hb	116 g/L	Male: (135-180)Female: (115 - 160)

Platelets	160 * 10 /L 9	(150 - 400)
WBC	72 * 10 /L 9	(4.0 - 11.0)
Neuts	5 * 10 /L 9	(2.0 - 7.0)
Lymphocytes	66 * 10 /L 9	(1.0 - 3.5)
Blood film	smudge cells	

Which of the following would imply a poor prognosis in this woman?

A. Female sex
B. Philadelphia translocation
C. Low B2-microglobulin
D. Del 17p
E. Age less than 70

D. Del 17p

del 17p is associated with a poor prognosis in CLL

This woman is suffering from chronic lymphocytic leukaemia (CLL), a slowly progressive disease. Most patients are either asymptomatic or present with lymphadenopathy. Only 10% of patients will have B symptoms of lymphoma. Half of the patients present with splenomegaly and about 15% with hepatomegaly. The key to making this diagnosis is the lymphocyte-predominant leukocytosis, with smudge cells on the blood film. Smudge cells, also known as smear cells, are cell remnants that arise during slide preparation and are the result of abnormally fragile lymphocytes in CLL.

From the options given above, the only poor prognostic indicator is del 17p mutation, which indicates resistance to standard chemotherapy regimens. B2-microglobulin levels are used in multiple myeloma where raised levels imply poor prognosis. It has also been included in the new (2016) CLL international prognostic index, where again high levels imply poor prognosis.

Philadelphia translocation, t(9;22), is a poor prognostic factor in ALL.Male sex, not female sex, is a poor prognostic factor.An age of fewer than 70 years old is a good prognostic marker

Question 84 of 89

A 16-year-old boy from Iran presents to the emergency department with dyspnoea, dry cough, and fevers.

On arrival, his oxygen saturations are 91% with a respiratory rate of 27/minute. His blood pressure is 110/70mmHg and heart rate 89/minute. There are widespread crackles on auscultation.

Chest x-ray shows diffuse bilateral interstitial infiltrates. You begin treatment for communityacquired pneumonia and send sputum samples for culture.

Blood tests:

Hb	95g/L	Male: (135-180)Female: (115 - 160)
Platelets	140* 10 /L 9	(150 - 400)
WBC	2.7* 10 /L 9	(4.0 - 11.0)
Neuts	0.7* 10 /L 9	(2.0 - 7.0)
Lymphs	1.7* 10 /L 9	(1.0 - 3.5)
Mono	0.1* 10 /L 9	(0.2 - 0.8)
Eosin	0.2* 10 /L 9	(0.0 - 0.4)
Na +	140mmol/L	(135 - 145)
K +	3.8mmol/L	(3.5 - 5.0)
Urea	9mmol/L	(2.0 - 7.0)
Creatinine	80µmol/L	(55 - 120)
CRP	234mg/L	(< 5)

His sputum sample grows *Pneumocystis jirovecii* and his antimicrobials are changed to include Cotrimoxazole.

You return to take a more detailed history. He denies any recent treatment with glucocorticoids or immunosuppressive agents. He denies intravenous drug use and informs you he is not yet sexually active. His mother is in attendance and tells you he has suffered from recurrent gastroenteritis throughout his childhood and she feels this led to him being smaller than his peers.

What is the most likely underlying unifying diagnosis?

A. Cryptosporidium
B. Hyper IgM syndrome
C. HIV
D. Acute lymphoblastic leukaemia
E. Malnutrition

A. Cryptosporidium

Hyper IgM syndrome characteristically presents with infections e.g. *Pneumocystis* pneumonia, hepatitis, diarrhoea

This patient has presented with pneumocystis pneumonia, an opportunistic infection caused by an underlying immune deficiency (hyper IgM syndrome).

Hyper IgM syndrome is a heterogeneous group of disorders characterised by defective classswitch recombination leading to raised serum IgM with low levels of IgG and IgA. The most common form is an X-linked recessive trait due to mutation of CD40LG gene affecting roughly 2/1,000,000 males. The mechanism involves impaired immune signalling between T and B cells leading to primary immune deficiency.

Hyper IgM typically presents in infancy as acute severe infection such as pneumocystis pneumonia or cryptosporidium. It may also present as chronic infections throughout childhood with failure to thrive. CMV is common and may lead to associated liver disease. It is also associated with increased risk of hepatocellular carcinoma, cholangiocarcinoma and neuroectodermal tumours. A minority of patients may present with autoimmune disease such as inflammatory bowel disease.

Acute lymphoblastic leukaemia is unlikely given the long history of recurrent infection. HIV is less likely as this patient denies any risk factors or exposure.

Cryptosporidium is an opportunistic infection and is commonly associated with hyper IgM syndrome. It is the likely causative organism for his recurrent gastroenteritis but does not explain his current presentation. Given the history of opportunistic infections, he should be investigated for causes of immunodeficiency.

Malnutrition is incorrect. Recurrent gastroenteritis in childhood may lead to malnutrition and secondary immunodeficiency. However, the question asks for an 'underlying unifying diagnosis'. This should include a cause for his history of recurrent gastroenteritis

Question 85 of 89

A 72-year-old woman presents to the emergency department with a painful and swollen leg. This has developed over two days, and she otherwise feels well in herself. She was recently away on holiday to Spain and returned four weeks ago. She is able to mobilise independently and has had no reduced periods of mobility. She has a background breast cancer diagnosed four years ago, which unfortunately relapsed and spread into her liver. She is now on hormonal treatment only.

On examination, her left leg is swollen and red, and the calf diameter is significantly larger on the left side. A doppler ultrasound scan demonstrates a left-sided deep vein thrombus.

What is the most appropriate anticoagulation strategy?

A. 2-4 months of low molecular weight heparin (LMWH)
B. 3-6 months of low molecular weight heparin (LMWH)
C. 3-6 months of a direct oral anticoagulant (DOAC)
D. Three months of warfarin
E. Six months of warfarin

3. 3-6 months of a direct oral anticoagulant (DOAC)

Cancer patients with VTE - 6 months of a DOAC

NICE updated their guidelines in 2020. DOACs are now preferred to LMWH for patients with active cancer.

As active cancer is likely to cause a continued pro-coagulopathic state, it is recommended to continue anticoagulation for longer than the standard 3 months

. Question 86 of 89

A 52-year-old woman presents to her GP complaining of fatigue and bruising. She has no significant medical co-morbidities. She is referred to the haematology clinic after an abnormal full blood count. Blood results are as follows:

Hb	112 g/L	Male: (135 - 180)Female: (115 - 160)
Platelets	150 * 10 /L 9	(150 - 400)
WBC	25.3 * 10 /L 9	(4.0 - 11.0)
Neuts	4.0 * 10 /L 9	(2.0 - 7.0)
Lymphs	16.2 * 10 /L 9	(1.0 - 3.5)
Mono	3.0 * 10 /L 9	(0.2 - 0.8)
Eosin	1.1 * 10 /L 9	(0.0 - 0.4)
LDH	250 U/L	(140 - 280 U/L)

Multicolour flow cytometry assay reports CD20+, CD23+,and CD5+ expression. She also undergoes fluorescence in situ hybridization (FISH) which reveals a deletion in the short arm of chromosome 17.

Which of the following features in this case is associated with a poor prognosis?

A. Age
B. FISH result
C. Flow cytometry assay result
D. Gender
E. LDH level

B. FISH result

del 17p is associated with a poor prognosis in CLL

The high white cell count composed mostly of lymphocytes points towards a diagnosis of chronic lymphocytic leukaemia (CLL). In CLL, deletion (del) of the short arm of chromosome 17 (17p) is found in under 10% of patients. This abnormality is associated with rapid progression of the disease, as well as a poor response to treatment strategies.

Prognosis is less favourable in those who are over the age of 70, rather than younger patients like this one.CD20+, CD23+, and CD5+ are surface antigens that are expressed in most CLL cases and shouldn't affect prognosis.Male sex, rather than female sex, is associated with a poor prognosis in CLL.Raised LDH is a marker for poor prognostic outcome, but it is normal in her case.

Question 87 of 89

An 18-year-old woman attends the emergency department with difficulty breathing.

On examination, she is cyanosed, her oxygen saturations are 84% on a finger probe, and her chest is clear.

She tells you that she has had a blue tinge around her mouth for her whole life and that she did have some investigations abroad before moving to the UK aged 4.

You take a blood gas, the sample is a dark brown colour. The results are as follows:

pH	7.35	7.35-7.45
PaCO2	4.5kPa	4.7 - 6.0
PaO2	12kPa	11 - 13

Given the likely diagnosis, what is the treatment of choice for the underlying condition?

A. 15L oxygen via a non-rebreathe mask
B. Ascorbic acid
C. Hyperbaric oxygen
D. IV methylthioninium chloride
E. Nitric oxide

B. Ascorbic acid

Ascorbic acid is the treatment of choice for NADH methaemoglobinaemia reductase deficiency

The chocolate brown blood sample, normal PaO2 but low oxygen saturations, and persistent cyanosis since childhood all point towards methaemoglobinaemia. Congenital NADHmethaemoglobinaemia reductase deficiency is usually diagnosed in childhood presenting withpersistent cyanosis with or without shortness of breath.Ascorbic acid is the correct answer, it is the treatment of choice for NADH methaemoglobinaemiareductase deficiency. **15L oxygen via a non-rebreathe mask** is incorrect. This patient may very well have 15L oxygen via a non-rebreathe mask applied in the emergency setting due to the low oxygen saturations, but this would not be the treatment for the underlying condition.

Hyperbaric oxygen is incorrect, this would be a treatment option for carbon monoxide poisoning.

IV methylthioninium chloride is incorrect, it would be the treatment of choice for acquired methaemoglobinaemia rather than congenital.

Nitric oxide is incorrect, this is actually a cause of acquired methaemoglobinaemia.

Question 88 of 89

An 83-year-old woman is seen in clinic by her oncologist. She has recently been diagnosed with chronic lymphocytic leukaemia (CLL). Her blood tests and clinical picture are stable and therefore she is being monitored and treatment has not been initiated. She is to undergo genetic testing to assess her prognosis.

Which of the following is associated with a poor prognosis in her condition?

A. IGHV mutation
B. t(8;21)
C. Trisomy 8
D. t(9;22)
E. del 17p

A. del 17p

del 17p is associated with a poor prognosis in CLL

In chronic lymphocytic leukaemia, del 17p is a poor prognostic factor. IGHV is associated with a good prognosis in CLL rather than a poor one. t(8;21) is associated with a good prognosis in acute myeloid leukaemia. Trisomy 8 is a poor prognostic factor in acute lymphoblastic leukaemia. It is very rarely found in CLL and the prognostic relevance is unknown. t(9:22) is the Philadelphia chromosome and is part of the diagnostic criteria for chronic myeloid leukaemia and carries a poor prognosis in acute lymphocytic leukaemia

Question 89 of 89

A 41-year-old patient receiving treatment for breast cancer presents to the Day Unit with a swollen right leg. She had a left mastectomy and axillary clearance 3 months ago and is currently receiving her second cycle of FEC (epirubicin, cyclophosphamide and fluorouracil) chemotherapy. Ultrasound doppler confirms a deep vein thrombosis of the right superficial femoral vein. The patients renal function is normal.

What is the most appropriate treatment for this patient?

A. Rivaroxaban for 4-6 weeks and reassess VTE risk
B. Warfarin for six months and reassess VTE risk
C. Rivaroxaban for 3-6 months and reassess venous thromboembolism (VTE) risk
D. Warfarin for 3 months and reassess VTE risk
E. Low molecular weight heparin for for 4-6 weeks and reassess VTE risk

C. Rivaroxaban for 3-6 months and reassess venous thromboembolism (VTE) risk

Cancer patients with VTE - 6 months of a DOAC

This patient is receiving treatment for active cancer. NICE guidance recommends that patients with active cancer and venous thromboembolism (VTE) should receive 3-6 months treatment with a direct oral anticoagulant such as rivaroxaban. Their risk of further VTE should then be reassessed and a decision made by the clinical team regarding ongoing VTE treatment or prophylaxis. Previously it was recommended that low-molecular weight heparin was used in this scenario.

Warfarin is avoided in patients with active cancer due to interaction with medications; the need for treatment pauses if patients become pancytopenic and increased bleeding risk.

ONCOLOGY

passmedicine PART 2

2024

Question 1 of 43

A 20-year-old asymptomatic woman is referred to the genetics clinic. Her grandmother died of breast cancer aged 54 and her mother and older sister have both recently been diagnosed with the disease.

Breast examination does not reveal any lumps and her recent ultrasound of the breasts did not reveal any suspicious lesions.

What should she be told about her breast cancer risk?

A. Her chance of having a BRCA1 mutation is 55-65%

B. Her chance of having a BRCA2 mutation is 55-65%

C. If BRCA1 positive, she has a 55-65% chance of developing breast cancer

D. If BRCA2 positive, she has a 55-65% chance of developing breast cancer

E. Given her family history, she has a 55-65% chance of developing breast cancer

C. If BRCA1 positive, she has a 55-65% chance of developing breast cancer

The BRCA1 and BRCA2 genes are tumour suppressor genes involved in DNA repair. Familial mutations in these genes are associated with an increased risk of breast and ovarian cancer. Carriers tend to develop these cancers at a younger age as they have an inherited germline mutation of one copy of the gene so only require a 'second hit' of damage to the second copy to inactivate it. BRCA mutations account for 20-25% of hereditary breast cancers and 5-10% of all breast cancer cases, They also account for 15% of all ovarian cancer.

If a woman with at least a 10% risk of breast or ovarian cancer (approximated from family history) will be offered genetic screening. This requires genetic counselling and living relative whose DNA can also be tested for the mutation, otherwise, the chance of identifying the mutation is too low. This has important ethical considerations with regard to consent.

The percentage risk of developing breast cancer by age 70 is 55-65% for BRCA1 and 45% for BRCA2 mutations.

The percentage risk of developing ovarian cancer by age 70 is 40% for BRCA1 and 15% for BRCA2 mutations.

Because of the high risks involved, women with a positive test result may choose to have enhanced screening for these cancers, prophylactic surgery (bilateral mastectomy and/or bilateral salpingo-oophorectomy) or chemoprevention with hormonal drugs.

Question 2 of 43

You are working as a registrar on the palliative care ward. A 58-year-old lady is admitted for symptom control. She has a past medical history of metastatic pancreatic cancer and diabetic nephropathy. She has been suffering from severe abdominal pain which has been unresponsive to titration of morphine, gabapentin and duloxetine.

Blood results are as follows:

Hb	75 g/l	Na +	138 mmol/l
Platelets	224 * 10 /l 9	K +	3.8 mmol/l
WBC	14.9 * 10 /l 9	Urea	18.6 mmol/l
Neuts	12.9 * 10 /l 9	Creatinine	288 µmol/l
eGFR	22 mL/min		

You decide to start an opioid which acts as a neuropathic agent by NMDA antagonism. What analgesic is most appropriate?

A. Methadone

B. Oxycodone

C. Buprenorphine

D. Paracetamol

E. Venlafaxine

A. Methadone

Methadone can be used as a third line opioid for patients with complex pain that is poorly responsive to other opioids and adjuvants

Methadone can be used as a third line opioid for patients with complex pain that is poorly responsive to other opioids and adjuvants. The patients pain has not responded to titration of morphine, combined with two neuropathic adjuvants. Methadone should therefore be considered.

The patients eGFR is les than 30 which also strengthens the decision to start methadone. Opioids which are safe in CKD 4 and 5 include fentanyl, buprenorphine and methadone.

Paracetamol and venlafaxine are not opioids.

Question 3 of 43

You are asked to provide advice on a patient who has metastatic renal cell carcinoma with pulmonary and bone metastases. They are being managed in their own home with community palliative care input, but their swallow has started to deteriorate. It is now felt that they are approaching the terminal phase of their illness and a decision has been made to start a syringe driver. They report good pain control with their current analgesia regime.

Their current analgesia regime is 60mg oxycodone modified release capsules twice a day with oxycodone instant release, taken as required. The oxycodone modified release is converted into the equivalent subcutaneous dose for an oxycodone syringe driver.

What would be the most appropriate dose of breakthrough analgesia to prescribe based on the current syringe driver dose?

A. Oxycodone I/R oral solution 20mg

B. Oxycodone I/R oral solution 10mg

C. Oxycodone subcutaneous injection 10mg

D. Morphine sulphate for injection 10mg

E. Morphine sulphate injection 20mg

C. Oxycodone I/R oral solution 10mg

Breakthrough dose = 1/6th of daily morphine dose

The breakthrough dose for analgesia should be one sixth of the total dose administered within 24 hours. If there are no changes to the total dose of oxycodone and it is converted to subcutaneous then the total dose is halved (120mg to 60mg). This is because the subcutaneous strength is twice that of oral oxycodone. The drug choice for breakthrough should match what is in the syringe driver as much as possible (stick with oxycodone) and the breakthrough should be a sixth of the total dose. Given that they need a syringe driver which is made up of oxycodone, the most appropriate drug choice would be subcutaneous oxycodone. Therefore, the breakthrough dose would be a sixth of 60mg= 10mg.

Question 4 of 43

You are looking after a 65-year-old man with a background of high-grade lymphoma who she is currently receiving R-CHOP chemotherapy for. She developed neutropaenic sepsis yesterday for which she was started on piperacillin/tazobactam, and gentamicin. She has been persistently pyrexial for the past 24 hours.

She has a background of complex pain for which she uses a 25micrograms/hour fentanyl patch. You are asked to review her due to confusion and visual hallucinations. On examination, she has pinpoint pupils, GCS 14 (M6, V4, E4), and you note myoclonic jerks.

Na +	136 mmol/L	(135 - 145)
K +	3.8 mmol/L	(3.5 - 5.0)
Bicarbonate	19 mmol/L	(22 - 29)
Urea	7.2 mmol/L	(2.0 - 7.0)
Creatinine	130 µmol/L	(55 - 120)
Bilirubin	14 µmol/L	(3 - 17)
ALP	95 u/L	(30 - 100)
ALT	82 u/L	(3 - 40)
Î³GT	86 u/L	(8 - 60)
Albumin	35 g/L	(35 - 50)

You suspect opioid toxicity. What is the most likely explanation for this?

A. Acidosis

B. Fentanyl and piperacillin/tazobactam drug interaction

C. Increased fentanyl absorption due to pyrexia

D. Liver dysfunction

E. Renal dysfunction

C. Increased fentanyl absorption due to pyrexia

Transdermal fentanyl absorption is increased by heat (e.g. hot water bottle) or pyrexia, potentially leading to opioid toxicity

Transdermal fentanyl absorption is increased by heat or pyrexia (which results in vasodilation and increased blood flow), potentially leading to opioid toxicity. This is therefore the correct option.

Acidosis is incorrect. Acidosis is not known to exacerbate opioid toxicity.

Renal dysfunction is incorrect. Fentanyl is hepatically metabolised by CYP450 3A4 with no active metabolites. Therefore it is the opioid of choice in renal failure.

Liver dysfunction is incorrect. Although the patient has a mild transaminitis, their synthetic liver function is normal, making it an unlikely contributing factor.

Piperacillin/tazobactam is incorrect. Fentanyl has no known interactions with piperacillin/tazobactam.

Question 5 of 43

A 63-year-old woman presents with unrelenting thoracic back pain. She finds that the pain is debilitating, especially as she helps look after her grandchildren. She has a past medical history of breast cancer diagnosed two years ago, for which she underwent a mastectomy with reconstruction. She also had postoperative radiotherapy and endocrine therapy. As she was found to be BRCA1 positive, she underwent a hysterectomy with bilateral oophorectomy as well. On examination, she is tender over the spine at T4, but neurological examination is unremarkable.

What is the most appropriate imaging investigation for her back pain?

A. MRI of the whole spine within 7 days

B. MRI of the thoracic spine within 7 days

C. MRI of the whole spine within 24 hours

D. MRI of the thoracic spine within 24 hours

E. Bone scan of the whole body within 7 days

A. MRI of the whole spine within 7 days

The correct answer is MRI whole spine within 7 days. This is a patient with known cancer presenting with highly suspicious back pain but no evidence of spinal cord compression. The most appropriate modality is MRI which can not only detect metastasis but also demonstrate patency or compression of the spinal cord. The whole spine should be scanned as to exclude further metastasis as 30-50% of patients have multi-level involvement by the time of presentation. The timing of the scan depends on suspicion of cord compression. For this patient, there is no evidence of neurological compromise and therefore the scan does not have to occur within 24 hours.

Question 6 of 43

You are looking after a 68-year-old man with aggressive multiple myeloma. He has intractable vomiting which has not improved with regular subcutaneous metoclopramide.

Blood results are as follows:

Calcium	4.2 mmol/L	(2.1-2.6)

You decide to start him on a syringe driver. What would be your anti-emetic of choice?

A. Aprepitant

B. Cyclizine

C. Levomepromazine

D. Nabilone

E. Ondansetron

C. Levomepromazine

Metabolic causes of nausea and vomiting, for example uraemia and hypercalcaemia, cause nausea and vomiting by stimulating dopamine type 2 (D2) receptors on the chemoreceptor trigger zone. This pathway is most effectively blocked by using a central dopamine antagonist such as haloperidol

Metabolic causes of nausea and vomiting (e.g. hypercalcaemia) are particularly sensitive to central dopamine antagonists such as **levomepromazine** making this the favoured choice.

Levomepromazine is a very broad spectrum anti-emetic that acts as an antagonist to multiple receptors including histamine type 1, muscarinic-cholinergic, dopaminergic 2, alpha-1 adrenoceptor, and 5HT-2 receptors. Thus it is a powerful anti-emetic that can be used in many scenarios.

Aprepitant is a substance P/neurokinin 1 receptor antagonist used to prevent chemotherapyinduced and postoperative nausea and vomiting. The patient in this clinical case has nausea secondary to a metabolic abnormality, and thus a dopamine antagonist is the preferred choice.

Cyclizine is a histamine H1-receptor antagonist which may have effects directly on the vestibular system and on the chemoreceptor trigger zone. It also exerts a central anticholinergic action The patient in this clinical case has nausea secondary to a metabolic abnormality, and thus a dopamine antagonist is the preferred choice.

Ondansetron is a specific 5HT3-receptor antagonist which blocks 5HT3 receptors in the gastrointestinal tract and in the CNS. The patient in this clinical case has nausea secondary to a metabolic abnormality, and thus a dopamine antagonist is the preferred choice.

Nabilone is a synthetic cannabinoid which is used to treat nausea and vomiting caused by cytotoxic chemotherapy, which is unresponsive to conventional antiemetics.

Question 7 of 43

A 54-year-old-man with a history of granulomatosis with polyangiitis attends for his annual review in the rheumatology clinic. He was diagnosed at age 35 where he presented with rapidly progressive glomerulonephritis and pulmonary haemorrhage and was treated with cyclophosphamide and steroids. He required a period of haemofiltration on the intensive care unit but has remained well since.

Today, he feels well after recently quitting smoking but reports occasional blood in his urine. On examination, his abdomen is soft and non tender, there is no pallor or oedema and his chest is clear. His investigations are below and his urine appears clear.

Hb	105 g/l
Platelets	300* 10 /l9
WBC	5* 10 /l9
Urinalysis	blood 3+ protein -
Na +	139 mmol/l
K +	4.2 mmol/l
Urea	5.6 mmol/l
Creatinine	98 µmol/l

What is his likely diagnosis?

A. Recurrence of glomerulonephritis

B. Transitional cell carcinoma

C. Renal calculi

D. Faulty urinalysis recording

E. Rhabdomyolysis

B. Transitional cell carcinoma

Use of cyclophosphamide in granulomatosis with polyangiitis is associated with increased risk of bladder cancer

The use of cyclophosphamide is linked to transitional cell carcinoma in patients with vasculitis. Given that his U&Es are normal, a recurrence of glomerulonephritis is unlikely. Renal calculi would cause loin to groin pain. Haematuria can often not be visible to the eye even when present. While rhabdomyolysis could give a positive dipstick, there are no other features to suggest this

Question 8 of 43

A 68-year-old man is admitted to the unit with an exacerbation of severe pain around his abdomen. He describes it as a tight band like pain which is burning in character. He has a past medical history of metastatic prostate cancer. His drug history includes morphine modified release (100mg, twice daily), duloxetine (60mg, twice daily), pregabalin (200mg, three times per day). He states that the pain initially responded well to the analgesics he is on however over the past few weeks it has got progressively worse and culminated in a severe attack of pain today.

You suspect that he has got severe neuropathic pain secondary to spinal metastases. The pain is now poorly controlled with a large dose of opioids and two neuropathic agents. Your consultant decides to start him on ketamine.

What acute complication is it important to monitor for?

A. Bladder dysfunction

B. Liver dysfunction

C. Raised intracranial pressure

D. Cardiomyopathy

E. Pulmonary fibrosis

C. Raised intracranial pressure

Ketamine can cause raised intracranial pressure. It is important to monitor for side effects such as headache, papilloedema, and vomiting

Ketamine can be used for neuropathic pain poorly responsive to titrated opioids and oral adjuvant analgesics (e.g. antidepressant and/or anticonvulsant) particularly when there is abnormal pain sensitivity (e.g. allodynia, hyperalgesia or hyperpathia). Ketamine is an NMDA receptor antagonists. Side effects to be aware of include hypertension, raised intracranial pressure and hallucinations.

Bladder and liver dysfunction can occur with long term use of ketamine. Therefore, if a patient is anticipated to have a reasonably good prognosis, alternative options should be considered to replace the ketamine, for example lidocaine infusions.

Question 9 of 43

A medical consult was sought from the nursing staff concerning a 68-year-old gentleman admitted with a urinary tract infection. He was diagnosed with terminal adenocarcinoma of the pancreas six months ago. He had complained of increasing severe abdominal pain for the last few weeks for which his GP commenced and then titrated morphine sulphate tablets (MST) achieving a temporary analgesic effect with each increment. By the time of admission he had been using 150mg BD and was unable to tolerate any further dose increases owing to drowsiness.

His drug history comprised of ciprofloxacin 250mg BD started four days ago for the management of urinary tract infection, paracetamol 1g QDS, tramadol 100mg QDS, MST 150mg BD, senna 2 tabs ON & Movicol one sachet BD.

In view of the continuing abdominal pain investigations were organised by the inpatient team revealing the following findings:

Hb	135 g/l
Platelets	224 * 10 /l9
WBC	11.2 * 10 /l9
Bilirubin	23 µmol/l
ALP	189 u/l
ALT	326 u/l
γGT	178 u/l
Albumin	34 g/l

Chest x-ray: Normal heart borders and lung fields, no evidence of subdiaphragmatic air. Abdominal x-ray: presence of faeces, no evidence of dilatation.

USS abdomen: multiple hypoechoic lesions within the liver, nil else abnormal noted. What is the next most appropriate management option?

A. Commence syringe driver containing diamorphine

B. Refer for immediate surgical consult

C. Refer for palliative radiotherapy

D. Convert MST to fentanyl

E. Commence trial with dexamethasone

E. Commence trial with dexamethasone

This gentleman has evidence of extensive hepatic metastases most probably arising from known adenocarcinoma of the pancreas. Despite an escalating opiate regimen he is unable to obtain satisfactory pain relief. One of the sources of pain associated with liver metastases is due to stretching and irritation of the liver capsule for which a trial of dexamethasone may provide an analgesic effect. Option 5 is, therefore, the most suitable option.

There is no evidence of obstruction or perforation and thus a surgical consult is not warranted, and palliative radiotherapy is not specifically indicated here (in contrast with bone metastases).

Switching to fentanyl will probably not provide any additional benefit in terms of tolerance compared with MST; it is however indicated in patients with poor renal failure owing to its predominant hepatic route of metabolism. For further information:

Question 10 of 43

A medical consult was sought regarding a 58-year-old gentleman admitted with pneumonia complaining of relentless hiccups. He was diagnosed with hepatocellular carcinoma six months ago and was deemed not for curative treatment owing to the extent of the disease. He was initially comfortable with no discernible symptoms but over the last four weeks developed hiccups. In conjunction with the palliative care nurse his GP had initially trialled domperidone and then haloperidol but with no improvement in his symptoms. He denied the presence of any other symptoms other than fatigue, and upon questioning denied the presence of abdominal pain, heartburn or early satiety.

His past medical history was comprised of alcohol dependence syndrome, chronic liver disease, hypertension and hypercholesterolaemia for which he was prescribed thiamine 100mg TDS, lactulose 10mls BD, spironolactone 100mg OD, propranolol 40mg OD, simvastatin 20mg OD as well as intravenous co-amoxiclav 625mg TDS for the treatment of his pneumonia. Examination revealed the presence of a cachectic male. His blood pressure was 108/58 mmHg, heart rate 78 bpm, respiratory rate 18/min and temperature 36.6ºC. Examination of his gastrointestinal system revealed the presence of a palpable mass in the right upper quadrant inferior to the right sterna edge and ascites with no tenderness. Examination of his neurological system revealed the absence of any focal neurological symptoms; likewise, examination of cardiovascular and respiratory systems was unremarkable.

Recent investigations revealed the following results:

Bilirubin	22 µmol/l
ALP	221 u/l
ALT	278 u/l
Protein	62 g/l
Albumin	18 g/l

Chest x-ray: normal heart borders and lung fields

Surveillance upper GI endoscopy four months ago: normal stomach mucosal surface, no evidence of portal hypertension

Which of the following options is the best option with a view to managing his hiccups?

A. Commence treatment with proton pump inhibitor

B. Commence treatment with levomepromazine

C. Commence treatment with dexamethasone

D. Commence treatment with midazolam infusion

E. Commence treatment with baclofen

C. Commence treatment with dexamethasone

This gentleman has known advanced hepatocellular carcinoma and is complaining of distressing hiccups. He does not have any sign of gastro-oesophageal reflux and therefore a trial of proton pump inhibitor is unlikely to be of benefit. In the presence of hepatic or cerebral cancer a trial of dexamethasone may induce some remission and is therefore suggested as an appropriate next line given he has already had a trial of treatment with a prokinetic and a typical antipsychotic with no remission; see http://www.palliativecareguidelines.scot.nhs.uk/guidelines/symptomcontrol/Hiccups.aspx for further information. There is also limited evidence for treatment with baclofen and levomepromazine; subcutaneous midazolam should be reserved if all else proves futile. Note also the BNF has a very useful section regarding prescribing in palliative care scenarios; in this instance it also suggests the use of antacids or nifedipine in addition to the above options.

Question 11 of 43

A 47-year-old woman presents to the emergency department with sudden onset of shortness of breath and pleuritic chest pain. Observations demonstrate marked hypoxia and she is given oxygen and analgesia and undergoes a chest X-ray followed by a CT pulmonary angiogram which shows a pulmonary embolism. There is also an incidental finding of a nodule suspicious of metastatic disease.

She is started on a treatment dose of low molecular weight heparin and admitted under the medical team for further assessment. During a more detailed history, she explains that she has lost one stone in weight over two months and been feeling progressively more tired as well. She denies any other symptoms. She has a past medical history of asthma and also underwent a caesarean section three years ago. She has two children, of which she breastfed both, and has a long history of use of oral contraceptive use. She is an ex-smoker of 5 pack-years and has minimal alcohol intake. Her grandfather died of prostate cancer, and her sister developed breast cancer at the age of 52.

A complete further examination was completed and no abnormalities were found. Routine blood tests are currently pending, and a flexible bronchoscopy for biopsy of the lung nodule has been arranged.

How should this patient be further investigated?

A. Tumour markers AFP and hCG, and mammography

B. CT abdomen and pelvis, tumour markers AFP and hCG, and a myeloma screen

C. Ultrasound abdomen and pelvis, and tumours marker AFP and hCG

D. CT abdomen and pelvis, and tumour markers AFP and hCG

E. CT abdomen and pelvis, myeloma screen, and colonoscopy

D. CT abdomen and pelvis, and tumour markers AFP and hCG

The correct answer is CT abdomen and pelvis, and tumour markers AFP and hCG. These investigations complete all those recommended by NICE for an unknown primary. This question highlights how to investigate a malignancy of unknown origin. This patient has a likely metastatic disease causing a pulmonary embolism with unclear clinical findings as to the location of the primary malignancy.

NICE recommends the following investigations for all patients:

- FBC, U&E, LFT, calcium, urinalysis, LDH
- Chest X-ray
- CT of chest, abdomen and pelvis
- AFP and hCG

NICE recommends the following investigations for specific patients:

- Myeloma screen (if lytic bone lesions)
- Endoscopy (directed towards symptoms)
- PSA (men)
- CA 125 (women with peritoneal malignancy or ascites)
- Testicular US (in men with germ cell tumours)
- Mammography (in women with clinical or pathological features compatible with breast cancer)

A is incorrect because it does not include CT scanning of the abdomen and pelvis. Mammography is tempting but NICE does not recommend it without any clinical findings which are suggestive of a breast malignancy. B and E are incorrect because a myeloma screen is not indicated without lytic bone lesions. E is also incorrect as colonoscopy should be symptom directed. C is incorrect as US is not an appropriate investigation.

Question 12 of 43

A 62-year-old lady with metastatic breast cancer is admitted with a general decline. She is found to have a pneumonia and treated with IV fluids and antibiotics. She makes limited improvement with these and antibiotics are stopped. The decision is made to focus on control of her symptoms.

She is started on a syringe driver of morphine sulphate to treat breathlessness and pain related to liver metastases. She then develops nausea and hiccups.

Which medication is the best choice to add to the syringe driver?

A. Cyclizine

B. Domperidone

C. Midazolam

D. Metoclopramide

E. Ondansetron

D. Metoclopramide

Cyclizine, domperidone and ondansetron are anti-emetics, though domperidone does not come in a subcutaneous preparation for syringe driver. Midazolam is an anxiolytic.

Metoclopramide is the first choice to treat hiccup as well as nausea. Other options include baclofen, nifedipine and chlorpromazine.

Question 13 of 43

A 60-year-old man has been admitted under urology with difficulty passing urine and back pain. A post-void bladder scan shows 980mls in his bladder. He was catheterised by the admitting doctor and now has haematuria.

He has a background of metastatic prostate cancer, atrial fibrillation, previous NSTEMI, hypertension, hypercholesterolaemia and hypothyroidism. His medications include warfarin and modified-release morphine 30mg every 12 hours.

He has been seen by the urology consultant who has said to put a 3-way catheter in if the haematuria does not settle and refer to the palliative care team for pain management. The palliative care specialist advised increasing the modified release morphine to 45mg every 12 hours.

What dose of immediate-release morphine should the patient be prescribed for breakthrough pain?

A. 5mg

B. 7.5mg

C. 10mg

D. 15mg

E. 20mg

D. 15mg

Breakthrough dose = 1/6th of daily morphine dose

To answer this question, you need to calculate the total daily dose of morphine. The amount of immediate-release morphine for breakthrough pain should be one-sixth (1/6) of the total daily dose.

The patient was previously on 60mg of morphine per day. One-sixth of this would be 10mg of immediate-release morphine for breakthrough pain.

The modified release morphine has now been up titrated to 90mg per day. Whenever background morphine is increased, it is important to calculate the new breakthrough pain dosage for patients to receive the maximal benefit. In this case, one-sixth of 90 is 15mg. Therefore the correct answer is 15mg of immediate-release morphine.

Question 14 of 43

You are looking after an 82-year-old man with a background of metastatic colorectal carcinoma. He is in significant pain and is on a syringe driver containing 6mg of alfentanil. He has end-stage renal failure.

What breakthrough analgesic will you prescribe?

A. 1mg subcutaneous alfentanil

B. 25mg subcutaneous morphine

C. 25mg subcutaneous oxycodone

D. 2mg subcutaneous alfentanil

E. 50mg subcutaneous oxycodone

A. 1mg subcutaneous alfentanil

Breakthrough dose = 1/6th of daily morphine dose

Palliative care guidelines recommend using 1/6 of the daily dose as a breakthrough. Alfentanil is a potent drug and is particularly useful in renal failure as it is metabolised by the liver, has no active metabolites, and has a very short half-life.

Question 15 of 43

An 82-year-old man is reviewed by the palliative care team on the ward. He has a past medical history of metastatic lung cancer. He is not for any further chemotherapy, surgery or radiotherapy. He has deteriorated rapidly in the prior few days and is considered to be in the last days of life. He is suffering from intractable nausea, the cause of which is not clear.

What is the most appropriate medication to manage his symptoms?

A. Buscopan

B. Levomepromazine

C. Metoclopramide

D. Morphine

E. Ondansetron

B. Levomepromazine

Levomepromazine is a 'dirty drug' which blocks lots of different receptors (e.g. ACh, DA, 5HT). It is therefore a broad spectrum anti-emetic. It is first line in the last days of life.

Levomepromazine is correct. This is a broad-spectrum anti-emetic - a 'dirty drug' targeting many receptors. It is therefore the most suitable choice where the cause of nausea is unknown and in the last days of life.

Buscopan is incorrect. This is indicated in palliative care for excess secretions rather than nausea.

Metoclopramide is incorrect. This would be a rational choice for nausea due to gastric stasis. However, where the cause is unknown and in the last days of life, a more broad spectrum antiemetic is preferred. Therefore, levomepromazine is preferable.

Morphine is incorrect. This is indicated in palliative care for breathlessness and pain rather than nausea.

Ondansetron is incorrect. This is a rational choice for chemotherapy or post-operative related nausea. However, it is a specific serotonin 5-HT3 receptor antagonist, with a low affinity for dopamine receptors and therefore has a more narrow spectrum of action than levomepromazine and is, therefore, less preferable in this circumstance.

Question 16 of 43

A 65-year-old woman presents to the emergency department with reduced consciousness. Her husband reports that she has been unwell for the past 3 days with coryzal symptoms. She has a past medical history of hypertension and metastatic pancreatic cancer for which she is under the palliative care team. Her regular medications include amlodipine, bisoprolol, paracetamol and a fentanyl patch for pain.

On examination, her temperature is 38.1ºC with a heart rate of 101 beats/min and a blood pressure of 118/66mmHg. Auscultation of the chest is clear with a respiratory rate of 8 breaths/min and oxygen saturations of 92% on air. Her abdomen is soft and non-tender and she is visibly jaundiced. She has a GCS of 11/15 (E2V4M5).

What is the most likely cause of this patient's reduced consciousness?

A. Delirium
B. Hepatic encephalopathy
C. Hypoglycaemia
D. Meningitis
E. Opioid toxicity

E. Opioid toxicity

Transdermal fentanyl absorption is increased by heat (e.g. hot water bottle) or pyrexia, potentially leading to opioid toxicity

Opioid toxicity is correct. This patient has signs of opioid toxicity including drowsiness and a slow respiratory rate. There are multiple differential diagnoses for an acutely unresponsive patient and, therefore, it is key to take a thorough history and examination. From this patient's collateral history it is learned that she uses a fentanyl patch for pain control. It is important to note that transdermal fentanyl absorption is increased by heat whether that be secondary to the application of a hot water bottle or pyrexia. This patient has a fever of 38.1ºC which is a likely contributing factor to her toxicity.

Delirium is incorrect. This is a very reasonable differential diagnosis given the history of fever in a patient with a frail comorbid status. However, delirium is less likely to cause this patient's reduced respiratory rate compared to opioid toxicity. Generally, fever is associated with tachypnoea and the opposite response should raise suspicion.

Hepatic encephalopathy is incorrect. Although this patient is jaundiced, it is likely to be cholestatic jaundice secondary to the underlying malignancy rather than decompensated liver disease. Furthermore, the acute onset of symptoms and lack of signs of hepatic encephalopathy makes the diagnosis less likely.

Hypoglycaemia is incorrect. This is an important differential diagnosis given the history of advanced malignancy and the likelihood of reduced oral intake. However, the presence of bradypnoea and opioid use makes opioid toxicity more likely.

Meningitis is incorrect. Fever and reduced consciousness are features of meningitis. However, the presence of bradypnoea and the use of strong opioid analgesics make opioid toxicity more likely.

Question 17 of 43

A 56-year-old man with metastatic small cell lung carcinoma is admitted to hospital with vomiting, ankle swelling and pruritus. He recently completed a course of palliative chemotherapy. He has a background of chronic obstructive pulmonary disease and hypertension and takes morphine sulphate (MST) for pain relief.

On examination, he is cachectic with peripheral oedema and skin excoriations. Heart rate is 96 beats per minute and blood pressure is 140/85 mmHg.

Tests show:

Hb	134 g/l
Platelets	185 * 10 /l9
WBC	5.5 * 10 /l9
Na +	146 mmol/l
K +	5.4 mmol/l
Urea	23 mmol/l
Creatinine	420 µmol/l
Urine dip:	Blood +
Urine osmolality	350 mOsm/L
Urinary sodium	45 mEqL
Microscopy:	red cells; casts.

What is the most appropriate pain relief for this patient?

A. Fentanyl patch
B. Diamorphine
C. Oxycodone
D. Pregabalin
E. Tramadol

A. Fentanyl patch

This patient has an acute kidney injury secondary to cisplatin chemotherapy. As a result, he needs adjustments to his pain relief. He is on regular, long-acting MST, so the most appropriate substitution is a fentanyl patch.

Oxycodone can be used in moderate renal failure, but only as breakthrough pain relief. Modified release preparations should be avoided. Diamorphine and tramadol should be avoided in renal failure. Pregabalin can be administered at reduced dose, but would not be an appropriate first-line substitution for MST.

Alfentanil and methadone are safe even in severe renal impairment and are administered subcutaneously. Alfentanil would be appropriate for this patient if he had unstable pain and required dose titration.

Question 18 of 43

A 69-year-old male patient presents with worsening mouth soreness and painful swallowing, progressively getting worse over the last week. He started radical radiotherapy for a base of mouth squamous cell cancer three weeks ago. He also has a PEG in situ. He was recently seen by the dietician and is now having fluid and Ensure nutritional supplements via his PEG. He had been given soluble paracetamol by the specialist nurse, but this has failed to control his pain.

On examination, his oral cavity appears inflamed with multiple small ulcers posterior to his lower lip. There are no patches or plaques inside. You change the paracetamol to liquid co-codamol and give him a topical lidocaine gel at 2% strength.

What is the most appropriate next step in management to control his symptoms?

A. Benzydamine mouthwash
B. Menthol mouthwash
C. Chlorhexidine mouthwash
D. Fluconazole
E. Metronidazole

A. Benzydamine mouthwash

The correct answer is benzydamine mouthwash. This gentleman is suffering from radiotherapyinduced mucositis. Given the location of his tumour and the high dose of radiotherapy necessary for curative therapy, mucositis is not an uncommon side effect. Many of these patients will have a PEG inserted prophylactically in anticipation of the difficulties in maintaining an oral diet with mucositis and oesophageal involvement. For this patient, already on co-codamol, benzydamine mouthwash can be very helpful in treating his symptoms. Menthol and chlorhexidine mouthwashes, often bought over the counter, would increase oral irritation. There is no evidence of fungal or bacterial infection currently.

Question 19 of 43

A 75-year-old man is admitted to the palliative care unit for end of life care. He has a past medical history of metastatic prostate cancer. His drug history includes a syringe driver with morphine, levomepromazine and midazolam. During personal care the nurses have stated that he suffers excruciating pain when being turned onto his side. His pain throughout the day is otherwise well controlled.

What is the most appropriate way to manage his pain?

A. Titrate the syringe driver morphine

B. Morphine sulphate modified release tablets

C. Oxycodone modified release tablets

D. Morphine sulphate immediate release liquid

E. Paracetamol

D. Morphine sulphate immediate release liquid

Incident pain is defined as pain which comes on as a result of an action or activity, for example during personal care. Sublingual fentanyl has rapid onset and is a short-acting opioid, and is therefore a good choice for treating this type of pain

Incident pain is defined as pain which comes on as a result of an action or activity, for example during personal care. Sublingual fentanyl has rapid onset and is a short-acting opioid, and is therefore a good choice for treating this type of pain. Similarly, morphine sulphate immediate release liquid is also suitable.

The patients pain during the day is otherwise well controlled therefore titrating up the syringe driver with more morphine is not appropriate. Not only will this not be particularly effective at treating the incident pain, it will also give rise to unnecessary side effects such as sedation and constipation.

Similarly, morphine sulphate modified release tablets have an action time of approximately 12 hours so they are therefore inappropriate for breakthrough pain as they will cause accumulation of morphine in the system predisposing to toxicity.

Question 20 of 43

A 64-year-old cachectic lady presents to the emergency department with difficulty in breathing. She reports smoking since she was 14 years old. Chest X-Ray reveals a large right sided pleural effusion. A sample is obtained under ultrasound-guidance for evaluation. CT does not identify a cause. The results of the pleural aspiration demonstrate an exudative effusion and histology is suggestive of an adenocarcinoma with positive TTF-1 stain.

What is the most likely cause for the effusion?

A. Non-small cell lung cancer
B. Small cell lung cancer
C. Ovarian carcinoma
D. Breast carcinoma
E. Mesothelioma

A. Non-small cell lung cancer

TTF-1 (thyroid transcription factor 1) is a protein which regulates the transcription of genes specific for the thyroid and lung. Lung adenocarcinomas and small cell carcinomas are usually TTF-1 positive. The histology in the above example is suggestive of an adenocarcinoma and therefore this is mostly likely a lung primary given the TTF-1 stain and history of smoking.

Non-small cell lung cancers include adenocarcinoma, large cell and squamous cell carcinoma.

Question 21 of 43

A 76-year-old woman with metastatic transitional cell carcinoma of the bladder is admitted to hospice with a 3-day history of hallucinations, confusion and pruritus. Her only other symptoms are intermittent vomiting and pelvic discomfort. She has a past medical history of hypertension and has recently completed a course of radiotherapy for bone metastases. She takes paracetamol, morphine sulphate, cyclizine, dexamethasone and a herbal flower remedy for anxiety.

On examination, she is cachectic and frail. The lungs are clear and heart sounds are normal. Pulse rate is 85 beats per minute and blood pressure 130/80mmHg. She is afebrile.

Neurological examination reveals myoclonus. She is mildly hypotonic and all reflexes are diminished. There is miosis on cranial nerve examination.

Blood tests show:

Hb	12.1 g/dl
Platelets	250 * 10 /l9
WBC	12 * 10 /l9
Na +	135 mmol/l
K +	4.3 mmol/l
Urea	5 mmol/l
Creatinine	80 µmol/l
Calcium (unadjusted)	2.1 mmol/l
Bilirubin	15 µmol/l
ALP	45 u/l
ALT	15 u/l
AST	20 u/l
Albumin	28 g/l

Urine dip: no abnormality What is the most likely diagnosis?

A. Hypercalcaemia
B. Opioid toxicity
C. Sepsis
D. Corticosteroid psychosis
E. Brain metastases

B. Opioid toxicity

This lady is presenting with opioid toxicity. She has vomiting, confusion, hallucinations and pruritus. Her symptoms have been precipitated by a reduction in her opioid requirement. This is a common occurrence in patients who have radiotherapy for bone metastases if their medication dose is not adjusted.

Hypercalcaemia could cause a similar presentation, but the corrected calcium is 2.4 mmol/L. This lady is hypoalbuminaemic due to her advanced cancer. The patient no symptoms of infection. She is apyrexial and her slightly raised white cell count is due to her advanced disease and dexamethasone treatment. Corticosteroid psychosis is possible, but would not cause the neurological signs or pruritus. Transitional cell carcinomas of the urothelial tract only rarely metastasise to the brain; the neurological signs suggest opioid toxicity rather than a focal lesion.

Question 22 of 43

A 65-year-old man is referred to the oncology team after presenting to the emergency department. The patient described a three-week history of progressive thoracic back pain. The pain was localised to a point between the patient's shoulder blades, and was exacerbated by movement and coughing.

In addition, over the previous 6 hours, the patient reported that he had been struggling to stand up unassisted, and was having difficultly walking. The sudden change in his mobility had been the event that had prompted his attendance at hospital. When questioned directly, the patient described intermittent tingling sensations in his lower legs, but denied any loss of sensation. There had been no dysfunction of his bladder or bowels.

The patient had been diagnosed with prostate cancer five years previously. This had been treated with a radical prostatectomy and hormone therapy. The patient had moved area two years previously, and had subsequently been lost to urological follow-up. Despite his previous cancer treatment, the patient stated that he was very fit and active, playing golf three times a week and taking an active role in the care of his grandchildren.

Peripheral nerve examination of lower limbs: tone generally increased bilaterally; power generally slightly reduced (4+/5) bilaterally; normal coordination; no impairment of light touch or sharp touch sensation; knee and ankle jerks brisk bilaterally, plantar responses up going bilaterally.

Digital rectal examination: preserved peri-anal sensation and normal tone. Bed-side bladder scan: 50 ml

MRI whole spine: metastatic invasion and collapse of T10 vertebral with spinal cord compression.

The patient was treated with analgesia and dexamethasone while definitive treatment was planned.

What is most appropriate first-line treatment of patient's spinal cord compression?

A. Surgical tumour resection and spinal cord stabilisation with adjuvant radiotherapy
B. Radiotherapy: single dose 8 Gy
C. Chemotherapy: docetaxel
D. Best supportive care
E. Chemotherapy: cabazitaxel

A. Surgical tumour resection and spinal cord stabilisation with adjuvant radiotherapy

The best predictor of neurological outcome with treatment in metastatic spinal cord compression is neurological status at the initiation of treatment. The patient in the question has presented very promptly and has only limited neurological compromise. Therefore, it is imperative that definitive treatment is provided promptly (ideally, within 24 hours) to optimise long-term outcome.

Definite treatment of metastatic spinal cord compression can include any combination of radiotherapy, surgery and chemotherapy. Recent evidence indicates that surgery is superior to radiotherapy in retaining or regaining neurological function and relieving pain. In the case of this patient, who has an excellent performance status, surgery followed by radiotherapy would likely offer a better outcome than radiotherapy alone.

Radiotherapy may be considered as first-line treatment in some circumstances. Recommended regimen vary, for example 20 Gy in five daily fractions. The radiotherapy regime offered as a answer to the question would be appropriate as palliative treatment for pain-control, making it an incorrect answer for this patient.

Chemotherapy have a role in the management of spinal-cord compression secondary to chemosensitive malignancies (for example, small-cell lung cancer or germ cell tumours), but would not be optimal treatment for this patient.

Question 23 of 43

A 56-year-old patient presents to hospital with confusion. He is a long-term smoker. He has a past-medical history of chronic obstructive pulmonary disease, hypertension, bipolar disease and long-term muscular back pain.

On examination he is confused with a Glasgow Coma Scale score of 14 out of 15. He has a pulse rate of 97 beats per minute with a blood pressure of 145/97 mmHg. Respiratory rate is 14 per minute with oxygen saturations of 95% on room air. He has bilateral vesicular breath sounds on auscultation with normal percussion. His abdomen is soft and non-tender with no masses. He has no focal neurology.

Investigations

Hb	102 g/dL
WCC	8.2 *10^9/l
Platelets	133 *10^9/l
Na+	146 mmol/L
K+	5.4 mmol/L
Cr	132 µmol/L
Ur	10.1 mmol/L
Albumin	26 g/L
Adjusted Calcium	3.78 mmol/L
Alkaline Phosphatase	76 IU/L
PTH	Undetectable
Serum ACE	45U/L (10-50)
Serum Electrophoresis	IgG 18 g/L (5-13 g/L) - Polyclonal
Urinary Electrophoresis	Undetectable
Chest X-ray	Right parahilar opacification 3x4cm
Lumbar Spine X-ray	Normal

What is the likely underlying diagnosis?

A. Squamous Cell Carcinoma
B. Multiple Myeloma
C. Sarcoidosis
D. Drug induced hypercalcaemia
E. Prostate carcinoma

A. Squamous Cell Carcinoma

The patient has severe hypercalcaemia. The commonest causes are hyperparathyroidism and cancer. The patient has a normal alkaline phosphatase without evidence of fracture or bone disease making bone metastases unlikely. The patient has a normal serum ACE with an atypical chest X-ray for sarcoidosis, although unilateral chest findings are possible.

The patient has features of multiple myeloma including anaemia, renal insufficiency and hypercalcaemia. However, there is a polyclonal, rather than a monoclonal increase in paraprotein.

The chest x-ray findings with a background of smoking suggest an underlying respiratory malignancy. This patient has squamous cell carcinoma of the lung with parathyroid related protein secretion causing hypercalcaemia. PrPTH is not deteced on a PTH assay. Hypercalcaemia can induce confusion and renal impairment.

Question 24 of 43

A 65-year-old man with metastatic squamous cell lung cancer is admitted to the Acute Medical Unit for the management of hypercalcaemia. He is currently taking slow-release morphine sulphate (MST) 90mg bd to control his pain along with regular naproxen and paracetamol. During his admission he complains of pain in his right arm which is the site of a known skeletal metastasis.

What is the most appropriate medication to prescribe to treat his acute pain?

A. Oral morphine solution 30mg
B. Oral morphine solution 15mg
C. Oral morphine solution 10mg
D. Add alendronate 70mg weekly
E. Tramadol 50mg

A. Oral morphine solution 30mg

This patient is describing break-through pain. Whilst bisphosphonates have a role in bone metastases they are not suitable for acute pain. A suitable break-through dose is therefore 1/6th of the total daily morphine dose i.e. 1/6th of 180mg = 30mg.

Question 25 of 43

A 77-year-old female is electively admitted for investigation of bilateral lower limb swelling onset over the past 3 years associated with limitation of exercise as a result. Her past medical history includes bilateral pulmonary emboli in 2010 and type 2 diabetes mellitus. On examination, nonpitting bilateral lower limb oedema is noted to the high thighs without abdominal involvement. Her heart sounds are normal with no added sounds, her chest is clear. Her abdomen is soft and non-tender without ascites or superficial swelling. A transthoracic echocardiogram demonstrates normal left ventricular function but was unable to produce good views of the right heart. A CT pulmonary angiogram demonstrates no embolus, a ventilation-perfusion scan demonstrates a low probability of pulmonary emboli. Ultrasound abdomen is unremarkable. The patient undergoes a right heart catheter, demonstrating a mean pulmonary arterial pressure of 14mmHg.

What is the likely cause of her lower limb swelling?

A. Left heart failure
B. Idiopathic pulmonary arterial hypertension
C. Right heart failure
D. Pelvic malignancy
E. Lymphoedema

E. Lymphoedema

Interpretation of the various investigations this patient has undertaken allows lymphoedema as a diagnosis of exclusion. The patient would have been referred to this standard set of investigations for pulmonary hypertension after clinically presenting as progressive exercise limitation and peripheral oedema, with the primary concern being right heart failure secondary to an increased pressure within the pulmonary vasculature.

Most importantly, the pulmonary arterial pressure is normal on the right heart catheter, ruling idiopathic pulmonary hypertension. Left heart dysfunction is excluded by a normal echocardiogram evaluation of the left ventricular function. CTPA and normal abdominal ultrasound, predominantly investigating the portal circulation, rule out causes of pulmonary hypertension. Lastly, the non-pitting nature of the oedema suggests a non-cardiac cause, making lymphoedema the most likely cause of lower limb swelling.

Question 26 of 43

A 65-year-old man is admitted to the hospital for palliative radiotherapy. He has a squamous cell carcinoma of the tongue that has metastasized to his neck and surrounds the carotid artery. There are no surgical options to treat his disease.

On day 2 of the admission, he experiences a catastrophic bleed from his neck.

What is the most appropriate medication to administer in this circumstance?

A. Buscopan
B. Chlorpromazine
C. Haloperidol
D. Midazolam
E. Morphine

D. Midazolam

In a massive terminal haemorrhage, a large dose of midazolam (10mg) can be given as part of 'crisis management' to relieve distress. Red or green towels or blankets should be available to soak up and mask the colour of blood

Midazolam is the correct answer. The patient has experienced a terminal haemorrhage as a consequence of his tumour eroding through the carotid artery. Given the lack of surgical options available to treat his condition, urgent symptom control is warranted to manage this distressing event. Intravenous midazolam is indicated in this horrific situation as a consequence of its anxiolytic and sedative effects, along with the rapidity of its onset.

Haloperidol is incorrect. This can be used to treat agitation in palliative care. However, intravenous midazolam is preferred in this catastrophic situation due to its rapidity of onset.

Morphine is incorrect. This is typically used to treat pain and breathlessness in a palliative care setting, rather than agitation.

Chlorpromazine is incorrect. This can also be used to treat agitation in a palliative care setting. However, midazolam is preferred in terminal haemorrhages.

Buscopan is incorrect. This is used to decrease the burden of secretions in palliative care. It is not used to treat agitation.

Question 27 of 43

A 64-year-old Nepalese woman who emigrated to the United Kingdom six months prior arrives to her GP. She has known multinodular goitre which has been untreated. However, her daughter who translates for her reports that she has noticed a tight sensation like she is being strangled. This is worse in the mornings and her daughter has noticed that her face does appear puffy. She has no weight loss, fevers or night sweats. She is otherwise well and has never smoked.

On examination, there is a large multinodular goitre that occupies a large part of the neck and measures 15-20cm in width. There is no audible stridor but there are several neck veins visible. You ask her to stretch out her hands up high to observe for a tremor and she starts to complain of the tightness. becoming worse. Her chest sounds are quiet and her chest is clear. There is dull percussion under the top third of the sternum.

TSH	0.07 mU/L (normal range 0.05-5.0)
T4	10 pmol/L (normal range 9-50)
T3	4.0 pmol/L (normal range 3.5-7.8)
D-dimer	0.15 (normal range 0-0.25)
Chest x-ray	Widened mediastinum, no focal lung lesion

What is the underlying cause of these new symptoms?

A. Multinodular goitre
B. Lymphoma
C. Thoracic artery aneurysm
D. Lung carcinoma
E. Superior vena cava thrombus

A. Multinodular goitre

Enlarged goitre can be a rare cause of SVC obstruction

This presentation is classical for superior vena cava obstruction and the worsening of her symptoms when she lifts her hands up is Pemberton's test which is positive in this case. The next decision is to seek the cause. The obvious cause here is a huge goitre which is recognised as a rare cause of superior cava obstruction. Lung carcinoma is possible but this lady is a non smoker and there is no cough or weight loss. Thoracic artery aneurysm would show a widened mediastinum but there would often be a history of connective tissue disease or uncontrolled hypertension. Lymphoma causing SVC obstruction would typically have mediastinal lymphadenopathy on chest x-ray. SVC thrombus would be unlikely given that the D-dimer is negative

Question 28 of 43

A 78-year-old gentleman with known metastatic prostate cancer is admitted due to lower back pain that is worse on sitting upright. He denies any sensory changes or weakness but is woken at night and cannot sleep. He takes aspirin, paracetamol and bicalutamide regularly.

On examination, he has normal tone, full power and downgoing plantars in both lower legs. He is exquisitely tender in the midline at L2 and L3.

Bilirubin	34 µmol/l
ALP	450 u/l
ALT	56 u/l
Albumin	32 g/l
Calcium	3.1mmol/l
MRI spine	sclerotic lesions seen in T9, L2 and L3

What medical therapy would offer greatest benefit to him?

A. Intravenous fluids
B. Denosumab
C. Spinal decompressive surgery
D. Omeprazole
E. Transcutaneous electrical nerve stimulation (TENS)

B. Denosumab

Denosumab is an unlicensed medication for bony metastasis pain

This gentleman has bony pain associated with bony metastases. This is a key question in identifying the problem and how to help him. There is no sign of nerve compression so spinal surgery will not benefit him. He is asymptomatic from his hypercalcaemia so

IV fluids would not benefit. Omeprazole will help protect his stomach from ulceration. For bony pain, NSAIDs usually are a good first line. Bisphosphonates and radiotherapy help but are not an answer. Denosumab is a third line when the above fail. TENS can be useful for all pains but is not evidence based for bony pain.

Question 29 of 43

You are fast-bleeped to the oncology ward to review a 72-year-old man with a background of metastatic, fungating buccal cancer. During initial MDT, it was deemed inoperable due to carotid artery involvement. He was initially admitted with hypercalcaemia but has deteriorated during his admission and is on the end-of-life pathway.

On your arrival, you note a large amount of blood around the patient with staff using towels and applying pressure to the neck. Nursing staff report that there was a sudden 'pop' and large volume bleeding began at the site of the fungating cancer. They state that bleeding has occurred before but not to this extent.

What is the most appropriate next step in management?

A. Adrenaline
B. Midazolam
C. Morphine
D. Terlipressin
E. Vascular surgery opinion

B. Midazolam

In a massive terminal haemorrhage, a large dose of midazolam (10mg) can be given as part of 'crisis management' to relieve distress. Red or green towels or blankets should be available to soak up and mask the colour of blood

This is an example of a catastrophic, terminal haemorrhage likely arising from carotid artery involvement. In these circumstances, patients can become very distressed and unfortunately, often there is very little that can be done. To aid in relieving stress it is advised to use red or green towels to help soak up blood as well as obscuring the colour of the blood, as well as giving a large dose of midazolam for relaxation.

Adrenaline can be used in specific circumstances of major haemorrhage but is not used in terminal haemorrhage. Given that the history explains that the patient is on the end of life pathway and that he is not for surgical intervention, adrenaline is not appropriate.

Morphine can be used for pain and breathlessness in palliative care but is not used in terminal haemorrhages.

Terlipressin can be used for major haemorrhage, particularly of gastrointestinal bleeding, however again, this is not appropriate in terminal haemorrhages.

The MDT has already decided that surgical intervention is not appropriate and therefore a vascular surgery opinion at this time would not be appropriate.

Question 30 of 43

A 60-year-old woman presented to the emergency department with recurrent episodes of severe headache and vomiting over past 3-4 days. She was known to have underlying metastatic breast cancer for which she was receiving palliative chemotherapy. She was also known to have underlying type 2 diabetes mellitus and osteoarthritis of bilateral knee joints.

On examination, her pulse rate was 105/min, respiratory rate was 16/min, blood pressure was 120/80 mmHg. Fundoscopy revealed early papilloedema.

A contrast enhanced CT brain was done which showed a solitary mass of 3 cm in the left cerebral hemisphere.

What is the next appropriate management?

A. Start 16 mg of i.v dexamethasone and prophylactic anticonvulsants
B. Start 16 mg of dexamethasone and arrange urgent MRI brain
C. Refer to neurosurgery for biopsy of the brain lesion
D. Refer to radiation oncologist for Whole brain radiotherapy
E. Stereotactic radiosurgery

B. Start 16 mg of dexamethasone and arrange urgent MRI brain

High dose dexamethasone is the mainstay of immediate management of brain metastasis. For lesions identified as solitary on contrast enhanced CT brain, MRI with gadolinium is indicated to assess the extent of intracranial disease. Patients presenting with seizure activity should be given anticonvulsant therapy. Patients with limited number of lesions(<4) can be considered for more aggressive treatment like neurosurgery or stereotactic radiotherapy but not the immediate step. Biopsy of the lesion is indicated if primary is unknown.Whole brain radiotherapy is indicated for multiple brain metastasis (>4).

Question 31 of 43

A 48-year-old woman is currently undergoing various cycles of chemotherapy for breast cancer. She asks the oncology team for advice as, 2-3 days post-chemotherapy, she experiences severe nausea and vomiting that lasts for several days. This is debilitating, despite taking antiemetics during the chemotherapy and afterward.

What is the most appropriate drug to prevent this delayed reaction?

A. Dexamethasone
B. Levopromazine
C. Lorazepam
D. Metoclopramide
E. Ondansetron

A. Dexamethasone

Dexamethasone is the most useful agent in preventing the delayed phase of chemotherapyinduced emesis

Dexamethasone has been shown by numerous trials to be the most effective agent in preventing delayed nausea and vomiting. As opposed to more immediate nausea and vomiting, for which antiemetics are useful, the delayed reaction (between 24 hours and 1-week post-chemotherapy) is not as effectively reduced by antiemetics.

Levopromazine is a 'dirty drug', acting at multiple receptor sites. It is a useful antiemetic, particularly parenterally, but sedates patients quite significantly and is generally reserved for palliative care and intractable symptoms.

Lorazepam, a benzodiazepine, has mild antiemetic properties and is not considered effective at reducing the delayed phase. It is generally used for its anti-anxiolytic properties.

Metoclopramide is a dopamine antagonist and an effective antiemetic. It may be used first-line for immediate nausea and vomiting. However, in the context of the delayed phase, dexamethasone is more useful.

Ondansetron is a serotonin receptor antagonist and another effective antiemetic used for chemotherapy-induced nausea and vomiting. With regards to the delayed phase, however, dexamethasone is the most effective agent.

Question 32 of 43

A 68-year-old man with metastatic prostate cancer is due to go for radiotherapy in an hour. The nurses tell you that he complained of pain during the transfer yesterday. His pain has been otherwise well controlled on a total of 60mg morphine daily.

How is it best to manage his pain control for the transfer today?

A. Nothing as it is only temporary pain
B. An additional dose of 10mg morphine 30 minutes prior to his transfer
C. An additional dose of 15mg morphine 1 hour prior to his transfer
D. An additional dose of 10mg morphine 1 hour prior to his transfer
E. An additional dose of 15mg morphine 30 minutes prior to his transfer

B. An additional dose of 10mg morphine 30 minutes prior to his transfer

A breakthrough dose of morphine should be given 30 minutes prior to his transfer as indicated in the BNF. 1 hour is too long prior as it should last him the transfer back and 30 minutes prior is enough for the morphine to have the desired effect. A breakthrough dose should be 1/6th of his total daily dose which in this case is 10mg. Therefore, 15mg is too high a dose for this patient.

Although it is temporary pain, it should still be treated as the patient's comfort and quality of life are important aspects of his care.

Question 33 of 43

A 72-year-old man presents to the emergency department with shortness of breath. He describes his breathlessness has progressively become worse over the last two weeks and is especially troublesome when leaning forward. His wife has commented that his appearance has changed too, with his face becoming more swollen and different in colour. He has a past medical history of right-sided heart failure, myocardial infarction, type two diabetes, COPD and small cell lung cancer diagnosed three months ago. He is an ex-smoker of 20 pack-years and drinks moderate amounts of alcohol.

On examination, he appears settled at rest. His face is swollen and slightly purple. His neck veins are distended and his JVP is markedly elevated to 4cm. Auscultation reveals bibasal crepitations. There is pitting oedema at the ankles. A chest X-ray demonstrates a widened mediastinum.

What investigation is most appropriate to establish the diagnosis?

A. ECG
B. Trans-thoracic echocardiogram
C. Trans-oesophageal echocardiogram
D. Bronchoscopy
E. CT chest

E. CT chest

The correct answer is CT chest as the patient has a clinical history consistent with obstruction of the superior vena cava (SVC). There are several symptoms and signs consistent with SVC obstruction, including breathlessness worse, leaning forward, distended neck veins and a swollen face. This, in combination with known lung cancer, suggests progression of the primary malignancy or lymph nodes compressing the SVC. Whilst some of the finds could be suggestive of acute pulmonary oedema, the chest X-ray is consistent with SVC obstruction. As a cardiac cause is unlikely, an echocardiogram or ECG is unlikely to be diagnostic. A bronchoscopy is a possible approach for obtaining a biopsy if an SVC obstruction is diagnosed and the source of the obstruction is unclear, but as this patient is known to have lung cancer this is not going to be helpful for this patient.

Question 34 of 43

A 40-year-old man is seen in the Emergency Department with breathlessness.

He was diagnosed with non-Hodgkin lymphoma 6 months ago and has been receiving chemotherapy. He is awaiting an appointment in haematology outpatients to assess his response to chemotherapy.

He describes becoming more breathless over the last week and feeling that his arms and face are puffy. He has had no chest pain, cough or fevers.

On examination his oxygen saturations are 92% on air and his heart rare is 112 beats per minute. His chest is clear and he is unable to lay flat for his abdomen to be examined due to is breathlessness. His face appears flushed and he has oedema of the arms and hands. His neck veins appear engorged.

Chest x-ray shows a widened mediastinum with extensive lymphadenopathy.

A CT scan of the thorax confirms superior vena cava obstruction (SVCO). The scan is reviewed by the haematology team who explain to him that his lymphoma has progressed despite chemotherapy.

He is started on steroids and a proton pump inhibitor.

What is the next step in the management of this gentleman's SVCO?

A. Chemotherapy
B. Observation
C. Radiotherapy
D. Stent insertion
E. Surgery

D. Stent insertion

SVCO is an oncological emergency and is most common in lung cancer and lymphomas. The immediate management is steroids with proton pump inhibitor cover for gastric protection.

Further options include stent insertion, radiotherapy and chemotherapy.

This gentleman is unable to lay flat so would not be able to tolerate radiotherapy. Radiotherapy also has the disadvantage of causing increased swelling acutely before it causes long term shrinkage.

Lymphomas are highly chemotherapy responsive tumours and this gentleman has only had firstline chemotherapy, therefore second-line chemotherapy should be planned in order to shrink his mediastinal disease.

Stent insertion is the most immediate management for relieving SVCO until chemotherapy can take effect. The patient's haemoglobin, platelets and clotting must be checked and optimised. In patients with end-stage disease, stents can still become blocked.

Surgery is not generally part of the management for SVCO.

Question 35 of 43

A 71-year-old woman is referred by her GP to the rapid access chest clinic with progressive shortness of breath and reduced exercise tolerance over 3 months and a chronic productive cough. When asked about her past medical history she states that she has had some pain for the past month or so in her left shoulder, which has been present at a constant level. She has been taking paracetamol and codeine for the pain which helps slightly. Otherwise, the patient has moderate COPD, for which she taken ipratropium bromide and salmeterol and mechanical back pain. She has a 15 year pack-history and drinks 1-2 units of alcohol per day.

Examination reveals some wasting of the hypothenar eminence of the left hand, although the chest sounds clear bilaterally with no added sounds. Heart sounds 1 and 2 are present with no murmurs.

What would be the most useful investigation following a chest x-ray?

A. Electromyogram studies
B. Computed tomography of the thorax
C. Bronchoscopy
D. Myeloma screen
E. Sputum microscopy, culture and sensitivity

B. Computed tomography of the thorax

The most likely diagnosis in this case is lung malignancy, in particular a Pancoast tumour, given this patient's wasting of the hypothenar eminence, shortness of breath and cough. The most useful investigation is a CT of the thorax - bronchoscopy is not as useful as the cancer is often too peripheral.

Question 36 of 43

You are asked to review a 67-year-old gentleman who was admitted to the medical emergency unit with pneumonia. He had been diagnosed with terminal small cell cancer of the lung which had extensively metastasised to his liver, thoracic vertebrae and femur bones. His past medical history was comprised of ischaemic heart disease, hypertension, hypercholesterolaemia and COPD.

His main complaint was of progressively worsening pain in his spine and legs. This was particularly bothersome at night; a trial of bisphosphonate therapy and radiotherapy did not provide any symptomatic relief. He was commenced initially on oral morphine sulphate solution which was converted to morphine sulphate tablets (MST); he had been on 60mg BD but this was titrated by the palliative care community nurse to 70mg BD a couple of days prior to admission with excellent analgesic effect. He was also using Oramorph solution 10mg on average 5-6 times a day for breakthrough pain.

He had been accordingly treated with intravenous antibiotics whilst on the ward making a full recovery. Prior to discharge, he was very keen on commencing transdermal treatment so as to minimise the number of oral medications taken.

What is the most appropriate starting dose to commence, with a view to completely discontinuing all oral opiate medication?

A. Commence Fentanyl 12 72 hr patch, paracetamol 1g QDS and oramorph solution PRN for breakthrough pain
B. Commence Fentanyl 25 72 hr patch, paracetamol 1g QDS and oramorph solution PRN forbreakthrough pain
C. Commence Fentanyl 50 72 hr patch, paracetamol 1g QDS and oramorph solution PRN forbreakthrough pain
D. Commence Fentanyl 75 72 hr patch, paracetamol 1g QDS and oramorph solution PRN forbreakthrough pain
E. Commence Fentanyl 100 72 hr patch, paracetamol 1g QDS and oramorph solution PRNfor breakthrough pain

D. Commence Fentanyl 75 72 hr patch, paracetamol 1g QDS and oramorph solution PRN forbreakthrough pain

This gentleman was on a rather complicated opiate analgesia regimen and the key to this question is calculate the total daily dose of morphine salt, to then convert it to the appropriate amount of fentanyl.

His total morphine salt intake was thus:

MST 70mg BD = 140mg of morphine Oramorph 10mg PRN = 50-60mg of morphine

Total = 190-200mg daily morphine salt intake

The BNF states that a fentanyl 75 patch is equivalent to 180mg daily intake of morphine salt whilst a fentanyl 100patch is equivalent to 240mg daily morphine salt intake. So as to avoid opiate toxicity the correct answer is thus to commence a fentanyl 75 patch and to supplement this with PRN Oramorph solution.

Question 37 of 43

A 68-year-old man presents with a five-week history of lower back pain that is worse with coughing. He has noticed that he has been requiring more painkillers to the point where he is struggling to manage, especially at night. The pain is worse when straining to pass stool. He lost three kg over the last four months as well. He has a past medical history of COPD, hypertension, type two diabetes mellitus, and hypercholesterolemia. He has been admitted three times in the last year for exacerbations of COPD requiring the recurrent use of oral prednisolone. His regular medications include Symbicort, tiotropium, atorvastatin, metformin, paracetamol, codeine and Movicol. He is an ex-smoker with a 50 pack-year history and recently opted for early retirement from his job as an accountant as he was struggling with tiredness. On examination, he appears tired and his clothes appear loose fitting. Examination of his chest is unremarkable, but there is a 1cm swelling above his left clavicle. His abdomen is soft, there is no tenderness over his spine, and a neurological examination is unremarkable.

Observations:

Saturations	95%
Respiratory rate	15/min
Blood pressure	148/87mmHg
Heart rate	74/min
Temperature	37.4°C

Blood tests:

Hb	128 g/l
MCV	72 fl
Platelets	327 * 10 /l9
WBC	12.4 * 10 /l9
Na +	136 mmol/l
K +	4.2 mmol/l
Urea	4.1 mmol/l
Creatinine	77 µmol/l

What is the most likely diagnosis that explains his symptoms?

A. Osteoporotic vertebral collapse
B. Spinal cord metastasis
C. Myeloma
D. Degenerative changes
E. Discitis

B. Spinal cord metastasis

The answer is spinal cord metastasis. This question highlights multiple subtle red flags in a history with back pain. He is over >55 and describes nocturnal and progressive pain worse with coughing and straining in the context of immunosuppression from repeated steroid use, weight loss, and increasing fatigue. Due to the high suspicion, the most diagnostic imaging investigation would be a whole spine MRI, which would differentiate between the above diagnoses. Osteoporotic fracture is suggested by the use of steroids but overall does not fit with the progressive nature and microcytic anaemia, nor do degenerative changes. There are very few symptoms to suggest infection such as fever and night sweats, but discitis cannot be fully excluded without imaging. Myeloma is a possible diagnosis, but so far there are no specific factors to suggest it, such as lytic bone lesions or deteriorating renal function.

Question 38 of 43

A 56-year-old male with non-hodgkin's lymphoma is on his third cycle of R-CHOP chemotherapy. He has struggled previous times with nausea but is two days following the most recent cycle and attended hospital due to intractable vomiting.

He is vomiting 20+ times a day with green to clear fluid. He does not feel hungry and struggles to swallow water. He is not having diarrhoea. He takes regular indapamide and as required ondansetron and this has not helped him despite the oncologist suggesting he double his dose of ondansetron.

On examination, he looks pale and has dry mucosa. He is alert, has a clear chest and soft abdomen but begins to retch on sitting forward. He last passed urine 6 hours ago and his observations show a heart rate of 115 beats per minute and blood pressure 90/65mmHg. He is started on intravenous fluids by the emergency department.

Na +	135 mmol/l
K +	3.5 mmol/l
Urea	6.5 mmol/l
Creatinine	98 µmol/l

What medical intervention would offer greatest benefit?

A. Cyclizine
B. Potassium salt replacement
C. Elective terminal ileostomy
D. Dexamethasone
E. Delay next course of chemotherapy

D. Dexamethasone

Dexamethasone with ondansetron is effective in refractory chemotherapy-induced vomiting

This gentleman has refractory vomiting despite ondansetron. He need assistance to ensure he can maintain fluids. Cyclizine is an antiemetic but bind histamine receptors so is most useful in labyrinthitis and motion sickness. Potassium salt replacement will probably make him vomit more but does replace his lost salts. Delaying the course of chemotherapy would be an option if nothing else could be offered. Surgery is not the way forward here. Dexamethasone is proven as an effective second line antiemetic in chemotherapy related nausea and vomiting. it is often given in lymphoma to prevent tumour lysis syndrome.

Question 39 of 43

A 38-year-old man presents with increasing shortness of breath and ankle swelling. He reports he is more short of breath, especially on exertion, which is not being relieved by the use of his salbutamol inhaler. He has also noticed his ankles are swollen by the end of the day.

His past medical history includes asthma, which is usually well controlled, and lymphoma, which was treated with chemotherapy several years ago. His only medications are his beclometasone and salbutamol inhalers. The results of his blood tests are shown below:

Hb	140 g/L	(135-180)
Platelets	359 * 10 /L 9	(150 - 400)
WBC	7.2 * 10 /L 9	(4.0 - 11.0)
Na +	137 mmol/L	(135 - 145)
K +	4.3 mmol/L	(3.5 - 5.0)
Urea	5.9 mmol/L	(2.0 - 7.0)
Creatinine	119 µmol/L	(55 - 120)

His ECG showed sinus rhythm with left axis deviation. An echocardiogram was done which showed dilation of all four chambers of the heart and a left ventricular ejection fraction of 40%.

Which drug can be attributed to causing this man's presentation?

A. Cyclophosphamide
B. Vincristine
C. Doxorubicin
D. Docetaxel
E. Bleomycin

C. Doxorubicin

Anthracyclines (e.g. doxorubicin) may cause cardiomyopathy

This man is presenting with features of heart failure and the echocardiogram results would be in keeping with a diagnosis of dilated cardiomyopathy. Doxorubicin (an anthracycline) is used in the treatment of lymphoma and is known to be cardiotoxic.

Cyclophosphamide can also be used to treat lymphoma and other haematological conditions such as leukaemia. A well-known side effect is haemorrhagic cystitis.

Vincristine can be used with doxorubicin to treat lymphoma and is associated with peripheral neuropathy.

Docetaxel is often used in the management of breast and lung cancers and can cause neutropenia.

Bleomycin can be used as part of lymphoma management and is associated with the development of pulmonary fibrosis.

Question 40 of 43

You are reviewing a 65-year-old man who has recently started treatment for urological cancer with Nivolumab (Opdivo) a type of checkpoint inhibitor.

He has been having treatment for 1 month and has had no side effects so far. He would like to know what to expect from this treatment.

What is the most common side effect this patient is likely to experience?

A. Dry itchy skin and rashes
B. Depression and anxiety
C. Dry cough
D. Retinal damage
E. Skin discolouration

A. Dry itchy skin and rashes

The most common side effect of immune checkpoint inhibitors is dry itchy skin and rashes

Immune checkpoint inhibitors are a type of immunotherapy that are increasingly being used to treat certain types of cancer, as an alternative to cytotoxic chemotherapy. They are currently only used to treat solid tumours. They work by enhancing the bodies own T-cell population to destroy the cancer cells. Their side effect profile reflects their mechanism of action and are, therefore, inflammatory and autoimmune in nature. They include:

- Dry, itchy skin and rashes (most commonly)
- Nausea and vomiting
- Decreased appetite
- Diarrhoea
- Tiredness and fatigue
- Shortness of breath and a dry cough.

Therefore, option one is the only correct answer.

Question 41 of 43

A 54-year-old male presents to the emergency department with sudden onset palpitations and shortness of breath which began whilst he was watching television a few hours previously. He describes an odd fluttering sensation in his chest with mild dyspnoea but no chest pain. He denies having experienced this before. On systems review, he also complains of severe muscle cramps and a slight tremor in his hands over the last few days. His past medical history includes hypertension and gout for which he takes amlodipine and allopurinol. He denies any history of exertional dyspnoea but does occasionally get central chest pain on exertion for which he has not sought help. He has recently been diagnosed with small cell lung cancer and underwent his first round of chemotherapy last week; although he is unsure which drugs are being used. He used to smoke a pipe until his recent diagnosis of cancer and drinks a few measures of whisky most evenings.

On examination, his respiratory rate is 22 breaths/min and his oxygen saturations are 96% breathing two litres of oxygen. His heart rate is irregularly irregular and approximately 130 beats/min, his blood pressure is 152/78 mmHg. An ECG shows atrial fibrillation with no ischaemic changes.

What is the most likely cause for his new atrial fibrillation?

A. Ischaemic heart disease
B. Chemotherapy induced hypokalaemia
C. Chemotherapy induced hypomagnesaemia
D. Chemotherapy induced cardiomyopathy
E. Alcohol

C. Chemotherapy induced hypomagnesaemia

The most common chemotherapy regime for small cell lung cancer is etoposide and cisplatin. A common side effect of cisplatin is hypomagnesaemia which can result in muscle cramps, tremor, cardiac arrhythmias, confusion amongst other features. This gentleman appears to have angina and may well have some level of ischaemic heart disease in the background which would increase his risk of atrial fibrillation. However, it seems logical given the strong temporal relationship between the chemotherapy and the other symptoms of hypomagnesaemia that this is the cause.

Question 42 of 43

A 63-year-old man is diagnosed with colorectal cancer. Following successful resection he starts adjuvant capecitabine but after six doses develops severe diarrhoea, weakness and desquamation of the hands, feet and face. On admission, he is pancytopenic and has electrolyte abnormalities requiring IV replacement. His initial blood tests are shown bellow.

Na +	140 mmol/l
K +	3.6 mmol/l
Urea	9.2 mmol/l
Creatinine	125 µmol/l
Hb	75 g/l
Platelets	35 * 10 /l9
WBC	0.4 * 10 /l9
Neutrophils	0.03 * 10 /l9
Adjusted Ca 2+	1.5 mmol/l
Mg 2+	0.32 mmol/l
Glucose	4.0 mmol/l

What is the most likely cause?

A. DPD (dihydropyrimidine dehydrogenase) deficiency
B. Infective gastroenteritis
C. Cancer recurrence
D. Pityriasis alba
E. Neutropenic sepsis

A. DPD (dihydropyrimidine dehydrogenase) deficiency

Capecitabine is the oral analog of 5-fluorouracil, a chemotherapeutic agent which is broken down, predominantly, by dihydropyrimidine dehydrogenase (DPD). Deficiency is autosomal recessive and will lead to a toxin buildup which in homozygous patients is usually fatal. The severe toxicity experienced by this patient are likely due to a heterozygous DPD deficiency.

Question 43 of 43

A 53 year old patient has undergone six cycles of R-CHOP (rituximab, cyclophosphamide, doxorubicin, vincristine and prednisolone) chemotherapy and is now attending the lymphoma follow up clinic. He complains of an ongoing and unrelenting tingling in his feet and legs as well as his right hand. This is associated with pain in the extremities that is worsened by the cold weather.

Examination reveals normal motor strength throughout all 4 limbs but marked reduction in proprioception in both legs below the knee and also the right hand. The is a 'glove and stocking' distribution of loss when tested for pin-prick sensation and a reduced joint position sense to the ankle in both lower limbs. Plantar reflexes are normal. During the examination you it is noted that the patient has sustained an injury to the sole of foot which he was unaware of.

What is the most likely causative agent for these symptoms?

A. Rituximab
B. Cyclophosphamide
C. Doxorubicin
D. Vincristine
E. Prednisolone

D. Vincristine

The symptoms being experienced above are classical for chemotherapy induced peripheral neuropathy. This is a condition that can occur in up to 40% of patients being given chemotherapy with agents such as platinum based drugs (cisplatin and oxaliplatin), vinca alakloids (vincristine and vinblastine), thalidomide and taxanes (paclitaxel and docetaxel). The treatment of chemotherapy induced peripheral neuropathy is difficult with patients having severe problems long after the causative agent has been given. In most cases it is irreversible. The best medication for treatment is gabapentin or pregabalin which should be titrated up to therapeutic levels and used in conjunction with another analgesic agents.

There are ongoing trials aiming to find a prophylactic agent to help prevent this painful sequealae of treatment, but as yet there has been no convincing evidence to suggest any one treatment.

Alrajhi Jad

PASSMEDICINE

INFECTIONS

Internal medicine

EDITED BY
DR. JAMIL ALRAJHI

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773-555-493
jameelalrajhi@gmail.com

Question 1 of 277

A 44-year-old man is brought to the emergency department by his wife with an acute onset of confusion. She reports that her husband had only just recently recovered from a bout of nausea and vomiting for the last 10 days since returning home from visiting family in Vietnam. He has no past medical history of note and takes no regular medications. His sister was treated for tuberculosis 6 months ago.

His observations are as follows:

- Temperature 38.3ºC
- Heart rate 116 beats/min
- Blood pressure 112/76 mmHg
- Respiratory rate 16 breaths/min
- Saturations 96% on air

On examination, he is disoriented to time and place but recognises his wife. He complains of a severe headache but the remainder of his neurological examination is unremarkable. On auscultation, his chest is clear and heart sounds are normal. His abdomen is soft and non-tender.

What is the most likely diagnosis?

A. Dengue fever
B. Disseminated tuberculosis
C. Japanese encephalitis
D. Meningitis
E. Yellow fever

C. Japanese encephalitis

Confusion in returned traveller from Asia -> Japanese encephalitis

This patient has **Japanese encephalitis**. Spread by Culex mosquitoes that breed in rice paddy fields, Japanese encephalitis is the most common cause of encephalitis in South East Asia, China and India. Most cases are asymptomatic. However, in more severe cases, symptoms include headache, fever, seizures and confusion. A prodrome of nausea and vomiting may be seen initially, as described by this patient. This patient is presenting with acute confusion and headache following a trip to Vietnam and therefore, a diagnosis of Japanese encephalitis is likely.

Dengue fever is a viral illness that presents with fever, headache and myalgia. A maculopapular rash early on in the illness is common, along with facial flushing. In some patients, the disease can progress to a viral haemorrhagic fever and dengue shock syndrome. However, it does not present with symptoms of acute encephalitis.

Disseminated tuberculosis is a less likely answer. Typically tuberculosis affects the respiratory system and systemic symptoms are present including fever, night sweats and weight loss. Although his sister was recently treated for tuberculosis, the absence of these constitutional symptoms and the more abrupt onset of illness suggests an alternative diagnosis.

Meningitis can have a viral or bacterial cause. However, the presenting symptoms are similar and include fever, headache and meningism. Vomiting may also feature. However, the absence of photophobia and meningism and the acute onset of confusion makes a diagnosis of encephalitis more likely than meningitis.

Yellow fever is a viral haemorrhagic illness common in Central and South America. It presents as a mild flu-like illness followed by a brief remission period before the onset of haematemesis, jaundice and oliguria. The presenting symptoms in this question do not describe any form of haemorrhage and are not typical of yellow fever, suggesting that it is not the underlying diagnosis.

Question 2 of 277

A 34-year-old man presents to the emergency department with a three-week history of mild right upper quadrant (RUQ) pain. He feels sweaty and feverish. He reports a three-week holiday to Indonesia one-year ago.

His observations are:

- Respiratory rate: 19/min
- Oxygen saturation: 98% on room air
- Heart rate: 104bpm
- BP: 138/72mmHg
- Temperature: 38.0 C
- GCS: 15/15

On examination, the patient is tender in the RUQ and appears sweaty. Otherwise, he examines normally.

An abdominal ultrasound is performed which shows a 7cm abscess in the right lobe of the liver. Stool microscopy shows spherical cysts.

What is the most appropriate treatment?

A. Abscess aspiration
B. Amoxicillin + ciprofloxacin + metronidazole
C. Metronidazole for 1 week
D. Metronidazole for 1 week, then diloxanide for 1 week
E. Praziquantel

D. Metronidazole for 1 week, then diloxanide for 1 week

Treatment for invasive amoebiasis should be followed by a luminal amoebicide to eradicate the cystic stage which is resistant to metronidazole and tinidazole (which are used against the invasive stage)

Metronidazole for 1 week, then diloxanide for 1 week is the correct answer. This patient has amoebiasis, with the spherical cysts representing *Entamoeba histolytica* cysts. The incubation period is from days to years. Amoebiasis has a worldwide distribution but is more common in tropical and subtropical regions. Metronidazole treats Entamoeba trophozoites (but has insufficient activity against cysts). Diloxanide or paromomycin are luminal agents that are active in the bowel - they treat the Entamoeba cysts but are poorly absorbed, and so are ineffective against organisms outside the bowel. Therefore a combination of medications is required.

Abscess aspiration is the incorrect answer. Indications for abscess aspiration in amoebiasis are:

- Abscesses in the left liver lobe (possible rupture into pericardium)
- Abscesses >10cm in diameter
- Severe pain
- Marked diaphragm elevation
- Abscesses that fail to respond to medical management Impending abscess rupture

Amoxicillin + ciprofloxacin + metronidazole is the incorrect answer. This is the recommended treatment for bacterial liver abscesses, but this will not treat amoebiasis. Patients with bacterial liver abscesses are likely to be significantly more unwell (septic). They will also not have the characteristic cysts or trophozoites on stool microscopy.

Metronidazole for 1 week is the incorrect answer. Metronidazole will treat the Entamoeba trophozoites, but not the cysts.

Praziquantel is the incorrect answer. Praziquantel is an anti-helminthic drug and will not treat *Entamoeba histolytica*, which is a protozoan. Praziquantel (in combination with percutaneous aspiration) can be used to treat hydatid liver cysts caused by *Echinococcus granulosus*. Hydatid cysts tend to be multi-loculated and asymptomatic.

Question 3 of 277

A 54-year-old man presents to the emergency department with two days of pain and a rash on his right thigh. He has recently had the chickenpox for the first time and was given a course of aciclovir, which he has now completed. The lesions have now healed. He has type 2 diabetes and is on metformin. He smokes ten cigarettes daily.

His observations are heart rate 121 beats per minute, respiratory rate 24/minute, blood pressure 85/45 mmHg, oxygen saturations 97% on air and temperature 39.4ºC.

On examination, he appears unwell. He is diaphoretic and anxious. There is a tense erythematous area on his right thigh, which is severely painful. His pulse is bounding and there is a flow murmur heard in the aortic area.

Blood tests:

Hb	121 g/L	Male: (135-180)Female: (115 - 160)
Platelets	580 * 10 /L ^{9}	(150 - 400)
WBC	25.2 * 10 /L ^{9}	(4.0 - 11.0)
Na $^+$	137 mmol/L	(135 - 145)
K $^+$	4.1 mmol/L	(3.5 - 5.0)
Urea	11.4 mmol/L	(2.0 - 7.0)
Creatinine	176 µmol/L	(55 - 120)
CRP	380 mg/L	(< 5)
Bilirubin	25 µmol/L	(3 - 17)
ALP	88 u/L	(30 - 100)
ALT	89 u/L	(3 - 40)
Î³GT	45 u/L	(8 - 60)
Albumin	29 g/L	(35 - 50)

Despite aggressive fluid resuscitation, his blood pressure remains 86/51 mmHg. \

Given the likely diagnosis, what is the most important treatment?

A. Hyperbaric oxygen therapy
B. Intravenous flucloxacillin
C. Intravenous immunoglobulin
D. Surgery
E. Tazocin and clindamycin

D. Surgery

Chickenpox is a risk factor for invasive group A streptococcal soft tissue infections including necrotizing fasciitis

Surgery is the correct answer. This man has septic shock secondary to necrotizing fasciitis as evidenced by fever, hypotension, rash and pain out of proportion to the clinical findings. Furthermore, he has evidence of end-organ dysfunction (acute kidney injury). The most important treatment for necrotizing fasciitis is surgical debridement of infected tissue. All other treatments are considered adjunctive. Both recent chickenpox infection and diabetes are risk factors for this condition.

Hyperbaric oxygen therapy is incorrect. There have been case reports of this treatment being used for necrotizing fasciitis but it is not the mainstay of treatment.

Intravenous flucloxacillin is incorrect. This antibiotic would be a good choice for simple cellulitis but necrotizing fasciitis is a more severe infection, typically caused by gram-negative, grampositive and anaerobic bacteria and therefore requires a broader antibiotic cover.

Intravenous immunoglobulin is incorrect. This can be used as adjunctive treatment to antibiotics and surgery in severe skin and soft tissue infections but is not initial therapy or most important.

Tazocin and clindamycin are incorrect. This is the correct antibiotic choice for necrotizing fasciitis as it gives a broad cover against aerobic and anaerobic organisms and clindamycin has the added benefit of inhibiting toxin production. However, the literature on necrotizing fasciitis is clear in regarding surgery as the most important intervention.

Question 4 of 277

A 31 year-old male originally from Russia who is known to be HIV positive presents with a rash. On examination there are purple-red cutaneous macules and papules on the back, neck and oral mucosal membrane.

Blood results reveal:

Hb	141 g/l
Platelets	327 * 10 /l^9
WBC	4.2 * 10 /l^9
Na $^+$	141 mmol/l
K $^+$	3.7 mmol/l
Urea	4.6 mmol/l
Creatinine	59 µmol/l
Bilirubin	27 µmol/l
ALP	97 u/l
ALT	44 u/l
Albumin	30 g/l
HIV viral load	14,000 copies / ml
CD4 cell count	124 cell/mm^3

A skin biopsy is performed and the report is below:

Spindle cells present. Mitotic activity is moderate. Abnormally dense and irregular blood vessels. Intracellular hyaline bodies present.

Which virus is likely to be responsible for these lesions?

A. CMV
B. EBV
C. HIV-2
D. HHV-8
E. HPV-8

D. HHV-8

Kaposi's sarcoma - caused by HHV-8 (human herpes virus 8)

Intracellular hyaline bodies present.

This is a description of AIDS-related Kaposi's sarcoma.

Kaposi sarcoma is a disease of blood vessels that was considered very rare before the start of the AIDS pandemic. AIDS is due to infection with human immunodeficiency virus (HIV).

There are four types of Kaposi sarcoma:

- The classic type of Kaposi sarcoma affects elderly men of Mediterranean and Middle European descent and in men in Sub-Saharan Africa.
- AIDS-associated Kaposi sarcoma.
- Endemic or African Kaposi sarcoma arises in some parts of Africa in children and young adults.
- Iatrogenic Kaposi sarcoma is due to drug treatment causing immune suppression.

Classic Kaposi sarcoma is rare and unassociated with HIV infection. It most often arises in middleaged to elderly men of Mediterranean or Jewish descent (less than 10% are women), particularly if they come from a rural environment. They have a higher than expected rate of diabetes mellitus.

AIDS-related Kaposi sarcoma, unlike other forms of the disease, tends to have an aggressive clinical course. It is the most common presentation of Kaposi sarcoma. It has become less common in the US and Europe because of effective HAART treatment for HIV disease.

Kaposi's sarcoma is caused by infection with human herpesvirus 8 (HHV8), also known as Kaposi sarcoma-associated herpesvirus (KSHV) or KS agent

Typical histologic findings include proliferation of spindle cells, prominent slit-like vascular spaces, intracellular hyaline bodies and extravasated red blood cells.

Question 5 of 277

A 24-year-old man presents to the emergency department with a sudden onset of fever, headache and malaise, in association with diarrhoea and cramping abdominal pain. He has no past medical history and is on no regular medications. He returned from the 'python cave' in the Maramagambo Forest in western Uganda six days previously. He had visited the cave to view the colonies of Egyptian fruit bats. He tells you he strictly followed precautions to prevent mosquito bites and took malaria prophylaxis for the duration of his time there.

His observations are heart rate 120 beats per minute, blood pressure 83/59 mmHg, respiratory rate 21/minute, oxygen saturations 95% on room air and temperature 39.4ºC.

On examination, he is confused with a Glasgow coma scale of 14/15 and is mildly jaundiced. There is no meningism or nuchal rigidity. His abdomen is moderately tender without evidence of peritonism. Chest auscultation is normal. His capillary refill time is 4 seconds, he has dry mucus membranes and decreased skin turgor. His heart sounds are normal with a soft systolic flow murmur audible.

Intravenous access is obtained and it is noted that he is oozing blood from his cannulae sites and also from his oral mucosa.

Urinalysis is unremarkable.

An ECG demonstrates sinus tachycardia.

Blood tests:

Hb	98 g/L	Male: (135-180)Female: (115 - 160)
Platelets	487 * 10 /L 9	(150 - 400)
WBC	3.8 * 10 /L 9	(4.0 - 11.0)
Na +	131 mmol/L	(135 - 145)
K +	5.1 mmol/L	(3.5 - 5.0)
Urea	13.1 mmol/L	(2.0 - 7.0)
Creatinine	280 µmol/L	(55 - 120)
CRP	50 mg/L	(< 5)
Bilirubin	84 µmol/L	(3 - 17)
ALP	267 u/L	(30 - 100)
ALT	432 u/L	(3 - 40)
Î³GT	180 u/L	(8 - 60)
Albumin	28 g/L	(35 - 50)
Prothrombin time	18.4 seconds	(11-14)

Plain radiography of the chest is normal. Three thick and thin blood films are negative.

What is the most likely diagnosis?

A. Hepatitis A
B. Leptospirosis
C. Malaria
D. Marburg virus
E. Typhoid

D. Marburg virus

Exposure to bats/caves is a risk factor for Marburg virus, which results in a viral haemorrhagic fever

Marburg virus is the correct answer. The patient has been to a specific region in Uganda that has previously been associated with outbreaks of Marburg virus. Exposure to bats and caves is also a risk factor for this viral hemorrhagic fever. This condition typically presents with an abrupt onset of fever, headache and malaise and progresses to include gastrointestinal symptoms, arthralgia/myalgia and subsequently haemorrhagic complications with multi-organ dysfunction as evidenced in this case. The incubation period before the onset of symptoms is typically 3-10 days. In addition to the geographic and zoonotic risk factors, it is the only diagnosis listed that could account for the haemorrhagic complications in this scenario (oozing from venepuncture sites and bleeding from mucosal membranes). Treatment is supportive and the condition is associated with a high mortality rate.

Hepatitis A is incorrect. While this could account for jaundice and transaminitis, it is typically a selflimiting illness and associated with fulminant liver failure only in very rare cases. It is not associated with haemorrhagic complications.

Leptospirosis is incorrect. While this condition can present with fever, headache and liver dysfunction, the traditional vectors for transmission are mice, rats, cows and pigs. It is transmitted classically by coming into contact with infected urine. While it is associated with subconjunctival haemorrhage, it is not usually associated with more diffuse haemorrhage as seen in this case. It is treated with antibiotics.

Malaria is incorrect. While this is the most common cause of the fever in the returning traveller, the patient has taken malaria prophylaxis and has had negative testing for malaria in addition to experiencing haemorrhagic complications, all of which make a diagnosis of malaria unlikely.

Typhoid is incorrect. While this is a common cause of fever in the returning traveller and is associated with fever and diarrhoea, it would not account for the haemorrhagic complications detailed in this scenario.

Question 6 of 277

A 35-year-old man presents to the travel walk-in centre due to a week of diarrhoea following a recent holiday to Brazil. He had a stool sample sent for ova, cysts, and parasites which tested positive for *Strongyloides stercoralis*.

What medication should be prescribed for the patient?

A. Azithromycin
B. Diethylcarbamazine
C. Ivermectin
D. Praziquantel
E. Triclabendazole

C. Ivermectin

Ivermectin is the drug of choice for treatment of strongyloidiasis

Ivermectin is the drug of choice for strongyloidiasis which acts by deactivating chloride channels which result in paralysis and death of the parasite either directly or by causing the worms to starve.

Azithromycin is an effective antibiotic for *Typhoid*, *Campylobacter* and often treatment of choice for traveller's diarrhoea. It is not effective for treatment of *Strongyloides*.

Diethylcarbamazine is not used to treat Strongyloides. It is the treatment of choice for *Wuchereria bancrofti*, *Loa Loa*, *Toxocara canis*.

Praziquantel is also not an effective treatment for Strongyloides. It is used to treat *Schistosoma haematobium*, *Paragonimus westermani* and *Clonorchis sinensis*.

Triclabendazole is the treatment of choice for *Fasciola hepatica* (the liver fluke) and is not the treatment of choice for Strongyloides.

Question 7 of 277

A 29-year-old man presented with fever, headache, a dry cough, and bloody diarrhoea. Symptoms started eight days ago. Eight weeks earlier he had been on an adventure holiday in Zambia with a group of friends, which involved rafting and visiting a game reserve. A couple of his friends had developed a similar illness. On examination, he was febrile, and was noted to have an urticarial rash on his trunk. He had a polyphonic wheeze in both lung fields and the tip of his spleen was palpable.

Hb	134g/l (130 - 180)
Platelets	350 * 10 /l (150 - 400)9
WBC	12.2 * 10 /l (4.0 - 11.0)9
Neutrophil	6.5 * 10 /l (1.5 - 7.0)9
Lymphocyte	1.7 * 10 /l (1.5 - 4.09
Eosinophil	3.4 * 10 /l (0.04 - 0.4)9
Monocyte	1.5 * 10 /l (0<0.8)9

What is the pathogen causing the patient's symptoms?ssMedicine

A. Dengue virus
B. Epstein-Barr virus
C. Human immunodeficiency virus
D. Leptospira icterohaemorrhagica
E. Schistosoma mansoni

E. Schistosoma mansoni

Acute schistomiasis is usually associated with marked eosinophilia, cough, bloody diarrhoea, and splenomegaly

The patient has a marked eosinophilia, and presents with systemic symptoms, cough, bloody diarrhoea, and splenomegaly eight weeks after exposure to fresh water in Africa. The most likely diagnosis in this scenario is Katayama syndrome, or acute schistosomiasis, caused by infection with a small blood fluke. It most often occurs with Schistosoma japonicum and schistosoma mansoni infections but can occur with any species. Acute schistosomiasis should be treated with praziquantel 40-60 mg/kg as a single dose and short course of steroids to control the inflammatory reaction. The dose of praziquantel should be repeated after six weeks when flukes are fully mature because immature flukes are relatively resistant to treatment.

Eosinophilia is not a feature of glandular fever, acute dengue, EBV infection, or HIV. The incubation period is too long for leptospirosis and dengue, which typically is 3-14 days.

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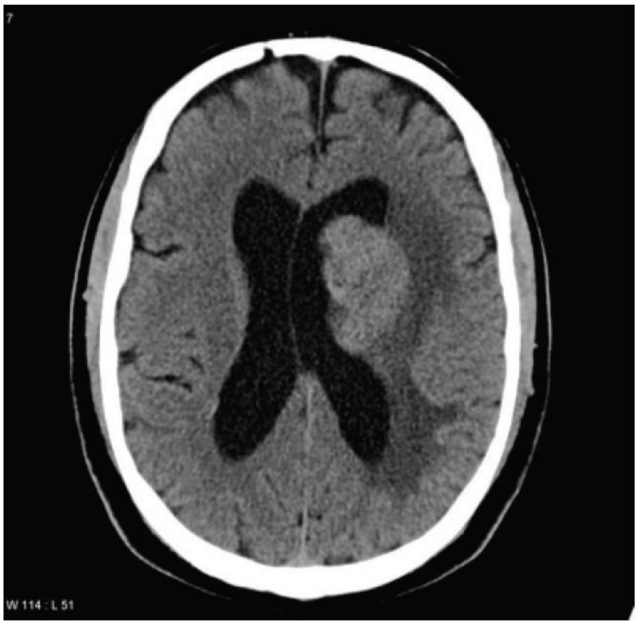
A 41-year-old man is admitted to the Emergency Department after having a seizure. He has no history of epilepsy but is known to be HIV positive. On examination he is still slightly confused post-ictally but there are no focal neurological signs. He is apyrexial and haemodynamically stable. His current medications includes efavirenz, tenofovir, lamivudine and co-trimoxazole. His latest CD4 count is 38 cells/µl. CT scan (without contrast) is shown below:



What is the most likely diagnosis?

A. Cerebral toxoplasmosis
B. Progressive multifocal leukoencephalopathy
C. Cryptococcal infection
D. CNS lymphoma
E. Cerebral tuberculosis

D. CNS lymphoma

HIV, neuro symptoms, single brain lesions with homogenous enhancement - CNS lymphoma

A hyper-attenuating mass adjacent to the left lateral ventricle is seen. The homogenous appearance, position and history (low CD4 count and current use of co-trimoxazole prophylaxis) are suggestive of primary CNS lymphoma rather than cerebral toxoplasmosis.

Question 9 of 277

A 32-year-old nurse presents to the the emergency department on the advise off occupational health. She contacted occupational health after she was coughed upon by a 25-year-old patient who is being treated with empirical antibiotics for bacterial meningitis. She felt contact on her face with droplets at the time. She has been feeling entirely well in herself. She has had appendicitis in the past as well as polycystic ovaries, but has no other medical problems. She takes metformin and oral contraception pills.

The 25-year-old patient was admitted 12 hours ago and has had a lumbar puncture which has confirmed meningococcal meningitis.

What is the most appropriate plan for the nurse?

A. Monitor for symptoms
B. Lumbar puncture
C. Give oral ciprofloxacin
D. Admit to hospital for IV antibiotics

E. Advise that the risk of transmission is not significant

C. Give oral ciprofloxacin

People who have been exposed to a patient with confirmed bacterial meningitis should be given prophylactic antibiotics if they been exposed to respiratory secretion, regardless of closeness of contact

This is a patient who has had a close contact with respiratory secretions of a person with confirmed meningococcal meningitis. Despite the fact that she has not had prolonged exposure, prophylaxis in the form of oral ciprofloxacin is needed.

The risk of transmission by this route is sufficiently significant to make monitoring for symptoms or not giving any treatment inappropriate.

A lumbar puncture is unlikely to be a helpful investigation for her as it cannot exclude that she will develop meningitis.

IV antibiotics are used to treat meningitis, whilst oral antibiotics can be used for prophylaxis of meningococcal meningitis.

Question 10 of 277

A 35-year-old woman presents to the ambulatory medical care unit with a 6-week history of an ulcer on the back of her right hand. It started as a 1cm painful nodule and gradually enlarged. It is now 3cm in diameter and is a firm raised purple/red lesion with some central ulceration. There are no lesions anywhere else and she is systemically well. She has no known past medical history and has not travelled outside of the UK in the last 5 years. She has no pets but works in a pet shop which specialises in tropical fish.

What organism is the most likely cause of this lesion?

A. Herpes simplex virus
B. Leishmania braziliensis
C. Mycobacterium marinum
D. Mycobacterium ulcerans
E. Sporothrix schenckii

C. Mycobacterium marinum

Fish tank granuloma is caused by *Mycobacterium marinum*

This presentation is typical of fish tank granuloma, which is caused by *Mycobacterium marinum*. The main clue is her exposure to fish tanks and lack of risk factors for the other organisms. As well as via contact of skin with the water in fish tanks or aquariums, the infection can less commonly be acquired through preparing fish or shellfish, or contact with fishing equipment. It generally presents as a slow-growing lesion, although it may disseminate or progress more quickly in immune-suppressed people. *Mycobacterium marinum* grows most effectively at 30 degrees, and so the lesions are usually found at the extremities.

Herpes simplex virus can cause lesions on the hand if the skin of the hand comes into contact with infected skin around the mouth or genitals - this is called herpetic whitlow. However, the lesion would usually be on the fingertips rather than the back of the hand, would start with vesicles rather than a nodule, and would generally progress more quickly.

Leishmania braziliensis causes cutaneous and mucocutaneous leishmaniasis and is found in Central and South America. Cutaneous leishmaniasis may present similarly to fish tank granuloma, however, the patient has not travelled abroad.

Mycobacterium ulcerans causes painful ulcers on the skin which are often called Buruli ulcers - however, it is found predominantly in tropical regions.

Sporothrix schenckii: is a dimorphic fungus which can cause similar lesions on the hands and arms after contact with infected plant matter - it is often referred to as rose gardener's disease. There tend to be several lesions which spread up the lymphatic system.

Question 11 of 277

A 23 year-old student presents with a febrile illness, seven days after return from holiday in Thailand. He delayed seeking medical attention because he thought it was 'just a virus', and he had experienced similar self-limiting symptoms when travelling to Vietnam several years earlier. He did not take malaria prophylaxis due to a previous adverse reaction to mefloquine. The illness began two days after his return, when he recorded his temperature at 38.4°C. He also complains of severe generalised joint pains and headache which he describes as 'behind the eyes'. Today he has developed nosebleeds and when he brushed his teeth this morning he noticed that his gums bled.

On examination his temperature is 38.2°C and there is a widespread macular rash with islands of sparing. There are some petechial haemorrhages on the limbs. Cardio-respiratory examination is unremarkable except for tachycardia of 114. Blood pressure is 123/79 mmHg. On palpation of the abdomen you discover tender hepatomegaly. Neurological examination is unremarkable.

On application of a tourniquet to take a blood sample you notice that the area under the tourniquet has become bruised.

Results of blood tests are as follows:

Hb	14.1 g/dl
MCV	94.2 fl
Haematocrit	0.57
Platelets	23 x10^9/l
WCC	6.1 x10^9/l
Na +	134 mmol/l
K +	4.6 mmol/l
Urea	3.8 mmol/l
Creatinine	80 µmol/l
ALT	64 IU/l
ALP	78 IU/l
Bilirubin	13 mol/l
Albumin	28 g/l
Rapid Malaria test	Negative

Thick and thin blood films	Pending

What is the most likely diagnosis?

A. Non-falciparum malaria
B. Dengue haemorrhagic fever
C. Lassa fever
D. Meningococcal septicaemia
E. HIV seroconversion

B. Dengue haemorrhagic fever

Retro-orbital headache, fever, facial flushing, rash, thrombocytopenia in returning traveller → ?dengue

This is a viral haemorrhagic fever, and the clinical picture is classic for dengue. The disease is endemic in South-East Asia. A defective immune response in secondary infections means that individuals who have been infected with one viral serotype may develop more severe disease when later infected with a different serotype. This patient may have developed self-limiting dengue fever on a previous trip to Vietnam, and now has secondary infection.

Dengue fever typically begins 7-10 days after infection with a rapid onset of fever, severe myalgia, and retro-orbital headache. A maculopapular rash with islands of sparing is characteristic. In the majority of cases there is self-resolution after 2-7 days. In a proportion of cases however, especially in secondary infection, the disease progresses to a more fulminant form known as dengue haemorrhagic fever. There is widespread vascular breakdown, with easy bleeding and bruising. Thrombocytopaenia is typical. Plasma leakage results in haemoconcentration, raising the haemocrit, and loss of plasma proteins into the extracellular space is reflected by the low albumin. The liver is often enlarged and tender, with deranged liver function tests. In severe cases this progresses to circulatory collapse and dengue shock syndrome. Diagnosis is by PCR (polymerase chain reaction) during the initial febrile phase and serology during the later stages. Management is supportive.

Non-falciparum malaria includes disease caused by *Plasmodium vivax* and *ovale*, and to a lesser extent *malariae* and *knowlesi*. *Vivax* is common in Thailand and typically causes less severe disease. Most malaria rapid diagnostic tests are antigen assays that detect only falciparum. Microscopy is the only way to confirm or exclude the diagnosis.

Lassa fever is a viral haemorrhagic fever endemic to West Africa.

Meningococcal septicaemia is a possibility, although one would expect him to be more unwell after five days of disease.

HIV seroconversion illness does not commonly cause bleeding manifestations.

Question 12 of 277

A 45-year-old man is urgently referred to dermatology clinic by his general practitioner after presenting with erythema migrans on his left calf. The patient was very concerned about the possibility that he had contracted Lyme disease following his recent walking holiday in the Scottish highlands.

The patient reported returning from his holiday 2 weeks ago and first noticed the rash within the last 24 hours. The patient reported that he had undertaken long walks in the countryside wearing shorts but that he had not noticed any ticks becoming attached to his body during his holiday. Aside from the rash on his leg (described below) the patient reported no other symptoms at the present time or during recent weeks. In particular, the patient denied any symptoms involving the central or peripheral nervous systems.

The patient was in generally good physical health and his only past medical history was hypertension treated with ramipril by his GP. The patient had no known allergies to any medications.

Examination of the patient's left leg demonstrated a circular erythematous rash 15 cm in diameter. The rash demonstrated an appearance typical for erythema migrans, featuring a central spot surrounded by a ring of clear skin and an outer ring of erythema. The patient reported that the area covered by the rash had expanded significantly in the 24 hours since he had first noticed it. A detailed examination of the remaining surface of the patient's skin did not identify any other significant rashes. Examination of the peripheral and central nervous system did not demonstrate any signs of focal neurology. Examination of the cardiovascular, respiratory and abdominal systems was unremarkable. There was no evidence of palpable lymphadenopathy.

What is the appropriate management of this patient's erythema migrans?

A. Serological testing for pathogenic Borrelia species
B. Treat with doxycycline for 14 days
C. No further investigation or treatment required due to absence of history of tick bite
D. Review patient in two weeks and arrange serological tests if onset of symptoms or signs ofLyme borreliosis
E. Review patient in two weeks and treat with doxycycline if onset of symptoms or signs of Lyme borreliosis

B. Treat with doxycycline for 14 days

First line treatment for early Lyme disease is a 14-21 day course of oral doxycycline

Lyme borreliosis is the commonest tick-borne infection in the northern hemisphere. It is relatively rare in the UK, with risk being greatest in rural forested areas such as the Scottish highlands, New Forest and Lake District. The illness is caused by pathogenic *Borrelia* species, members of the spirochete phylum.

Clinical features of Lyme borreliosis include erythema migrans in 90% of symptomatic individuals, which tends to occur to 2-40 days following exposure. Erythema migrans has the classical appearance described in the question but may be homogeneous in the early phase and can be confused for cellulitis or a simple insect bite. Rarely, a non-specific febrile illness without rash can occur. Neurological involvement consisting of meningitis or cranial nerve palsy can occur in early infection.

Erythema migrans is a clinical diagnosis and does not require serological confirmation. Therefore, the patient should be treated immediately with a 2-3 week course of oral doxycycline or amoxicillin, even in the absence of a history of tick attachment. Early treatment of erythema migrans is associated with a good clinical outcome and it would be inappropriate to delay treatment until the onset of other symptoms.

Serological testing should be reserved for individuals in which there is diagnostic uncertainty, neurological symptoms or those who are immunocompromised. Individuals presenting following a tick bite should not be treated or undergo investigation in the absence of erythema migrans or other symptoms of Lyme borreliosis.

Question 13 of 277

An 80-year-old man presents to the Emergency Department with a 3-day history of headache, photophobia and vomiting. He is pyrexial and tachycardic on arrival. He has a past medical history of hypertension, type 2 diabetes and ischaemic heart disease. He was born in the UK and has never lived outside the UK. On examination, he has neck stiffness and photophobia and is mildly confused.

A lumbar puncture is carried out immediately after the administration of antibiotics and shows results as below. Blood glucose at the same time is 7.8 mmol/L.

Red blood cells	5	(0)
White cells	150	(<3)
Cell count differential	90% lymphocytes	(N/A)
Protein	1.5 g/L	(0.18 - 0.45)
Glucose	2.6 mmol/L	(Relative to blood glucose)

What is the most likely causative organism?

A. Haemophilus influenzae
B. Listeria monocytogenes
C. Mycobacterium tuberculosis
D. Neisseria meningitidis
E. Herpes simplex virus

B. Listeria monocytogenes

Listeria may be suggested by CSF showing a lymphocytic pleocytosis, raised protein and reduced glucose

This patient clearly has clinical meningitis, as evidenced by his headache, neck stiffness, photophobia and fever. A degree of confusion suggests that there may also be some encephalitis. The correct answer is ***Listeria monocytogenes***. This diagnosis is suggested by the CSF results showing predominantly lymphocytes, with reduced glucose. Listeria meningitis tends to occur in neonates, pregnant women, the elderly and the immunocompromised. It most commonly presents

with sepsis and meningism and is more likely than other bacterial causes of meningitis to also cause encephalitis or focal neurological deficits.

Haemophilus influenzae is incorrect. This organism can cause meningitis but it mainly does so in infants and children. The CSF would be expected to contain predominantly neutrophils rather than lymphocytes.

Herpes simplex virus is incorrect. HSV might explain why the patient is encephalitic as well as meningitic and you would expect to see a lymphocytosis in the CSF, but the CSF glucose would be normal not low.

Mycobacterium tuberculosis is incorrect. Tuberculous (TB) meningitis could give a similar CSF picture as in this patient, but is less likely given that the patient has no epidemiological risk factors for TB acquisition. TB meningitis also tends to present less acutely.

Neisseria meningitidis is incorrect. In this case, you would expect to see a predominance of neutrophils in the CSF. Lymphocytes can sometimes also be present if there has been a delay between starting antibiotics and performing the lumbar puncture (partially treated meningitis) but this is not the case here where we are told that the lumbar puncture has been done immediately after giving antibiotics.

Question 14 of 277

A 23-year-old man presents to the emergency department with a fever and shortness of breath. He has a past medical history of type 1 diabetes and has had a recent hospital admission with diabetic ketoacidosis. He now has an insulin pump. He does not smoke or drink alcohol.

Observations:

- Heart rate 120 beats per minute \
- Blood pressure 92/48 mmHg
- Respiratory rate 22/minute
- Oxygen saturations 94% on room air
- Temperature 38.4C

On examination, he appears diaphoretic and unwell. Chest auscultation reveals coarse crackles at the right base.

Blood tests:

Hb	138 g/L	Male: (135-180)Female: (115 - 160)
Platelets	189 * 10 /L 9	(150 - 400)
WBC	14.7 * 10 /L 9	(4.0 - 11.0)
Na +	138 mmol/L	(135 - 145)
K +	4.2 mmol/L	(3.5 - 5.0)
Urea	8.2 mmol/L	(2.0 - 7.0)
Creatinine	99 µmol/L	(55 - 120)
CRP	221 mg/L	(< 5)

A chest -ray demonstrates consolidation in the right lower zone.

He is commenced on empirical broad-spectrum antibiotics to treat sepsis secondary to pneumonia.

At 24 hours, a blood culture grows *Klebsiella pneumoniae* , which is identified as producing carbapenemase.

What is the most appropriate antibiotic choice?

A. Ceftriaxone
B. Co-amoxiclav
C. Meropenem
D. Piperacillin/tazobactam
E. Tigecycline

E. Tigecycline

Carbapenemase producing enterobacteriaceae (CPE) are multi-drug resistant coliforms. They can be treated with polymyxins (e.g. colistin), tigecycline, fosfomycin or aminoglycosides (e.g. gentamicin)

Tigecycline is the correct answer. The patient has developed pneumonia and sepsis secondary to infection with *Klebsiella pneumoniae* , which has been identified as a carbapenemase-producing organism. This is a class of bacteria known as carbapenemase-producing enterobacteriaceae. These are gram-negative bacteria that are resistant to carbapenem and extended-spectrum cephalosporin antibiotics. Effective antibiotics against this class of bacteria include polymyxins (e.g. colistin), tigecycline, fosfomycin or aminoglycosides (e.g. gentamicin). Risk factors for CPE infection include immunosuppression (e.g. diabetes) and previous hospital admission.

Meropenem is incorrect. This bacteria is carbapenem-resistant. Meropenem is a carbapenem.

Ceftriaxone is incorrect. CPE are resistant to the cephalosporin class of medication. This is effective against a wide variety of gram-negative and gram-positive aerobes and anaerobes and would be a reasonable choice for the treatment of hospital-acquired pneumonia on an empirical basis. However, it is not effective against CPE.

Piperacillin/tazobactam is incorrect. This class of antibiotic is effective against a broad spectrum of gram-positive and negative bacteria including *Pseudomonas* but is not effective against CPE.

Co-amoxiclav is incorrect. This has a wide spectrum of activity against various gram-negative and gram-positive aerobes but is not effective against CPE.

Question 15 of 277

A 31-year-old gentleman presents with fever, headache, abdominal pain and a rash on the chest 3 weeks after visiting South America.

On examination the temperature is 38.2ºC. There is a rash on the chest consisting of rose-coloured blanching papules. The respiratory rate is 20 breaths/min and the heart rate is 58 beats per minute. The chest is clear to auscultation. The abdomen is diffusely tender and there is mild splenomegaly.

Initial blood results are as follows:

Hb	128 g/l
Platelets	184 * 10 /l9
WBC	3.9 * 10 /l9
Na +	131 mmol/l
K +	3.3 mmol/l
Urea	7.2 mmol/l
Creatinine	141 µmol/l
Bilirubin	46 µmol/l
ALP	147 u/l

ALT	96 u/l
Albumin	38 g/l
CRP	52 mg/l

What is the most appropriate initial antimicrobial therapy?

A. Ampicillin
B. Chloramphenicol
C. Trimethoprim-sulfamethoxazole
D. Cefotaxime
E. Streptomycin

D. Cefotaxime

This is a fairly classical presentation of typhoid fever, also known as enteric fever. This is a potentially fatal multisystemic illness caused primarily by *Salmonella enterica*.

The rash here refers to rose spots which occur in up to 30% of people infected with this organism. Characteristically, rose spots are seen in untreated typhoid fever. They usually occur between the second and fourth week of the illness. They characteristically present as groups of 5-15 pink blanching papules distributed between the level of the nipples and umbilicus.

The treatment of choice is cefotaxime or ceftriaxone.

Ciprofloxacin may be used as an alternative in sensitive organisms.

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A 48-year-old farmer presents with a four-day history of headache, pyrexia and vomiting. He has recently been undergoing chemotherapy for chemotherapy for mantle cell lymphoma and successfully completed his fourth cycle three days ago. During his treatment, he has tried to maintain an active lifestyle and has continued to work on his dairy farm. He has no other past medical history, he does not smoke or drink. On examination, the patient is drowsy and pyrexic at 38.6 degrees. You note no rashes on his skin. His displays neck stiffness and photophobia. You are unable to formally perform a neurological examination but you note no obvious facial asymmetry and the patient is moving all four limbs. Both plantars are downgoing.

Blood tests are as follows:

Hb	98 g/l
Platelets	78 * 10 /l9
WBC	0.9 * 10 /l9
Neutrophils	0.3 * 10 /l9
Na +	146 mmol/l
K +	4.3 mmol/l
Urea	8 mmol/l
Creatinine	99 µmol/l
CRP	170 mg/l

A lumbar puncture is performed and the cerebrospinal fluid examination is as follows:

WCC	200 x 10 /litre (70% neutrophil 25% lymphocytes)6
RBC	4 x 10 /litre6
Glucose	1.7 mmol/l (normal 3.3-4.4 mmol/l)
Microscopy	No organisms on gram stain
Appearance	cloudy

The patient is immediately commenced on intravenous ceftriaxone for suspected bacterial meningitis. At 48 hours after initial admission, blood and cerebrospinal fluid cultures are still awaited. The patient has demonstrated no change in clinical state.

What is an appropriate additional therapy?

A. Hourly neurological monitoring
B. Intravenous acyclovir
C. Intravenous ampicillin
D. Intravenous rifampicin
E. Intravenous amphotericin B

C. Intravenous ampicillin

A diagnosis of meningitis is clear. However, the context is that of a patient who is immunocompromised with recent chemotherapy, presenting with a relatively subacute cause of neutropenic sepsis. The additional information that he continues to work on a dairy with possible exposure to unpasteurised milk increases the chance of listeria exposure, for which intravenous ampicillin is the most appropriate treatment. The patient has demonstrated no improvement in 48 hours; antibiotic escalation would be appropriate. It should be noted that on CSF examination, the absence of an organism on Gram staining does not equate to the absence of an organism! Only one-third of listeria meningitis patients are likely to grow an organism on Gram stain. In addition, listeria meningitis is a classic example of bacterial meningitis when a significant proportion of lymphocytes may be in CSF (>25%), instead of predominant neutrophilia. These include TB, rickettsial and cryptococcal meningitis. With no mention of previous or active TB exposure and no pulmonary symptoms on a relatively short history, TB meningitis is only a low-risk possibility. Intravenous amphotericin B would be appropriate for fungal meningitis only.

Question 17 of 277

Jenna is an 18-year-old woman who was initially admitted with a fever and disseminated rash. She had not been previously vaccinated and was in contact with her 2-year-old cousin who had developed a fever and disseminated blisters and vesicles containing clear fluid. She began to have a fever and flu-like symptoms 2 weeks after seeing her cousin. Similar clear-fluid filled vesicles and blisters developed 3 days later and she was admitted for further observation.

After 3 days, she noticed that while most of her skin lesions are healing, one of the lesions on the thigh appears to be red and becoming hot to touch. An area of skin approximately 3x3cm was erythematous. The skin was marked and she was commenced on IV flucloxacillin. Over the coming 12 hours, the erythema around this lesion continued to spread. The pain around her leg increased in intensity, requiring morphine to take the edge off the pain. A blueish discolouration begins to develop around the rash.

Given the likely complication that has developed, what is the likely organism that has caused the complication?

A. Enterococcus faecalis
B. Clostridium perfringens
C. Staphylococcus aureus
D. Streptococcus bovis
E. β- haemolytic Group A Streptococcus

E. β- haemolytic Group A Streptococcus

Chickenpox is a risk factor for invasive group A streptococcal soft tissue infections including necrotizing fasciitis

The development of fevers along with blisters and vesicles raises the suspicion that this is caused by chickenpox. Besides, this is more likely as she has been exposed to a child who has had similar symptoms. Chickenpox tends to be milder in children but can cause significant morbidity in adults.

The development of a rapidly evolving rash along with significant pains out of proportion to the rash seen should always raise alarm bells for necrotising fasciitis. Blueish discolouration of the skin is also suggestive of this. A surgical review should be sought immediately.

Invasive group A *Streptococcus*, a β-haemolytic *Streptococcus*, has been implicated as the cause for necrotizing fasciitis in patients with chickenpox. Extremely broad-spectrum antibiotics are initially used with the choices tailored to bacterial sensitivities when known.

Staphylococcus aureus can also result in necrotising fasciitis but is more commonly associated with patients who have other underlying medical conditions like diabetes.

Enterococcus faecalis does not tend to cause skin infections and are often associated with infections like endocarditis.

Streptococcus bovis is a gamma-haemolytic *Streptococcus* is most often associated with colorectal cancer associated endocarditis. It is not associated with skin infections.

Clostridium perfringens can cause necrotising fasciitis and presents as gas gangrene. This will present with crepitus under the skin which is not seen here.

Question 18 of 277

A 44-year-old vet presents with a fever to his GP. He has had the fever for 4 days and it seems to come and go. He also mentions that he has felt generally lethargic for the past week, with occasional lower back pain and aching limbs. On examination, he is tender in his sacral region and has an enlarged liver and spleen. Blood tests reveal the following:

Hb	136 g/L	Male: (135-180)Female: (115 - 160)
Platelets	160 * 10 /L 9	(150 - 400)
WBC	3.8 * 10 /L 9	(4.0 - 11.0)
Bilirubin	7 µmol/L	(3 - 17)
ALP	50 u/L	(30 - 100)
ALT	23 u/L	(3 - 40)
Î³GT	10 u/L	(8 - 60)
Albumin	43 g/L	(35 - 50)

Which of the following pathogens is the most likely cause of this man's presentation?

A. Cryptosporidium
B. Brucella
C. Bartonella henselae
D. Echinococcus granulosis
E. Leptospira

B. Brucella

Unwell vet with fever, malaise, arthralgia and lower back pain ? Brucellosis

This man presents with features suggestive of brucellosis, including fluctuating fevers, malaise, arthralgia, sacroiliitis, hepatosplenomegaly and mild leukopenia. Brucellosis is a zoonosis: an infectious disease that jumps from a non-human animal to humans. It is caused by the gramnegative, rod-shaped *Brucella* species of bacteria. It is more common in the Middle East and in those that work with animals, such as this man. It is spread by aerosol and considered a bioterrorism agent. Rose Bengal plate test is used in suspected Brucellosis. One can remember this as B-Rose in B-Ruce-losis.

Cryptosporidiosis is a protozoal disease that typically affects the immunocompromised and causes diarrhoea.

Bartonella henselae is a bacteria that causes cat-scratch disease, another disease that is more common in vets. Although it may present with flu-like symptoms, it does not typically cause hepatosplenomegaly. There is typically a history of a bite or scratch from a cat, with a lesion developing over the area and painful lymphadenopathy. As a side note *Bartonella bacilliformis* can cause Oroya fever, which may present with hepatosplenomegaly and fever, but typically there is lymphadenopathy also, with marked haemolysis.

Echinococcus granulosis is a tapeworm that causes hydatid disease, another disease that is more common in vets. However, hydatid disease causes cysts that typically affect the liver or lungs, and is more likely to present with abdominal pain, jaundice, weight loss, and if affecting the lungs then a cough and shortness of breath.

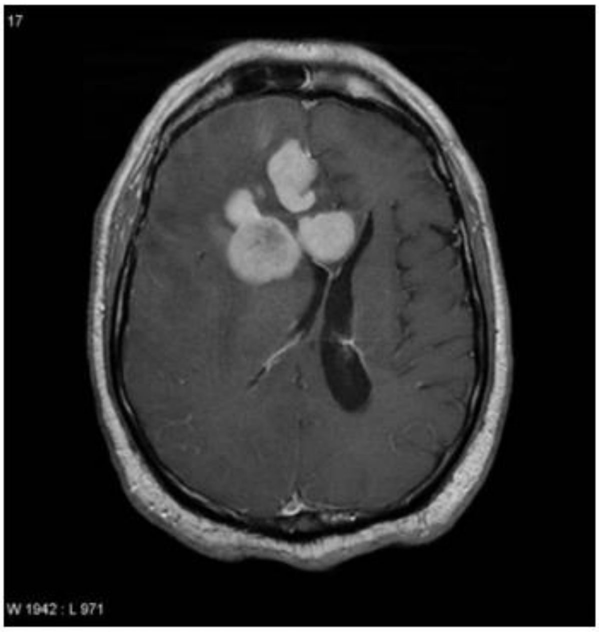
Leptospirosis is a possible diagnosis here, as it often presents in vets, and can present with flu-like illness and hepatosplenomegaly. The sacral tenderness, leukopenia and normal LFTs make this less likely. Leptospirosis will typically cause leukocytosis, anaemia, thrombocytopenia, even in the absence of DIC. Even when clinical manifestations are mild, hyperbilirubinemia is often present and mild elevations of serum transaminases are frequently seen. In exams, leptospirosis will normally present with bilateral conjunctival suffusion, which is conjunctivitis without exudate. However, in reality, this occurs in <60% of patients.

Question 19 of 277

A 45-year-old man is reviewed in the HIV clinic. He was diagnosed with HIV 25 years ago and has enjoyed good health on highly active antiretroviral treatment until around 4 years ago when he had an episode of *Pneumocystis jirovecii* pneumonia.

Today he is somewhat confused. His partner reports that he has been complaining of headaches for several weeks and has also been uncharacteristically aggressive.

His latest CD4 count is 29 cells/µl. An MRI (T1 C+) is requested:



What is the most appropriate treatment?

A. Sulfadiazine + pyrimethamine
B. Sulfadiazine + pyrimethamine + steroids
C. Steroids + methotrexate
D. Rifampicin + isoniazid + pyrazinamide + ethambutol + steroids

E. Amphotericin B

C. Steroids + methotrexate

HIV, neuro symptoms, single brain lesions with homogenous enhancement - CNS lymphoma

The MRI scan demonstrates a large multilobulated mass in the right frontal lobe. It homogeneously enhances and extends to involve the caudate and the periventricular area. There is also a significant mass effect. These findings in addition to the history are highly suggestive of primary CNS lymphoma. The most appropriate initial treatment is steroids (which should reduce tumour size and hence mass effect) and methotrexate. Other future options include whole brain irradiation and surgery.

You would not expect the enhancement to be homogenous with toxoplasmosis, it is usually in a ring pattern.

Question 20 of 277

A 34-year-old man presents to the emergency department with two weeks of fevers, malaise, myalgia, arthralgia and low back pain. He has no significant past medical history. He does not take any medications. He works as a veterinarian. There is no recent travel.

Observations:

- Heart rate 94 beats per minute
- Blood pressure 121/88 mmHg
- Respiratory rate 19/minute
- Oxygen saturation 96% on room air
- Temperature 38.2ºC

On examination, there is focal tenderness at the left sacroiliac joint. Cardiovascular, respiratory and abdominal examinations are unremarkable. He is diaphoretic and an associated foul smell is noted. There is no joint swelling.

Urinalysis is unremarkable.

Plain radiography of the chest is normal. Blood tests:

Hb	138 g/L	Male: (135-180)Female: (115 - 160)
Platelets	189 * 10 /L 9	(150 - 400)
WBC	3.4 * 10 /L 9	(4.0 - 11.0)
Na +	138 mmol/L	(135 - 145)
K +	4.2 mmol/L	(3.5 - 5.0)
Urea	4.2 mmol/L	(2.0 - 7.0)
Creatinine	89 µmol/L	(55 - 120)
CRP	45 mg/L	(< 5)
Bilirubin	12 µmol/L	(3 - 17)
ALP	89 u/L	(30 - 100)
ALT	35 u/L	(3 - 40)
Î³GT	44 u/L	(8 - 60)
Albumin	36 g/L	(35 - 50)

What is the likely diagnosis?

A. Brucellosis
B. HIV seroconversion
C. Leptospirosis
D. Q fever
E. Tuberculosis

A. Brucellosis

Unwell vet with fever, malaise, arthralgia and lower back pain ? Brucellosis

Brucellosis is the correct answer. The patient presents with fevers, malaise, arthralgia, myalgia and low back pain. The location of the back pain suggests sacroiliitis. Profuse sweating associated with a foul smell is typical of this condition. It is often said to be 'hay-like'. His work as a veterinarian is a relevant occupational exposure.

Leptospirosis is incorrect. This can cause a febrile illness with arthralgia and myalgia. However, it is typically associated with subconjunctival haemorrhages and headache and pulmonary haemorrhage and jaundice in severe cases. Foul-smelling sweat is not a typical feature.

Q fever is incorrect. While he works as a vet and this is a relevant occupational exposure, this condition is typically characterised by an atypical pneumonia, which is absent in this case. Sacroiliac involvement is not a feature.

Tuberculosis is incorrect. This can cause arthralgia, fevers and in rare cases can involve the sacroiliac joint. However, the occupational exposure and foul-smelling sweat making brucellosis the more likely diagnosis, especially in the absence of a relevant travel history.

HIV seroconversion is incorrect. While this can cause a febrile illness, myalgia, arthralgia and a low white blood cell count. However, the relevant occupational exposure, sacroiliac joint involvement and foul-smelling sweat make a diagnosis of brucellosis more likely.

Question 21 of 277

A 32-year-old man has been brought to the hospital with severe shortness of breath.

Initial observations with the paramedics were taken. These included a respiratory rate of 32/min, oxygen saturations of 72% on room air, a pulse of 120/min, a blood pressure of 123/72mmHg, and a temperature of 38.3ºC.

On examination in the emergency department, he is sat on the bed leaning forwards. There is evidence of drooling and a vomit bowl full of saliva is next to him. There is audible stridor from the edge of the bed. Oxygen has now been applied and saturations are 90% on 15L.

A neck x-ray was performed due to concerns for a foreign body and has shown a 'thumb sign'.

What is the most appropriate next step in management?

A. Continuous positive airway pressure (CPAP)
B. Endotracheal intubation
C. High flow oxygen
D. Intramuscular adrenaline
E. Nebulised adrenaline

B. Endotracheal intubation

Endotracheal intubation may be necessary to protect the airway in acute epiglottitis

The above scenario describes a severe case of acute epiglottitis. Patients approaching extremis tend to lean forwards to aid their breathing and may have pooling of saliva due to an inability to swallow. The 'thumb sign' on the neck x-ray is indicative of an oedematous epiglottitis. Given the significant desaturation in oxygen along with audible stridor, the most appropriate intervention would be endotracheal intubation.

Given the degree of obstruction in this case, urgent anaesthetic input should be sourced due to the high likelihood that this patient will require intubation. This should not be delayed to trial CPAP. It is also possible that CPAP will lead to the displacement of the epiglottis and worsen the obstruction.

High flow oxygen can be used in patients that have low oxygen saturation who are otherwise stable. This would not be appropriate in the above scenario given the stridor and a need to urgently secure the airway.

Intramuscular adrenaline would be beneficial if this was stridor associated with anaphylaxis, however, there is no evidence of this from the scenario.

Nebulised adrenaline can be used in croup but is not a part of management in epiglottitis.

Question 22 of 277

A 25-year-old woman is started on antiretroviral therapy for HIV. She attends the GP 1 week later complaining of dizziness and nightmares. She is unable to work due to feeling muddled and disconnected.

Which drug is responsible?

A. Abacavir
B. Efaverinz
C. Lamivudine
D. Tenofovir
E. Zidovudine

B. Efaverinz

Efaverinz is a non-nucleoside reverse transcriptase inhibitor (NNRTI) and is frequently known to cause disturbing dreams and other cognitive disturbances in 50% of patients in the first month of treatment.

The most concerning side effect of Abacavir is a hypersensitivity reaction that can lead to fever, malaise, nausea and skin rash which can be very severe. It is strongly associated with a specific allele at the human leukocyte antigen B locus, HLA-B*57:01.

Lamivudine and Zidovudine are nucleoside analogue reverse transcriptase inhibitors (NRTIs). The NRTIs were the first class of antiretroviral drugs developed. As with all drugs in their class, they can cause a potentially fatal lactic acidosis & severe hepatomegaly with steatosis when used alone or in combination with other antiretrovirals.

Tenofovir is a nucleotide analogue reverse transcriptase inhibitors (NtRTI), it is a prodrug, the commonest side effects are gastrointestinal.

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A 57-year-old male with chronic hepatitis B infection presents to the Emergency Department with increasing confusion. His wife states that his specialist has not started him on antivirals yet. He was diagnosed six months ago. His past medical history includes tuberculosis that has been successfully treated 10 years ago.

His wife stated also that he slept more than usual in the last week and he was more lethargic. On examination the patient was confused, disoriented with a yellowish discoloration of his eyes. There is moderate peripheral oedema and moderate ascites with no signs of hepatomegaly.

Blood investigations were as follows:

Hb	102 g/l
Platelets	250 * 10 /l9
WBC	9.5 * 10 /l9
Bilirubin	42 µmol/l
ALP	135 u/l
ALT	370 u/l
γGT	70 u/l
Albumin	32 g/l
α-fetoprotein	15 kU/l

What is the most likely cause of this presentation?

A. Portal vein thrombosis
B. Budd-Chiari syndrome
C. Hepatitis D infection
D. Hepatocellular carcinoma
E. Normal progression of the disease

C. Hepatitis D infection

This patient has developed acute liver decompensation. Hepatitis D infection is an uncommon but important cause of this presentation. It typically occurs in a patient with a history of stable chronic hepatitis B infection that suddenly acutely deteriorates.

Budd-Chiari syndrome (BCS) and portal vein thrombosis are not the correct options because it is unlikely to have any of them in the absence of hepatomegaly. In addition, the high level of the transaminase enzyme suggests viral infection rather than thrombus formation. Note that the transaminase enzyme should be only mildly elevated in BCS. Fever and nausea are also important symptoms that are usually present in BCS.

Hepatocellular carcinoma usually presents with very high levels of alpha-fetoprotein.

Question 24 of 277

A 32-year-old woman attends the infectious diseases clinic for an urgent review. The patient had presented to the emergency department the previous weekend complaining of various constitutional symptoms. A set of blood cultures had been taken as the patient was febrile at presentation. Assessment in the emergency department had not identified a clear cause for the patient's illness and she had been discharged home with advice to attend her GP surgery if her symptoms did not resolve in the coming days. The patient had been recalled to hospital for review after the blood cultures taken during her presentation yielded a positive result. Selected investigation results from the patient's emergency department attendance are given below.

At her review appointment, the patient recounted her recent symptoms. She described a 3-week history of intermittent fever, malaise and reduced appetite. On direct questioning, she did recall experiencing excessive overnight sweating on a few occasions but had not experienced joint pains. The patient denied other symptoms, including those associated with the respiratory, urinary tract and neurological systems.

The patient was otherwise healthy, with no significant past medical history. She took no regular medications and had no known medication allergies. The patient was a chartered accountant and lived with her husband and 5-year-old daughter. The patient had returned from a holiday in Turkey 4 weeks previously. During her holiday she had visited relatives in a rural village and recalled consuming local dairy products that she suspected had not been pasteurised.

On general examination, the patient appeared to be tired and pale. Examination of the patient's abdomen revealed palpable hepatomegaly and splenomegaly. Examinations of the respiratory, cardiovascular, neurological and musculoskeletal systems were unremarkable. Selected investigation results from the patient's emergency department attendance are given below.

Haemoglobin	117 g / dL
White cell count	3.9 x 10>3 / microlitre
Neutrophils	2.1 x 10>3 / microlitre (reference 1.8-7.8)
eGFR	> 90 ml / min / 1.73 m2
Alkaline phosphatase	134 U / L (reference 35-100)
ALT	57 U / L (reference 3-36)
Bilirubin	24 micromol / L (reference < 26)
C-reactive protein	68 mg / L (reference < 7)
Urine dipstick	No abnormality detected
Blood cultures	Small gram-positive coccobacilli; consistent with *Brucella* species
Chest x-ray	Clear lung fields; no evidence of consolidation or effusion

The likely diagnosis of Brucellosis was discussed with the patient. She expressed a very strong preference to receive treatment on an outpatient basis so that she would not be separated from her family.

What is the appropriate treatment regime?

A. Doxycycline monotherapy for 2 weeks
B. Proguanil with atovaquone for 3 days
C. Clarithromycin and amoxicillin for 1 week
D. Isoniazid, rifampicin, pyrazinamide and ethambutol for 8 weeks
E. Doxycycline and rifampicin for 6 weeks

E. Doxycycline and rifampicin for 6 weeks

Brucellosis is the most common bacterial disease contracted from contact with animals, most commonly sheep and goats. Infection can be acquired from consumption of unprocessed animal products in endemic countries of the Mediterranean and middle East such as Syria, Iraq, Turkey, Greece and Tunisia. Brucellosis can also be acquired by occupational exposure of laboratory workers or individuals with close contact with animals.

Brucellosis presents with non-specific symptoms and signs and so cases can go undiagnosed for several months. In addition to the typical symptoms and signs experienced by the patient in the question, brucellosis can also cause migratory arthritis, lymphadenopathy and malodorous perspiration. Rare complications include pneumonia, pulmonary effusion, peripheral neuropathy, meningoencephalitis and endocarditis. Positive blood cultures provide the most definitive diagnosis of brucellosis, although various serological tests are also available. Microbiological diagnosis is also possible from the culture of samples obtained from infected organs (for example, synovial fluid or cerebrospinal fluid). Brucellosis also causes non-specific abnormalities of routine blood tests as in the case of the patient in this question.

Oral treatment of brucellosis is possible with a 6-week course of doxycycline and rifampicin. An alternative treatment regime combines 6 weeks of oral doxycycline with 3 weeks of either intramuscular streptomycin or intravenous gentamicin. Treatment involving parenteral antibiotics has a lower failure rate but is less convenient for the patient.

The other possible answers are antimicrobial treatment regimes for *Helicobacter pylori*, tuberculosis, Lyme disease and malaria.

Question 25 of 277

A 35-year-old man is reviewed in respiratory clinic with symptoms of a chronic cough with occasional haemoptysis, night sweats and significant unintentional weight loss. The patient is originally from the Russian Federation and on direct questioning through an interpreter discloses that he had spent five years in a Moscow prison for drugs offences. Aside from a period of alcohol abuse in his early twenties, the patient denies any other medical history and takes no medications. He currently is living in shared accommodation and works on a building site, although he is struggling to continue to work due to his current illness.

General inspection showed the patient to be cachexic with tobacco stained fingernails. Auscultation of the chest revealed some reduced air entry in the upper zones bilaterally. There was palpable lymphadenopathy in the anterior cervical chain.

Please see results of investigations below.

Chest x-ray: patchy shadowing in both upper lobes with evidence of cavity formation on the right side; no pneumothorax; normal mediastinal shadow.

Sputum microscopy: acid fast bacilli

Phenotypic indirect drug susceptibility testing: rifampicin (resistant); ofloxacin (sensitive); moxifloxacin (sensitive); isoniazid (resistant); amikacin (sensitive); capreomycin (sensitive).

What is the most appropriate treatment regime for this patient?

A. Combination therapy with 5 drugs for 24-36 months
B. Combination therapy with 5 drugs for 18-24 months
C. Combination therapy with 4 drugs for 24-36 months
D. Combination therapy with 4 drugs for 12 months
E. Combination therapy with 4 drugs for 18-24 months

B. Combination therapy with 5 drugs for 18-24 months

The patient presents with a typical history for active pulmonary tuberculosis. His history of incarceration in a prison in the Russian Federation makes him at high risk of having contracted a multi-drug resistant strain of TB. Microbiology results confirm resistance to rifampicin and isoniazid consistent with multi-drug resistance, however without resistance to any fluoroquinolone or second line injectable agents that would suggest extensively drug resistant TB.

Recommended treatment for multi-drug resistant TB requires 18-24 months of at least 5 drugs. Depending on the microbiological testing services available, the regime may be individualised to an individual patients infection or based on a standardised regime based on local resistance patterns.

Question 26 of 277

A 72-year-old man presents to the tuberculosis clinic.

He has a 6-month history of lower back pain that has been gradually getting worse. He has tried taking analgesia, but this is now no longer helping the pain and he is becoming less able to mobilise.

He lives with his wife on their farm. He has been fully independent until his recent deterioration in mobility.

Past medical history includes hypertension, diet-controlled type 2 diabetes mellitus and benign prostatic hypertrophy.

He reports that he had tuberculosis when he was 24, but this was treated. He can't remember what he was treated with.

An MRI spine shows a lumbar 4/5 discitis and a biopsy of this tissue is arranged. Acid-fast bacilli are grown on culture of the biopsy.

What treatment regime should be started?

A. 2 months of isoniazid, rifampicin, pyrazinamide and ethambutol with a further 4 months of isoniazid and rifampicin
B. 6 months of isoniazid, rifampicin, pyrazinamide and ethambutol with a further 6 months ofisoniazid and rifampicin
C. 6 months of isoniazid, rifampicin, pyrazinamide and ethambutol
D. 2 months of isoniazid, rifampicin, pyrazinamide and ethambutol with a further 7 months ofisoniazid and rifampicin
E. 3 months of isoniazid, rifampicin, pyrazinamide and ethambutol with a further 6 months ofisoniazid and rifampicin

A. 2 months of isoniazid, rifampicin, pyrazinamide and ethambutol with a further 4 months of isoniazid and rifampicin

Treatment for bone and joint tuberculosis is recommended to continue for 2 months with the initial phase consisting of quadruple therapy and the remaining 4 months of dual therapy.

It is recommended not to extend treatment for residual complications such as collapsed discs or bending of the spine, although there is some debate about this.

Immune reconstitution disease occurs typically 3-6 weeks after starting treatment often presents with enlarging lymph nodes

Question 27 of 277

A 45 year old man is diagnosed with HIV following attendance at Genitourinary clinic. As part of his routine HIV work up his HLA B*5701 result is reported as positive.

Which drug will not be included in his ART regimen as a result of this information?

A. Tenofovir

B. Emtricitabine

C. Lamivudine

D. Stavudine

E. Abacavir

E. Abacavir

Abacavir can cause a severe and sometimes fatal hypersensitivity reaction. It is a cell mediated delayed hypersensitivity reaction which occurs around 6 weeks after starting treatment. Symptoms are of fever, rash, malaise and gastrointestinal symptoms.

Patients without HLA B*5701 are highly unlikely to develop hypersensitivity. Those with the HLA B*5701 allele have a 50% risk and therefore in these individuals abacavir should be avoided.

It is therefore routine to test HLA B*5701 status on all newly diagnosed patients with HIV.

Question 28 of 277

A 54 year old Caucasian man with HIV, is stable on Tenofovir, Emtricitabine and Kaltera (Lopinavir/Ritonavir). He has a 60 pack year smoking history and has been told his blood pressure is high previously but had never previously been to see his GP about it to get it treated. His GP is unaware of his HIV diagnosis and his main point of healthcare contact is with the Genito-urinary medicine services.

However over the last two years he has been troubled by gradually worsening cough, and shortness of breath. He has not lost weight. This has spurred him to start seeing his GP. He says that his GP initially gave him a blue inhaler and did some tests and told him he had COPD. Six weeks ago his GP gave him a Seretide 500 inhaler (one puff twice a day) and started him on Rampril (2.5mg daily) for his high blood pressure. The purple inhaler had provided some relief to his breathlessness but four days ago it ran out and he hasnt yet been back to see his GP. He still has two weeks worth of Ramipril tablets.

For the last few days he has been feeling faint and dizzy especially when standing up. His breathlessness and cough are no worse than usual. He has no fever. On examination his blood pressure is 91/76 his pulse is 94. He is afebrile, his respiratory rate is 24 and his oxygen saturations are 92% on room air. He has a mild wheeze on auscultation of his chest. His heart sounds are faint and his JVP is visible 3cm above his sternal angle. There is no pitting odema. There is nothing else of note on examination.

An ECG on admission is normal.

Hb	14.0 g/dl
Platelets	199 * 10 /l9
WBC	6.2 * 10 /l9
Na +	130 mmol/l
K +	5.9 mmol/l
Urea	5.6 mmol/l
Creatinine	85 µmol/l
CRP	7 mg/l

What is the diagnosis?

A. Adrenal tuberculosis
B. Pericardial tuberculosis
C. Adrenal insufficiency secondary to inhaled corticosteroid withdrawal
D. Ramipril induced hypotension
E. WaterhouseFriderichsen syndrome

C. Adrenal insufficiency secondary to inhaled corticosteroid withdrawal

A very difficult question. The timing of events is key.

Fluticasone in Seretide is metabolised by P450 and Ritonvir inhibits this allowing very high levels of systemic steroid with consequent adrenal insufficiency. Cessation of Seretide has caused this Addisonian event (low sodium, high potassium is the clue).

Disseminated TB would be unusual with no fever or weight loss but is an important differential, especially in the context of HIV infection. All of his findings on examination are consistent with COPD.

Waterhouse-Friderichsen syndrome is bilateral adrenal haemorrhage in the context of meningococcal septicaemia.

Question 29 of 277

You see Mr Smith, a 35-year-old man-who-has-sex-with-men (MSM) in clinic. He was diagnosed with HIV 5 years ago, commencing combination antiretroviral therapy (cART). Following a number of alterations to his cART due to side effects he responded well to a combination of tenofovir, emtricitabine and ritonavir boosted atazanavir. His plasma viral load (pVL) of HIV RNA has remained undetectable and his adherence has been good.

In clinic today Mr Smith reports 5 weeks of drenching night sweats, a dry cough and some subjective weight loss, going up a belt buckle during this time period. On examination you note that he appears pale and auscultation of the chest elicits crepitations in the left upper zone. Your perform a chest X-ray which demonstrates a cavitating lesion in the left upper lobe. You arrange induced sputum samples which confirm a diagnosis of pulmonary tuberculosis.

You explain your diagnosis to Mr Smith and the need to urgently commence him on antituberculosis chemotherapy. Whilst he is happy to commence treatment, he is adamant that he does not want to risk his viral control and states that he is not willing to consider altering his cART regime at present.

What is the most appropriate management step to treat Mr Smith?

A. Hold his ART
B. Commence rifabutin, isoniazid, ethambutol and pyrazinamide
C. Commence isoniazid monotherapy
D. Switch protease inhibitor to an novel ARV agent
E. Commence rifampicin, isoniazid, pyrazinamide and ethambutol

B. Commence rifabutin, isoniazid, ethambutol and pyrazinamide

The correct answer for this scenario is to commence rifabutin, isoniazid, ethambutol and pyrazinamide.

This scenario tests your knowledge of rifamycins in the treatment of tuberculosis. Rifamycin containing anti-tuberculosis chemotherapy regimes are superior to those which do not contain a rifamycin agent. However, in this scenario the patient is also taking taking a protease inhibitor as part of their antiretroviral therapy.

Rifampicin, the common rifamycin agent in anti-tuberculosis regimes, is a potent inducer of liver enzymes, such as cytochrome P450. Furthermore, it up-regulates the expression of P-glycoprotein in the gastrointestinal tract. This leads to reduced absorption and increased metabolism of certain medications, protease inhibitors being one of these (ritonavir booster atazanavir). Coadministration of a protease inhibitor with rifampicin therefore will often lead to subtherapeutic levels of the protease inhibitor.

In this scenario, the patient is stable on his current antiretroviral treatment and not keen to switch at present. Therefore, the British HIV Association (BHIVA) suggest the substitution of rifampicin for an alternative rifamycin agent (rifabutin or rifapentine), which has less inducing action of cytochrome P450 and therefore allows the patient to continue their current HIV treatment regime. Regimes containing rifabutin (dose 150mg three times a week) have been shown to have equivocal in outcomes of treatment in the few small observational studies performed in patients with HIV/TB co-infection.

Question 30 of 277

A 28-year-old man attends clinic for his 6 monthly HIV check. He was diagnosed with HIV 4 years ago and has been stable on Eviplera (emtricitabine/rilpivirine/tenofovir). He was recently started on a new medication by his GP.

Blood results are as follows:

Hb	134 g/l	Na +	136 mmol/l
Platelets	345 * 10 /l 9	K +	4.2 mmol/l
CD4	580 * 10 /l (normal range 500-1500) 9	Urea	6.5 mmol/l
Viral load	1480 copies/ml* 10 /l 9	Creatinine	65 µmol/l

What new medication was started which would explain these results?

A. Omeprazole
B. Ciclosporin
C. Tacrolimus
D. Celecoxib
E. Diclofenac

A. Omeprazole

PPIs are contraindicated in patients on Eviplera (emtricitabine/rilpivirine/tenofovir). They reduce the absorption of rilpivirine which can cause viral blips, with subsequent virological failure and resistance

Concurrent use of tenofovir with ciclosporin, tacrolimus, celecoxib or diclofenac, can increase the risk of nephrotoxicity.

Proton pump inhibitors are absolutely contraindicated in patients on Eviplera (emtricitabine/rilpivirine/tenofovir). They reduce the absorption of rilpivirine which can cause viral blips, with subsequent viro00 logical failure and resistance.

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A 23-year-old medical student attends the emergency department after accidentally cutting herself in the kitchen, with significant blood loss. In addition to first aid for the wound, routine blood tests are checked. Results are as follows:

Hb	145 g/L	Male: (135-180)Female: (115 - 160)
Platelets	235 * 10 /L 9	(150 - 400)
WBC	7.2 * 10 /L 9	(4.0 - 11.0)
Eosinophils	0.91 * 10 /L 9	(0.0-0.4)

Follow-up is organised in the infectious diseases clinic. The patient has recently returned from her elective in Malawi, where she spent 6 weeks with 2 friends. She reports taking all recommended vaccines before travel. Malaria prophylaxis was taken as advised and she completed the course. She denies swimming in any water but did frequently walk barefoot around the banks of the lake. She recalls one episode where her foot became itchy and developed a serpiginous rash, advancing up the foot over several hours before resolving. She did not seek medical help.

What treatment is recommended first-line for this condition?

A. Artemisinin combination therapy
B. Ivermectin
C. Melarsoprol
D. Nifurtimox/eflornithine combination therapy
E. Praziquantel

B. Ivermectin

Ivermectin is the drug of choice for treatment of strongyloidiasis

The correct answer is **ivermectin**. This patient is likely to have developed strongyloidiasis, a parasitic infection caused by the nematode Strongyloides stercoralis. This is a widely-distributed helminth but the majority of cases are seen in sub-Saharan Africa and South America. Infection is frequently asymptomatic and the first clue is peripheral eosinophilia. In this case, the episode described in Malawi is consistent with larva currens, a rash that develops when the worm penetrates the skin before entering the bloodstream. From here they spread through the lungs, over the epiglottis, are swallowed, and settle in the small intestine, where they lay their eggs. Respiratory symptoms are rare but cough and wheeze may develop during pulmonary migration. In chronic infection, abdominal pain and diarrhoea may occur. In the context of immunocompromise, Strongyloides hyperinfection syndrome can occur, presenting with bloody colitis and pneumonitis, often with pleural effusions. The treatment of choice in all cases is ivermectin, although albendazole and thiabendazole are alternatives.

Artemisinin combination therapy is incorrect. This is the treatment of choice for most cases of uncomplicated malaria. Malaria does not tend to cause eosinophilia or skin lesions, and she has been compliant with prophylaxis. These factors suggest this is very unlikely to be the diagnosis.

Melarsoprol is incorrect. This is an arsenic-based treatment option for East African sleeping sickness, caused by Trypanosoma brucei rhodesiense. This is a rapidly progressive form of sleeping sickness, initially causing an influenza-like illness with arthralgia and lymphadenopathy but quickly progressing to altered mental status and death. Melarsoprol is associated with significant toxicity and would not be used for strongyloidiasis.

Nifurtimox/eflornithine combination therapy is incorrect. This is a treatment option for West African sleeping sickness, caused by the protozoan parasite Trypanosoma brucei gambiense. This causes a slowly progressive illness characterised by behavioural change, alterations to the sleepwake cycle, and finally coma. There are no symptoms here to suggest this and it is not typically associated with eosinophilia.

Praziquantel is incorrect. This is the treatment of choice for schistosomiasis, a tapeworm infection caused by a variety of species of schistosomes. The organism uses freshwater snails as an intermediate host before being released into the water, where it penetrates human skin and settles in the gastrointestinal or genito-urinary tract depending on the species. There may be an itchy rash at the time of infection, known as cercarial dermatitis, but this would not move up the limb or be described as serpiginous. Schistosomiasis often presents with eosinophilia, but in this case, the patient has been careful to avoid going in the water, making this unlikely to be the cause.

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A 53-year-old lady has been admitted under isolation to a tertiary infectious diseases centre in London. She has recently arrived back to the United Kingdom following 2 years of ecological work in lowland Nepal and has presented with 4 weeks of worsening malaise, fevers, night sweats, pallor and abdominal pain. She had lived during her time in Nepal in a small village where she reported numerous fly bites during the evening hours. She reports taking her malaria prophylaxis consistently.

Examination shows her to look pale, unwell and her skin appears to have an unnatural grey tinge. No rash nor joint swelling was noted. She has a temperature of 37.9ºC, oxygen saturation 97% on air, heart rate 84 beats per minute, respiratory rate 23 breaths per minute and blood pressure 128/75 mmHg. Auscultation of the chest reveals bilateral vesicular breath sounds and her heart sounds are normal with no murmurs. Abdominal examination reveals a soft abdomen with tenderness in the upper quadrants. Her liver edge is palpable 5cm below the right costal margin and an additional mass is palpable in the umbilical area which moves with ventilation.

Investigation results are as follows:

Hb	102 g/L	Female: (115 - 160)
Platelets	$134 * 10^9/L$	(150 - 400)
WBC	$3.6 * 10^9/L$	(4.0 - 11.0)
Na^+	144 mmol/L	(135 - 145)
K^+	3.7 mmol/L	(3.5 - 5.0)
Urea	6.3 mmol/L	(2.0 - 7.0)
Creatinine	105 µmol/L	(55 - 120)
CRP	73 mg/L	(< 5)
Thick blood film	No evidence of malaria parasites	
HIV 1 and 2 surface antigen/antibodies	Negative	
Hepatitis A, B, C, D and E	Negative	
Chest X-ray	No focal consolidation, masses or collapse	
quantiFERON testing	Negative	

Which of the following investigation would be the gold standard to confirm the most likely diagnosis?

A. Direct agglutination test (DAT)
B. Lumbar puncture and CSF analysis
C. Stool sample analysis
D. Blood cultures
E. Splenic aspirate

E. Splenic aspirate

Leishmaniasis is spread through sand-fly vectors from animal reservoirs and remains endemic through areas of Africa, Asia, South America and the Middle East. Visceral leishmaniasis can cause hepatosplenomegaly, spiking fevers, pancytopenia and with certain strains blackening of the skin leading to the alternative name of black fever (kala-azar). The gold standard for diagnosis would be the demonstration of amastigotes on splenic or bone marrow aspirates.

Serological testing, such as DAT, is useful in the diagnosis of leishmaniasis but it is not the gold standard due to lack of evidence demonstrating consistently higher sensitivities and specificities along with a regional variation of the parasites complicating this. They are however highly useful in endemic areas where it is a cheaper, less technically challenging and occasionally a readily available alternative.

Lumbar puncture and CSF analysis are not useful investigations in the diagnosis of leishmaniasis unless an alternative or additional pathology is suspected.

A stool sample is of no use in the diagnosis of leishmaniasis which is a vector born disease transmitted through sandfly bites.

Blood cultures can be useful in demonstrating blood-born parasites in immunocompetent hosts with sensitivity rates up to 60%. This, however, remains below that of the current gold standard.

Question 33 of 277

A 45-year-old homeless gentleman presents with a two-month history of a cough, occasional haemoptysis and weight loss. His chest X-ray shows right upper lobe consolidation. A diagnosis of tuberculosis is confirmed by microscopy and he is started empirically on rifampicin, isoniazid, pyrazinamide and ethambutol, with pyridoxine supplements. Eight weeks later, culture results show that the Mycobacterium tuberculosis is resistant to isoniazid.

What should his further treatment regime be?

A. Four months of rifampicin, isoniazid, pyrazinamide and ethambutol, with pyridoxine supplements
B. Six months of rifampicin
C. Four months of rifampicin and ethambutol, with pyridoxine supplements
D. Four months of rifampicin and ethambutol
E. Add a quinolone and aminoglycoside

D. Four months of rifampicin and ethambutol

In confirmed isoniazid resistance, ethambutol is continued for 6 months of treatment

In fully sensitive Mycobacterium tuberculosis infection, after two months, the pyrazinamide and ethambutol are stopped and rifampicin and isoniazid (with pyridoxine support) are continued for a further four months. However, in cases of confirmed isoniazid resistance, rifampicin and ethambutol are continued for the further 4 months of treatment, with discontinuation of the isoniazid (and supportive pyridoxine) and pyrazinamide.

This is not multi-drug resistant tuberculosis (does not respond to at least isoniazid and rifampicin), so it is not appropriate to add a quinolone or aminoglycoside.

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A 22 year old male attends the Genitourinary Medicine clinic which you are working in. He was contacted and asked to attend having been in direct, recent sexual contact with gonorrhoea. He is asymptomatic.

What is the best course of action?

A. No action required as he is asymptomatic
B. Treat with ceftriaxone, perform a sexual health screen and call back for a test of cure ifpositive
C. Give ceftriaxone and azithromycin, no need for a screen
D. Perform a screen and call back to treat if positive
E. Give ceftriaxone, perform a screen, and call back to give azithromycin if positive

B. Treat with ceftriaxone, perform a sexual health screen and call back for a test of cure ifpositive

The British Association of Sexual Health and HIV have clear guidelines on the investigation and management of gonorrhoea.

Recent sexual contact with person in whom gonococcal infection has been confirmed is an indication for treatment.

Treatment of uncomplicated gonorrhoea infection consists of ceftriaxone intramuscularly as a single dose. Due to increased resistance to a range of antimicrobials in recent years follow up 'test of cure' is recommended in all confirmed cases.

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A 31 year-old accountant was referred to the general medical clinic with persistent nasal stuffiness and intermittent epistaxis. He had been seen by the ear, nose and throat team who had commenced him on a course of intranasal steroids without benefit. His past medical history was unremarkable. His travel history included a gap year in South America 10 years previously. He was a non-smoker and drank 23 units of alcohol per week.

On examination, his temperature was 36.2ºC, heart rate 68 beats per minute, respiratory rate 16 breaths per minute, blood pressure 126/82 mmHg. There was some superficial ulceration of the nasal mucosa bilaterally. The chest was clear on auscultation and heart sounds were normal.

Investigations:

Haemoglobin	145 g/L
White cell count	7.0 * 10 /l9
Neutrophil count	4.0 * 10 /l9
Lymphocyte count	3.0 * 10 /l9
Eosinophil count	0.4 * 10 /l9
Platelets	300 * 10 /l9
Sodium	144 mmol/L
Potassium	4.1 mmol/L
Urea	6.1 mmol/L
Creatinine	71 mol/L
Alkaline phosphatase	56 IU/L
Alanine aminotransferase	35 IU/L
Gamma-glutyl transferase	21 IU/L
Bilirubin	13 mol/L
Albumin	39 g/L
Fasting plasma glucose	5.3 mmol/L

What is the most likely causative organism?

A. Trypanosoma cruzi
B. Plasmodium ovale
C. Leishmania braziliensis
D. Leishmania donovani
E. Chlamydia trachomatis

C. Leishmania braziliensis

The most likely diagnosis in this case is mucosal leishmaniasis. The patient probably became infected with cutaneous leishmaniasis during his visit to South America, and received no/incomplete treatment. Mucosal leishmaniasis can occur years to decades after initial infection; typical features include nasal stuffiness and epistaxis in the early stages, progressing to ulceration and destruction of the nasal mucosa. Leishmania donovani is a recognised cause of visceral rather than mucocutaneous leishmaniasis.

Question 36 of 277

A 30-year-old male with HIV and a viral load of 10^6 and CD4 dropping from 500 to 300 cells/µl since his last visit. The above mentioned results are taken while he is on his third line of therapy.

What is the most likely cause of his repeated treatment failures?

A. Non-compliance
B. Infection with multi-resistant HIV strain
C. Co-infection with a new HIV strain
D. Superinfection with a new HIV strain
E. CYP interactions

A. Non-compliance

By far the most common cause of treatment failure in HIV therapy is treatment non-compliance.

Re-infection with new HIV strain can occur via co-infection or superinfection. Co-infection is defined as infection with 2 strains either simultaneously or within a brief period of time before infection with the first strain has an established immune response. Arbitrarily, HIV coinfection would occur within the first month or so of infection. Superinfection is defined as infection with a second strain after the initial infection and the immune response to it has been established.

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A 23-year-old man presents to the emergency department with a fever and a rash having recently returned back from a back-packing trip in rural Thailand where he had been trekking. He complains of general malaise for the last few days, a frontal headache and a rash he has noticed from last night. On examination he had tender cervical lymphadenopathy and a maculopapular rash over his trunk. There was also a small painless erythematous lesion on his lower calf with a necrotic centre.

Hb	95 g/l
Platelets	60 * 10 /l9
WBC	2.5 * 10 /l9

What is the most likely diagnosis?

A. Malaria
B. Dengue fever
C. Scrub typhus
D. Leptospirosis
E. HIV conversion illness

C. Scrub typhus

The eschar makes scrub typhus the most likely diagnosis. Malaria is of course common in this region and one would expect similar laboratory results, however the rash makes this diagnosis very unlikely. Dengue fever presents with a rash and fever, but also with associated arthropathy, classically lower back pain.

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A 40-year-old-year presents to the outpatient department with three separate lesions on his hands. He reports the skin lesions developed over several months and does not remember an initial trigger or injury resulting in the nodular-like ulcers.

The patient reports he has just arrived from rural Tanzania where he has lived most of his life. He is HIV positive on treatment but has not attended any recent clinic appointments. He reports he has no other medical conditions and is not on any other medications.

Fine needle aspiration of the lesions shows the presence of protozoa particles. A picture of one of the patient's lesions is shown below.

What is the most likely cause of the patient's skin lesion?

A. African trypanosomiasis
B. Chromoblastomycosis
C. Kaposi's sarcoma
D. Leishmaniasis
E. Leprosy

D. Leishmaniasis

Leishmaniasis is caused by the protozoa parasite *Leishmania tropica* or *Leishmania mexicana* which are transmitted via sandfly mainly in the tropics and sub-tropics of Africa, Asia and the Americas. Cutaneous leishmaniasis is the commonest form of leishmaniasis, which presents an open sore at the bite sites. The lesions can develop over several months or even years leaving unpleasantlooking scars.

African trypanosomiasis is caused by the protozoa parasite *Trypanosoma bruce* which is transmitted via tsetse flies. The condition causes systemic symptoms in the form initially of fevers, headaches and joint pain, before progressing to cognitive impairment. Cutaneous lesions are not commonly associated with the condition.

Chromoblastomycosis is normally found in rural region of tropical and subtropical climates however it is a chronic fungal infection of the skin. It can be caused by a number of different fungi that are implanted under the skin, often by thorns or splinters. Analysis of skin scraping would confirm the presence of muriform cells, indicative of the fungal infection.

Kaposi's sarcoma (KS) is a type of cancer that can form nodular, blotchy-like lesions red, purple, brown, or black in colour on the skin of the lower limbs, back, face and genitals, as well as mouth and organs lesions. KS is a result of immune suppression and infection by human herpesvirus 8; it is an aids defining condition and therefore is normally seen in untreated HIV. Diagnosis is via confirming the presents of human herpesvirus 8.

Leprosy is caused by the slow-growing, acid-fast bacillus, *Mycobacterium leprae* and is transmitted by humans via respiratory droplets. Diagnosis is confirmed via a skin smear from a lesion that is stained for mycobacteria.

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An elderly gentleman presents with a three day history of bloody diarrhoea and feverishness. He has no significant travel history. His past medical history is listed as hypertension, osteoarthritis and gout. On examination his temperature is 38.0ºC, heart rate 95/min, blood pressure 120/80 mmHg and his abdomen is soft and non-tender. A stool sample has grown *Salmonella*.

What is the best treatment?

A. Metronidazole
B. Doxycycline
C. Clarithromycin
D. Ciprofloxacin
E. Amoxicillin

D. Ciprofloxacin

The BNF recommends treating invasive diarrhoea (causing bloody diarrhoea and fever) with ciprofloxacin. Most viral or bacterial gastroenteritis do not require treatment. The BNF recommends antibiotics for bacterial gastroenteritis in severe infections or in immunocompromised patients. Clarithromycin is used for traveller's diarrhoea and non-invasive diarrhoeal illnesses when treatment is necessary.

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A 40-year-old gentleman was admitted with fever, dry cough, headache, abdominal pain and diarrhoea. Around ten days previously he had been complaining of intermittent fevers and night sweats but otherwise felt well. In the last 3 days, he had developed generalised abdominal pain and watery diarrhoea, along with a dry cough and headache. Prior to this he had felt constipated only having opened his bowels once in three days which he thought was unusual as he had eaten a large amount of fruit off the market stools in South Korea where he had recently been on a business trip.

On examination, he was notably jaundiced, had a macular rash over his chest and had tender hepatomegaly. Observations revealed a temperature of 40.1ºC, heart rate 38/min, regular and a blood pressure of 130/90 mmHg. ECG showed a sinus bradycardia.

Blood tests revealed:

WBC	14.0 * 10 /l9
Neutrophils	12.0 * 10 /l9
CRP	230 mg/l
Bilirubin	52 µmol/l
ALP	80 u/l
ALT	200 u/l
Albumin	32 g/l

What investigation would you do to obtain the diagnosis?

A. Blood cultures
B. Abdominal ultrasound
C. HIV test
D. Lumbar puncture
E. ECG

A. Blood cultures

Typhoid or enteric fever is the commonest serious tropical disease requiring treatment from Asia. Incubation period 7-18 days (range: 3-60 days). The highest incidence is found in south-central Asia and south-east Asia.

Patients most commonly present with fever, headache, constipation/diarrhoea, malaise, anorexia, dry cough, abdominal pain, hepatosplenomegaly, Rose spot rash, bradycardia and potentially misleading symptoms including meningism may occur.

Transmission is usually from contaminated food or water, occasionally direct faecal-oral transmission, shellfish taken from sewage-polluted areas, ingestion of contaminated milk and milk products and flies may cause human infection through the transfer of infectious agents to foods. Around 25% of those who contract typhoid fever become chronic carriers, as bacteria persist in the biliary tract after symptoms have resolved.

Investigations may reveal raised WCC, deranged LFTS. Blood cultures have the highest yield within the first week of symptoms (sensitivity 40-80%). Diagnosis can also be made from stool and urine cultures (become positive after the first week) and bone marrow cultures.

Treatment is with quinolones most commonly ciprofloxacin. However, there has been increasing reports of quinolones resistance in patients returned from Asia and intravenous ceftriaxone may be used. If quinolone resistance is confirmed azithromycin or cefixime are suitable oral alternatives for uncomplicated disease. Treatment should be continued for 14 days to reduce the risk of relapse. The addition of steroids may be helpful in severe cases

Complications can include GI bleeding, intestinal or biliary perforation, acalculous cholecystitis, pneumonia, myocarditis, pancreatitis, UTIs, osteomyelitis, meningitis and typhoid encephalopathy,

Question 41 of 277

A 33-year-old male with a previous history of asthma and HIV presents for review to HIV clinic. He has been noticing weight gain, marks on his abdomen and his partner has noticed that his face has been looking more heavy-set over the last two months. He was diagnosed as having HIV at the age of 19 following needle sharing and use of heroin. He has had good retroviral control following starting treatment at the age of 20 with tenofovir, emtricitabine, atazanavir and ritonavir. His asthma has been well controlled with only salbutamol until six months ago when due to recurrent exacerbation due to upper respiratory tract infection his treatment was escalated to include regular fluticasone. Repeat blood tests show an undetectable viral load and a CD4 count of 900 cells/microliter.

What is the most likely cause of his symptoms?

A. Weight gain secondary to tenofovir use

B. Weight gain secondary to emtricitabine use

C. Weight gain due to reduced exercise tolerance

D. Endogenous Cushing's syndrome

E. Iatrogenic Cushing's syndrome

E. Iatrogenic Cushing's syndrome

The correct answer is iatrogenic Cushing's syndrome. HIV protease inhibitors are potent P450 inhibitors. This means that there are many possible interactions that should be checked before starting treatment. In this gentleman, hepatic enzyme inhibition has resulted in increased steroid dose from his regular inhaler for his asthma. This also explains the physical description showing standard Cushingoid features. Endogenous Cushing's syndrome is possible but is less likely, and weight gain from other causes would not explain all the physical changes described.

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A 55 year-old patient presents with a 7 day history of nausea, breathlessness and feeling generally unwell. He is an HIV positive man who was recently started on anti-retroviral therapy in South Africa. On examination his chest is clear. His investigations are:

Chest X-ray: No abnormality seen

CD4 count	380 cells / mm³
Amylase	800 units / dL
Lactate	3.8 mmol / L

What is the most likely cause?

A. Pneumocystis jirovecii pneumonia (PCP)
B. Gallstone pancreatitis
C. Epstein-Barr Virus
D. Nucleoside reverse transcriptase inhibitor (NRTI) therapy
E. Non-nucleoside reverse transcriptase inhibitor (NNRTI) therapy

D. Nucleoside reverse transcriptase inhibitor (NRTI) therapy

NRTIs - in particular stavudine, didanosine and zidovudine - classically cause mitochondrial toxicity as an unwanted side effect. This can result in nausea, pancreatitis, lactic acidosis and lipoatrophy as demonstrated above.

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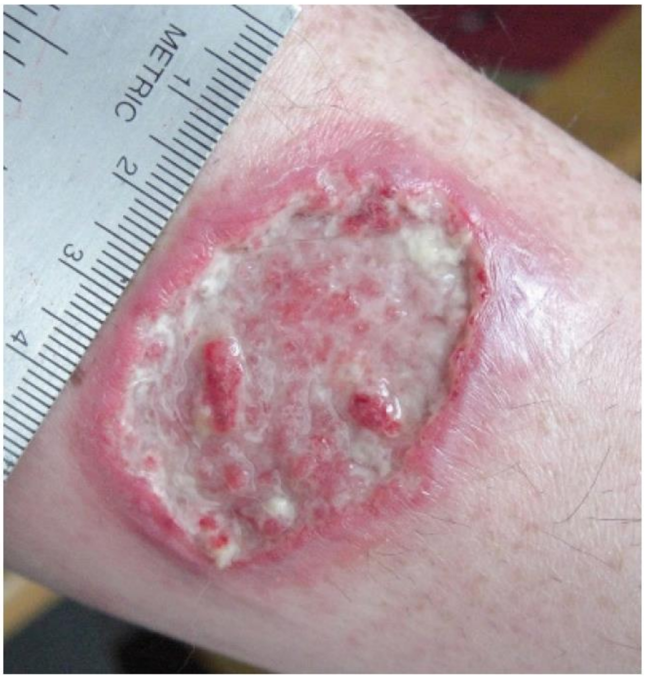
A 25-year-old woman of Caucasian origin presents to the dermatology clinic for review. She has been volunteering in India for the last 12 months and has been living with a local family. She first noticed a small lesion on her lower leg around 6 weeks ago. It has since progressed significantly, so she has returned to the UK to seek medical attention. The lesion is painless. She has no other lesions elsewhere on the body. There is no past medical history of note but her brother has ulcerative colitis.

Her skin is examined, as shown below:



What is the most likely cause of this skin lesion?

A. Buruli ulcer
B. Cutaneous leishmaniasis
C. Fish tank granuloma
D. Pyoderma gangrenosum
E. Yaws

B. Cutaneous leishmaniasis

The image shows a large shallow ulcerating lesion on the lower leg, with a moist surface. It is on an exposed area of the body and is a solitary lesion. Combined with her travel history, the most likely cause is **cutaneous leishmaniasis** . Lesions in cutaneous leishmaniasis can also be dry or covered with a crust or scab. Leishmaniasis is transmitted by bites of the sandfly and is due to a number of species of the protozoan parasite *Leishmania* . The main species causing cutaneous leishmaniasis in Asia, Africa, the Mediterranean and the Middle East are *Leishmania major*, *Leishmania tropica* and *Leishmania aethiopica*. Cutaneous leishmaniasis in Central and South America is caused mainly by *Leishmania braziliensis* and *Leishmania mexicana*.

Buruli ulcer is incorrect. This is another tropical ulcerating disease caused by *Mycobacterium ulcerans* . However, most cases occur in sub-Saharan Africa and tropical parts of Australia, and it is rare in Asia. It is also less likely in this patient because it tends to affect children under the age of 15, and affects people living in poverty in rural areas. It is not commonly seen in travellers.

Fish tank granuloma is incorrect. This condition is caused by *Mycobacterium marinum* and can be seen in people who have had exposure to fish tanks or are involved in fish or shellfish preparation. However, unlike the lesion seen in this patient, skin lesions are generally plaque-like or nodular, rather than ulcerating. Lesions can occur on the legs but are more common on the hands and arms.

Pyoderma gangrenosum is incorrect. This diagnosis is less likely in this patient because the ulcers of pyoderma gangrenosum are generally very painful, and have a blue or purple undermined edge, neither of which are present in this case. The patient also has no personal history of an underlying condition associated with pyoderma gangrenosum.

Yaws is incorrect. This condition is one caused by Treponema pallidum subspecies pertenue. It is endemic in a small number of countries in Africa, Asia and the South Pacific, but is most common in Papua New Guinea. The primary stage presents with a papule that can later ulcerate. It is most common in children between 5 and 10. It is unlikely as a diagnosis in this patient because it is extremely unusual in returning travellers as it requires close skin contact (non-sexual contact) with open skin lesions for transmission. It has also been eradicated from India.

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A 34-year-old lady presents to the emergency department.

She moved to the UK from India 10 years ago but moved to the area 3 years ago. She works as a medical coder in the hospital.

She presents with a 3-day history of severe generalised headache, a stiff and painful neck, photophobia, and fever. Her husband reports she is drowsy and has been confused since that morning.

She is generally well and takes no medications. Her only previous medical history is of groin lymphadenopathy that was investigated 5 years ago. A biopsy was taken which showed a caseating granuloma. She had no further follow-up at that time.

Investigations:

Chest x-ray lung fields clear. No obvious abnormalities Blood tests:

Haemoglobin	138 g/L
White cells	20.3x10^9/L
Platelets	310x10^9/L
CRP	>350mg/L
Glucose	6.8 mmol/L

Cerebrospinal Fluid (CSF): Appears turbid

Opening pressure	20 cmCSF
Protein	3280 mg/dL
White cells	466 cell/µL (polymorphonucleocytes 280 cell/µL and lymphocytes 206 cell/µL)
Red cells	4 cell/µL
Glucose	1.6 mmol/L
Lactate	5.6 mmol/L
CSF gram stain and PCR	pending

What is the best therapeutic regimen for this patient?

A. Start empirical ceftriaxone, vancomycin and dexamethasone
B. Await gram stain and viral PCR prior to starting a targeted therapy
C. Start aciclovir, ceftriaxone and vancomycin
D. Start isoniazid, rifampicin, pyrazinamide and ethambutol
E. Start isoniazid, rifampicin, pyrazinamide and ethambutol as well as ceftriaxone,vancomycin and dexamethasone

E. Start isoniazid, rifampicin, pyrazinamide and ethambutol as well as ceftriaxone,vancomycin and dexamethasone

This lady could have either bacterial meningitis or tuberculosis (TB) meningitis from her CSF results.

Although she has no chest signs of previous TB, she has had a previous biopsy suggesting she may have TB in her lymphatic system. This makes TB meningitis the most likely diagnosis, however, treatment should certainly be started empirically. This rules out answer 2 as the correct one. Since the gram stains and stain for acid fast bacilli would not yet be back from the lab, treatment should be started for both bacterial and TB meningitis.

Answers 1 and 3 do not include TB therapy and answer 4 does not cover for bacterial meningitis, therefore, these are incorrect answers.

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A 54-year-old man presents to the emergency department with one day of fever and a severely painful left leg. He has a past medical history of hypertension. He takes ramipril. He smokes cannabis occasionally and drinks approximately 10 units of alcohol a week. He has recently completed treatment with aciclovir for an episode of chicken-pox.

His observations are heart rate 111 beats per minute, blood pressure 86/48 mmHg, respiratory rate 21/minute, oxygen saturations 97% on room air and temperature 38.4ºC.

On examination, his left thigh is erythematous, tense and tender to touch. There is palpable crepitus.

Blood tests:

Hb	108 g/L	Male: (135-180)Female: (115 - 160)
Platelets	555 * 10 /L 9	(150 - 400)
WBC	23.1 * 10 /L 9	(4.0 - 11.0)
Na +	131 mmol/L	(135 - 145)
K +	4.8 mmol/L	(3.5 - 5.0)
Urea	14.2 mmol/L	(2.0 - 7.0)
Creatinine	191 µmol/L	(55 - 120)
CRP	351 mg/L	(< 5)

Given the likely diagnosis, what is his most significant risk factor the development of this condition?

A. Acute renal failure
B. Cannabis use
C. Chickenpox infection
D. History of hypertension
E. Weekly alcohol intake

C. Chickenpox infection

Chickenpox is a risk factor for invasive group A streptococcal soft tissue infections including necrotizing fasciitis

Chickenpox infection is correct. The patient has necrotizing fasciitis as evidenced by clinical evidence of soft tissue infection, with severe pain and crepitus. Preceding chickenpox infection is a risk factor for this condition.

Acute renal failure is incorrect. Chronic renal failure is associated with the development of necrotizing fasciitis. In this case, he has developed an acute kidney injury in the context of septic shock. Renal failure is a consequence of necrotizing fasciitis rather than a cause in this case.

Cannabis use is incorrect. Intravenous drug abuse is a risk factor for necrotizing fasciitis but not inhaled cannabis use.

History of hypertension is incorrect. Necrotizing fasciitis is associated with chronic disease. However, the conditions commonly implicated are diabetes and peripheral vascular disease rather than hypertension.

Weekly alcohol intake is incorrect. Alcohol abuse is associated withe the development of necrotizing fasciitis but he drinks within normal limits (i.e. < 14 units/week).

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A 35-year-old man presents to the emergency department with three days of headache, fever, diarrhoea and malaise. He has recently returned from Angola, where he was taking pictures of neolithic wall paintings. He has no past medical history and is on no regular medications. He works as a photo-journalist for a major international publication.

His observations are heart rate 124 beats per minute, respiratory rate 21/minute, blood pressure 85/54 mmHg, oxygen saturations 96% on room and air and temperature 39.3ºC.

Clinical examination reveals an unwell man. His capillary refill time is four seconds and his skin turgor is increased. He is oozing blood from his mucus membranes. Chest auscultation is normal. Heart sounds are normal with no murmurs. His abdomen is soft, diffusely tender but not peritonitic.

Intravenous access is obtained and intravenous fluids and empirical broad-spectrum antibiotics are administered. It is noted that he is oozing blood from his cannulae sites.

Blood tests:

Hb	111 g/L	Male: (135-180)Female: (115 - 160)
Platelets	543 * 10 /L 9	(150 - 400)
WBC	5.2 * 10 /L 9	(4.0 - 11.0)
Na +	129 mmol/L	(135 - 145)
K +	4.2 mmol/L	(3.5 - 5.0)
Urea	14.1 mmol/L	(2.0 - 7.0)
Creatinine	156 µmol/L	(55 - 120)
CRP	80 mg/L	(< 5)
Bilirubin	28 µmol/L	(3 - 17)
ALP	111 u/L	(30 - 100)
ALT	143 u/L	(3 - 40)
Î³GT	99 u/L	(8 - 60)
Albumin	28 g/L	(35 - 50)

Based on the probable diagnosis, to which zoonotic vector has he likely been exposed?

A. Bats
B. Birds
C. Cattle
D. Pigs
E. Rats

A. Bats

Exposure to bats/caves is a risk factor for Marburg virus, which results in a viral haemorrhagic fever

Bats are the correct answer. This man has evidence of viral haemorrhagic fever (VHF) with hypotension and multi-organ dysfunction. He has returned from a region where Marburg virus has previously been found. Additionally, he has been taking photos of neolithic wall paintings, which are typically found in caves. Exposure to bats and caves are risk factors for Marburg virus.

Birds are not the correct answer. While there is some suggestion that birds can carry infected ticks from continent to continent and thus spread viruses causing (VHF), this is not a common vector for the animal to human transmission of these diseases.

Cattle is incorrect. While people can occasionally contract Crimean-Congo haemorrhagic fever from exposure to infected cattle, there has never been an outbreak of this condition in Angola and he has not been exposed to slaughtered animals.

Pigs are incorrect. This is not a common zoonotic vector for transmission of viral haemorrhagic fever.

Rats are incorrect. While exposure to rats can result in hantavirus and haemorrhagic fever and renal dysfunction, this is more common in Asia, the Americas and Europe, rather than Africa.

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A 43 year old man attends for follow up at the Infectious Diseases clinic. He is an HIV-positive patient who has been well while on treatment for the last 13 years. His current treatment regimen is emtricitabine, atazanavir and tenofovir. He has however begun to notice altered taste and a difficulty in swallowing over the last couple of months. In the last few days he has even noticed a white coating in his mouth that is not removed by vigorous scrubbing with his toothbrush. He describes being compliant with his medications and not taking any illicit drugs. He no longer has unprotected sexual intercourse. The only change that is identified is that he recently visited his GP with non-specific symptoms and was started on a new medication.

CD4 count in clinic 4 months ago 526

CD4 count currently 210

What medication is the GP likely to have started?

A. Lansoprazole
B. Aspirin
C. Amitriptyline
D. Paracetamol
E. Simvastatin

A. Lansoprazole

Interactions between anti-reteroviral drugs and other more common medications are many and varied. It is a complex area but is one that is becoming more prominent given the larger prevalance of HIV. HIV is no longer a death sentence and with current treatments patients can expect to have an almost normal life expectancy. Full knowledge of all the possible interactions is unrealistic but some of the main ones should be kept in mind when prescribing any new medication. There is an excellent resource kept by the University of Liverpool that can be found at https://www.hiv-druginteractions.org/

In the case detailed above the patient is likely to have been started on a PPI such as Lansoprazole. Lansoprazole is known to dangerously lower the efficacy of atazanavir and this is exhibited by the dramatic fall in CD4 count. This patient also seems to have developed oesophageal candidiasis and so urgent management to manage his HIV is required to avoid potentially deadly opportunistic infections.

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A 48-year-old woman originally from South Africa has lived in the UK for 17 years and works as a librarian. She was diagnosed with HIV 18 months ago after she discovered her husband had been having an affair. Her lowest CD4 count was 211 /mm3. She was commenced on Anti-Retroviral Therapy (ART) on diagnosis with Tenofovir, Emtricitabine and Kaletra (Lopinavir/Ritonavir) and Cotrimoxazole.

Her past medical history prior to diagnosis was unremarkable. She has been well on her ART until 3 weeks ago when she was seen in HIV clinic with a 4-week history of a cough and weight loss of 3 kilogrammes.

She had a chest x-ray showing new right apical nodular opacities and had scanty Acid Fast Bacilli on sputum smear microscopy. Her CD4 count was 312 /mm3 with and she was commenced on standard TB therapy with Rifampicin, Isoniazid, Ethambutol and Pyrazinamide, as well as switching Kaletra to Efavirenz.

Today she has been brought into hospital by the Police, she was found naked wandering in her street, accosting passers-by and asking them to help her remove a device planted under her skin by the CIA to monitor her thoughts.

Her observations are within normal limits. She is moderately difficult to examine but there is no focal neurology detectable. She has mildly reduced air entry in her right upper lobe and her examination is otherwise normal.

Results:

Hb	8.1 g/dl
MCV	84 fl
Platelets	86 * 10 /l9
WBC	4.5 * 10 /l9
CRP	27 mg/l
ESR	41 mm/hr

Urea & electrolytes, liver function tests and thyroid function tests were normal. A CT head and lumbar puncture were also unremarkable.

What is the cause of her admission?

A. TB meningitis
B. HIV encephalopathy
C. Ethambutol toxicity
D. Efavirenz toxicity
E. Immune thrombocytopaenic purpura

D. Efavirenz toxicity

This is Efavirenz toxicity.

Rifampicin causes potent enzyme induction and therefore dramatically reduced the level of Protease Inhibitors. Efavirenz is less susceptible to inducing effects Rifampicin, therefore the switch to Efavirenz was logical.

Efavirenz however can cause neuropsychiatric toxicity (due to variable metabolism which is genetically determined) which is the cause of psychosis here. Normal LP excludes TB meningitis. Ethambutol causes optic neuritis, so monitoring of visual acuity during treatment is essential. HIV encephalopathy is possible but unlikely to present so acutely and the temporal relation to Efavirenz introduction, in this case, is the key.

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A 26-year-old woman presents to the emergency department with right upper quadrant pain which is worse when she takes a deep breath in. Her symptoms started three days earlier and have been progressively getting worse. She has no past medical history and takes no medications apart from the oral contraceptive pill and paracetamol to help control the pain. She has also noticed an increase in vaginal discharge over the last two weeks and has noticed an unpleasant smell with it. She works in an investment bank. On examination, she has right upper quadrant tenderness, but the abdomen is soft and no organomegaly is noticed. Abdominal ultrasound demonstrates the presence of gallstones in the gallbladder but is otherwise normal.

What treatment is most likely to resolve the cause of her underlying problem?

A. Cholecystectomy
B. Trimethoprim
C. Antiviral treatment
D. Antiretroviral treatment
E. Ceftriaxone

E. Ceftriaxone

Intramuscular ceftriaxone is the treatment of choice for *Gonorrhoea*

The correct answer is ceftriaxone. This woman has unfortunately developed Fitz-Hugh-Curtis syndrome following pelvic inflammatory diseases. The key aspects are RUQ pain worse with breathing; symptomatic of peri-hepatic adhesions. This combined with vaginal discharge are highly suggestive of Fitz-Hugh-Curtis syndrome, which can be treated with antibiotics. Cholecystectomy would be appropriate management if the US had found evidence of cholecystitis. Trimethoprim would be appropriate if there was evidence of urinary symptoms, whilst there is no evidence to suggest that this is an AIDS-defining illness needing antiretroviral treatment. Antiviral treatment would imply viral hepatitis, but there is no clear evidence of hepatitis.

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A 28-year-old female presents with a 4-day history of fevers and joint pain. She has recently returned from a 3 month gap year trip to the South-East Asia three days ago and reports no ill health during her travels. She has no past medical history, does not smoke, drinks minimal alcohol and denies the use of illicit drugs. During her travels, she reports two episodes of unprotected sexual contact with a non-regular partner. Although she knew she would be entering a malaria area and was indeed bitten by mosquitoes on a number of occasions, she did not take any malaria prophylaxis.

On examination, heart sounds and chest examination are both normal. A maculopapular rash is noted on her left thigh and right upper arm, with bilateral conjunctival injection. Abdominal examination reveals a soft abdomen with no masses. She has a significantly joint and muscle pains, limiting your neurological examination. She is alert and orientated to time and place, scoring 10/10 on abbreviated mental testing. Her blood tests are as follows:

Hb	109 g/l
Platelets	45 * 10 /l9
WBC	3.5 * 10 /l9
Na +	137 mmol/l
K +	3.8 mmol/l
Urea	7.1 mmol/l
Creatinine	100 µmol/l
CRP	70 mg/l
Bilirubin	7 µmol/l
ALP	30 u/l
ALT	162 u/l

Her first malaria film is negative and a chest radiograph is unremarkable.

What is the most likely diagnosis?

A. Gonorrhoea with reactive arthritis
B. HIV seroconversion
C. Dengue fever
D. Malaria falciparum
E. Typhoid

C. Dengue fever

Retro-orbital headache, fever, facial flushing, rash, thrombocytopenia in returning traveller → ?dengue

The context of this patient should alert you to that of fever in a returning traveller. This is a patient who presents with a combination of fever, myalgia and arthralgia, skin rash, conjunctival injection, raised ALT, thrombocytopaenia and leucopenia, strongly suggestive of dengue fever. The key differential for a returning traveller from South East Asia, where ingestion of contaminated water is possible, is to consider is also typhoid fever, which also presents with a skin rash (classically a rose spot) with diarrhoea and vomiting much more prominent. Although she had unprotected sex during her travel, gonorrhoea presents more typically with a migratory arthritis and tenosynovitis with discharge. Leucopenia is classical of dengue fever and does not necessarily suggest immunodeficiency, possibly secondary to HIV.

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A 39-year-old pet shop owner is referred to the dermatology clinic due to lesions on his right hand. He describes noticing a lump over his 2nd metacarpophalangeal joint 2 months ago, this has since increased in size and crusted over, and further similar lesions have appeared. He denies pain or itching and has tried a number of topical emollients without improvement. He has no past medical history of note and no allergies.

On examination, there are multiple crusted lesions measuring 1-2cm in diameter on his knuckles and the dorsum of his hand. There are no other skin or mucosal changes.

A biopsy is taken for analysis:

Skin biopsy	Granulomatous dermatitis with inflammatory infiltrate

What is the most likely causative organism?

A. Human papilloma virus
B. Mycobacterium leprae
C. Mycobacterium marinum
D. Mycobacterium tuberculosis
E. Pseudomonas aeruginosa

C. Mycobacterium marinum

Fish tank granuloma is caused by *Mycobacterium marinum*

This patient has an infection with the bacterium ***Mycobacterium marinum***, a species of mycobacteria commonly contracted through exposure to swimming pool water or contact with fish e.g. through fish tanks. This patient is a pet shop owner and has likely contracted the illness through occupational exposure. Notably, in such patients, the dominant hand is most frequently affected. After a 3-4 week incubation period, a single lump or pustule appears that breaks down to form a crusted lesion. The knuckles are commonly affected and a biopsy of the lesion demonstrates granulomatous dermatitis and inflammatory infiltrate within the dermis. Treatment is with antibiotics e.g. doxycycline.

Human papilloma virus (HPV) is a common cause of viral warts that are commonly located on the cutaneous surface (e.g. hands, knees and feet) or genitalia. Common warts found on the hands (also known as verruca vulgaris) are hyperkeratotic, rough and papillomatous lesions. However, on dermoscopy, multiple papillary capillaries can be visualised rather than granulomatous dermatitis with inflammatory infiltrate.

Mycobacterium leprae causes leprosy. Symptoms of leprosy include hypopigmented patches of skin associated with sensory loss. The areas of skin affected by the disease are not limited by exposure, in contrast to this patient's presentation.

Mycobacterium tuberculosis can cause cutaneous symptoms such as tuberculous gumma from the haematogenous spread of disease, lupus vulgaris and miliary tuberculosis where there are multiple millet-sized red skin lesions. These lesions are not seen in this patient and there is no other history to suggest underlying tuberculosis.

Pseudomonas aeruginosa is implicated in skin infections such as external otitis or hot tub folliculitis (acquired from chlorinated hot tubs). Infection is particularly more common in the immunocompromised and is unlikely to be the causative pathogen in this case.

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A 65-year-old man has just returned from a trip to India one week ago. He has had bloody diarrhoea and fevers for the last two weeks and noted rose coloured spots on his abdomen yesterday. Apart from a prosthetic aortic valve, he has no significant past medical history. His blood tests indicate raised inflammatory markers and stool microbiology has found a gramnegative bacillus identified as a non-typhoidal *Salmonella*. Sensitivities are pending.

Which one of the following options is best initial empiric management?

A. Amoxicillin + clavulanic acid
B. Gentamicin
C. Ciprofloxacin
D. Clindamycin
E. Not for antibiotics

C. Ciprofloxacin

According to the NICE guidelines, anyone above the age of 50, immunocompromised or has cardiac valve disease/endovascular abnormalities should be treating empirically with ciprofloxacin 500mg BD when they have been diagnosed with non-typhoidal *Salmonella* gastroenteritis.

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An 80-year-old lady presents from a nursing home with a one-day history of fevers, drowsiness and vomiting. She has been unwell, not eating for the past 3 days according to her carer. Recently she has been exhibiting behavioural changes, crying and agitation when lights are turned on in the room, and holding her neck. She has a back ground of Alzheimer's dementia, hypertension, congestive cardiac failure(CCF) and chronic kidney disease.

On examination, she is febrile with a temperature of 38.1oC. Heart rate 112bpm, respiratory rate 20breaths per minute and oxygen saturations of 94% on air. On auscultation, there are bibasal crepitations and cardiovascular examination is consistent with mild CCF. There is neck stiffness and photophobia on cranial nerve examination, pupils equal and reactive to light. Kernig's sign is positive.

Investigations:

Na+	129mmol/l
K+	4.8 mmol/l
Urea	10.9 mmol/l
Creatinine	123 µmol/l
Serum glucose	5.9mmol/l
C Reactive protein	95mg/l
Haemoglobin	126 g/l
White cell count	16.4 x 10^9/L
INR	1.2

Cerebro-spinal fluid (CSF) analysis:

Opening pressure	25 cmH20
Protein	1.8 g/L
Glucose	2.6 mmol/l
White cell count	>1000 per mm³
Gram Stain	Gram-positive rods seen
Colour	Cloudy, turbid

What is the likely causative organism?

A. Streptococcus pneumoniae
B. Listeria monocytogenes
C. Staphylococcus aureus
D. Neisseria meningitidis
E. Haemophilus influenzae

B. Listeria monocytogenes

This lady has bacterial meningitis as evidenced from her LP results. There is only one gram-positive bacilli in the list and that is *Listeria monocytogenes*, a common causative organism of meningitis in the extremes of age.

Gram-positive bacilli (or rods): ABCDL

- Actinomyces
- Bacillus anthracis
- Clostridium,
- Corynebacterium
- Diphtheria
- Listeria monocytogenes

Gram positive cocci:

- Streptococci
- Staphylococci

Gram-negative cocci: NNM

- Neisseria (Neisseria meningitides, Neisseria gonorrhoea)
- Moraxella

Gram negative bacilli (or rods): almost every other pathogenic gram negative bacteria.

- Salmonella spp.
- Escherichia spp.
- Pseudomonas
- Bacteroides

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A 24-year-old male patient is referred to HIV clinic following positive testing on screening in a local GUM clinic. His CD4 count is 1020 cells/microlitre and viral load is 10,123 copies/ml. He is offered to start antiretroviral treatment or monitoring. He has a past medical history of depression and has emigrated from Nigeria three years ago. He is planning to return abroad in two years.

What benefit has been established from starting early antiretroviral treatment as opposed to deferring treatment?

A. Increased efficacy
B. Reduced risk of AIDS and mortality
C. Reduced drug resistance
D. Reduced toxicity
E. Reduced anxiety

B. Reduced risk of AIDS and mortality

The correct answer is the reduced the risk of AIDS and mortality. BHIVA has made the recommendation to offer antiviral therapy regardless of the CD4 count. This is partly due to results of the SMART study which demonstrated reduced chance of AIDS, a serious non-AIDS event or death by 57% with similar results in high, middle and low-income countries. Data on virological efficacy, drug resistance and toxicity is not yet established. Reduced anxiety may also be a benefit but has not been as definitively established.

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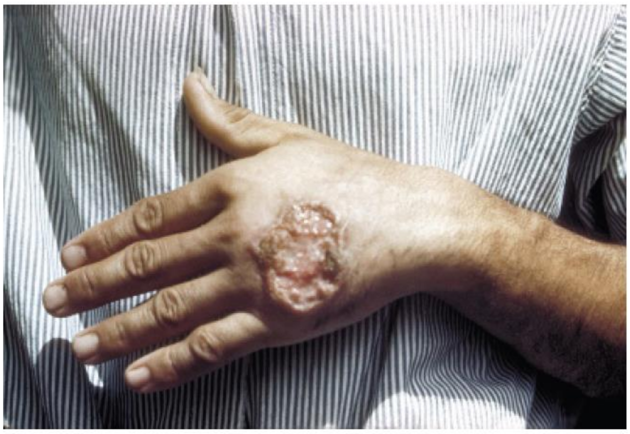
A 42-year-old man attends the GP with a lesion on his left hand. He says it first appeared as a red spot 2-3 weeks ago when he returned from Iraq. However, since then the lesion has been progressively enlarging. There is no history of pain or itch and he otherwise feels well. He is normally fit and well and takes no regular medications.

On examination, his observations are all within the normal range. His hand appears as shown below. On further inspection, there is sparing of the mucous membranes and no further lesions are found on his body.



What is the most likely diagnosis?

A. Anthrax
B. Chagas disease
C. Leishmaniasis
D. Leprosy
E. Lyme disease

C. Leishmaniasis

This patient has a superficial cutaneous lesion on his left hand with central ulceration. The surrounding skin is not affected. Given the history of rapid growth from a small red papule and a recent tour in Iraq, it is likely that this patient has leishmaniasis, a parasitic disease spread by sandflies. There are different species of leishmania that are responsible for different manifestations of the disease, with cutaneous leishmaniasis being the most common. Initially, a small red papule appears at the site of the sandfly bite which gradually enlarges followed by central ulceration before spontaneously resolving. The time to resolution varies between 2 months and 1 year. The Middle East, North Africa, and Asia are more common areas for contracting the disease.

Anthrax is a bacterial disease caused by *Bacillus anthracis*. Infection is associated with exposure to infected carcasses and the introduction of the organism into the body through a superficial wound. An initial pustule appears at the site of infection which progresses to a black eschar (a necrotic-looking ulcer with a black scab). The infection usually resolves spontaneously, but in some can lead to death. The lesion shown in this question is not that of a black eschar but rather a crusted ulcer from a sandfly bite. This makes leishmaniasis the correct diagnosis.

Chagas disease is a parasitic disease spread by triatomine or 'kissing' bugs. The disease is prevalent in Latin America and consists of an initial acute phase and chronic phase. In the earlier stages of the disease, symptoms include generalised malaise, fever, headaches, and myalgia along with anorexia, nausea, and vomiting. In others, a chagoma (an indurated, oedematous skin lesion) may be seen. In contrast, the patient in this question has returned from the Middle East with a cutaneous ulcerated lesion, making Chagas disease unlikely.

Leprosy is a granulomatous disease caused by *Mycobacterium leprae* that affects the peripheral nerves and skin. Initial symptoms include numbness, typically with loss of temperature followed by light touch, pain, and finally deep pressure. Cutaneous lesions follow later in the disease and appear as hypopigmented patches of skin with associated sensory loss. This patient has a painless, crusted ulcer with no other lesions or neurology making leprosy an unlikely diagnosis.

Ticks are well-known to spread Lyme disease, a bacterial infection caused by Borrelia *burgdorferi* . The most common dermatological manifestation of Lyme disease is a bulls-eye rash that appears at the site of the tick bite. Other symptoms include fever, lethargy, and arthralgia that are not described in this question.

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A 63 year old man, originally from Ukraine is admitted to hospital with a right sided pleural effusion. On admission he is febrile at 37.8 C. He reports 6kg of weight loss, and night sweats over the previous 4 months.

His pleural effusion is exudative with a pH of 7.4.

A CT chest/abdo/pelvis reveals a right sided pleural effusion, multiple enlarged lymph nodes in his abdomen and mediastinum. A pleural biopsy reveals scanty Acid Fast Bacilli and he is commenced on treatment for tuberculosis with Rifampicin, Isoniazid, Ethambutol, Pyrizinamide. PCR confirms this to be Mycobacterial Tuberculosis complex with wild type rpoB, katG, inhA genes. His HIV serology is positive and his CD4 count is 47 cells/mm3

One week following admission he is commenced on anti-retroviral treatment with Emtricitabine, Lamivudine and Efavirenz with Co-trimoxazole.

Three weeks later he becomes more unwell. He has daily fevers up to 39°C.

On examination he is febrile 38.1 with an associated tachycardia of 119 beats/min, his blood pressure is 112/67 mmHg and respiratory rate is 22 breaths/min. His oxygen saturations are 93%. He has reduced air entry at the right base, but no other abnormality on examinations.

His results are as following:

Hb	10.1 g/dl
MCV	82 fl
Platelets	97 * 10 /l9
WBC	7.8 * 10 /l9
Neutrophils	6.3 * 10 /l9
Lypmphocytes	1.2 * 10 /l9
Eosinophils	0.02 * 10 /l9
ESR	62 mm/hr
Bilirubin	9 µmol/l
ALP	312 u/l
ALT	153 u/l

γGT	456 u/l
Albumin	22 g/l
CRP	67 mg/l
Lactate	1.1 mmol/l

Other results were as follows;

Urea and electrolytes: normal.
Clotting screen: normal
Blood cultures: negative (x3)
Urinalysis: normal
ECHO: normal
CXR: right pleural effusion
USS abdo: several abdominal nodes visible, nil else.
Toxoplasmosis serology: negative
CMV: IgG negative IgM: negative
EBV: IgG positive IgM: negative
Cryptococcal antigen: Negative
Leishmania serology: negative

What is the most likely diagnosis?

A. Pneumococcal pneumonia
B. Drug resistant tuberculosis
C. Infectious mononucleosis
D. Immune reconstitution inflammatory syndrome
E. Burkitts Lymplhoma

D. Immune reconstitution inflammatory syndrome

This is a classic history of Immune Reconstitution Inflammatory syndrome (IRIS). IRIS is a paradoxical worsening of symptoms, due to increasing inflammatory activity with a recovering immune system. It is seen in the context of anti-retroviral therapy.

In this case we are reassured of a correct diagnosis to TB with the PCR result, and the initial PCR results suggest a fully susceptible organism (although this will need to be confirmed by culture and sensitivity testing.) You may not have a luxury of a confident diagnosis of TB, and patients with HIV may have many different opportunistic infections. Therefore IRIS is a diagnosis of exclusion. In this case an extensive screen has revealed no alternative cause for his symptoms.

The diagnosis of IRIS depends on all three of :

1. Antecedants:

- Diagnosis of TB fulfilling WHO clinical criteria
- Initial response to anti-retroviral treatment

2. Clinical (1 major criteria or 2 minor)

- Major: New or worsening cold abscess lymph nodes, radiological features of TB, CNS TB or TB serositis
- Minor: New or worsening constitutional symptoms, respiratory symptoms or abdominal pain.

3. Alternative explanations for clinical deterioration must be excluded.

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A 17 year old girl presents to A&E accompanied by her anxious mother. She was born in England, but both her parents were born in a rice growing community in rural China. She has recently returned from a holiday where she was visiting her family in China. They live on a farm where she had close contact with dogs, sheep and pigs.

She arrived home one week ago. She was taking Mefloquine for malaria prophylaxis.

She was complaining of fever and a headache yesterday. Today she has been confused and disorientated.

She is previously fit and well, she has had all her immunisations according to the UK immunisation schedule, with normal growth and development. She does well at college and is a keen member of her college canoe club. She has no allergies.

On examination she is febrile at 38.9°C but haemodynamically stable. She appears confused and has an obvious difficultly walking. Examination of her cranial nerves is difficult but unremarkable. She has normal power and increased tone in her arms, which is more pronounced on the left side, with hyperreflexia bilaterally. She has several writhing involuntary movements of her upper limbs during the consultation.

A CT scan shows hypodensity in the thalami and basal ganglia bilaterally, more pronounced on the left side.

A lumbar puncture reveals a lymphocytic CSF with a raised protein.

What is the most likely diagnosis?

A. Herpes Simplex Encephalitis
B. Japanese Encephalitis
C. Alcohol consumption
D. Rabies
E. Mefloquine toxicity

B. Japanese Encephalitis

The most likely diagnosis in this case is **Japanese Encephalitis**. The patient's history of travel to a rural area in China, where she had close contact with animals, combined with her presenting symptoms of fever, headache, confusion, and difficulty walking, as well as the CT scan findings of hypodensity in the thalami and basal ganglia bilaterally are all consistent with Japanese Encephalitis. This disease is a mosquito-borne flavivirus infection that predominantly affects people living or travelling to Asia.

Herpes Simplex Encephalitis is less likely due to the patient's travel history and exposure to animals. Although Herpes Simplex Encephalitis can also present with fever, headache, and confusion, it typically has a more acute onset and would show different findings on imaging studies (such as temporal lobe involvement on CT or MRI).

Alcohol consumption is an unlikely cause for this patient's presentation. While excessive alcohol intake can lead to neurological symptoms such as ataxia and confusion, there is no indication from the patient's history that she has been consuming large amounts of alcohol. Furthermore, her CT scan findings and lumbar puncture results do not support this diagnosis.

Rabies could be considered given the patient's close contact with animals during her trip; however, her clinical presentation does not fit the classic signs of rabies. Rabies usually presents with hydrophobia (fear of water), hypersalivation, and progressive paralysis. Additionally, while rabies can affect the central nervous system leading to encephalitis-like symptoms, it would not produce the specific CT scan findings seen in this case.

Finally, **Mefloquine toxicity** is an unlikely diagnosis because although mefloquine can cause neuropsychiatric side effects like dizziness or insomnia in some individuals, the patient's symptoms and investigation results are much more consistent with Japanese Encephalitis. Mefloquine toxicity would not typically cause the neurological findings observed in this case, nor would it explain the CT scan and lumbar puncture results.

Question 59 of 277

A 45-year-old male from Afghanistan presents with a rash all over his body. He describes the rash and suggests that it was initially multiple specks of light skin that were not raised or roughened. they have now progressed to plaques, some of which have become nodular.

He is otherwise well and his past medical history is significant only for the successful treatment of visceral leishmaniasis in his home country some 5 years ago.

What is the most likely underlying diagnosis?

A. Leprosy
B. HIV
C. Post kala azar dermal leishmaniasis (PKDL)
D. Histoplasmosis
E. Pityriasis versicolor

C. Post kala azar dermal leishmaniasis (PKDL)

This is a classic description of post kala azar dermal leishmaniasis (PKDL) from an area where the disease is endemic. Leishmaniasis can manifest as cutaneous, muco-cutaneous and visceral disease. PKDL is a chronic skin condition that arises after the treatment of visceral disease. The skin condition can arise months or years after successful treatment and often presents with erythematous or hypo-pigmented macules that may progress to become nodular. Clinically the lesions look very similar to pityriasis versicolour, however this has a predilection for the chest and back and is likely to have a scaly appearance rather than nodular. Perhaps the most important differential is leprosy and lesions should be tested for anaesthesia which would be strongly indicative for leprosy over PKDL.

Although HIV can cause a myriad of skin pathology there is nothing given in the question to make this a likely diagnosis and the best answer is therefore PKDL.

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A 40-year-old male presented to the Emergency Department with a decreasing level of consciousness. He had just returned from the Hajj pilgrimage in Saudi Arabia. He was completely healthy during his travels, but he started to develop a fever two days after his return. The fever was associated with a severe frontal headache and photophobia. These symptoms persisted for the last two days and he started to become sleepier.

On examination: blood pressure 100/80 mmHg, pulse rate 120/min, temperature 39.2ºC, respiratory rate 27/min. He had evidence of neck stiffness. Blood investigations showed:

Hb	123 g/l
Platelets	130* 10 /l9
WBC	13* 10 /l9

What is the most likely diagnosis?

A. Meningococcal meningits type A
B. Meningococcal meningits type B
C. Meningococcal meningits type C
D. Malaria
E. Hemorrhagic fever

A. Meningococcal meningits type A

The candidate should have no difficulty in recognising that this patient has meningitis. However, good knowledge of the epidemiology of meningitis is required to answer the question completely.

The most common cause of meningitis in Saudi Arabia during Hajj is meningococcal meningitis type A. It is also the leading cause of epidemics of what is known as the meningitis belt in Africa along the equator line including Sudan, Ethiopia and Nigeria.

Type B and Type C are mainly responsible for sporadic cases of meningitis in Europe and South America.

Question 61 of 277

A 54-year-old intravenous drug user is newly diagnosed with HIV, after presenting with progressive shortness of breath. He was diagnosed with pneumocystis pneumonia and commenced on appropriate treatment.

His CD4 count is 54 cells/mm3 and his viral load is 1.7 x10^7 copies per ml.

As part of his routine workup, it is also revealed that he has co-infection with hepatitis C. A test for HIV viral tropism is reported as dual tropism virus.

Which drug is unlikely to be effective in this man's treatment?

A. Maraviroc
B. Raltegravir
C. Tenofovir
D. Ribavirin
E. Abacavir

A. Maraviroc

HIV binds to the CD4 cell via the CD4 receptor. This interaction also depends on viral interaction with the CD4 co-receptor. There are two forms of CD4 co-receptor CCR5 and CXCR4.

The test for viral tropism determines which of these co-receptors HIV will bind to. Maraviroc blocks HIV binding to the CCR5 receptor, and therefore is an effective drug in a 'CCR5 tropic' virus. However, it is not effective if the virus is 'CXCR4 tropic' or 'dual tropic'. The tropism of HIV will not affect the use of any of the other drugs listed.

It is routine to test HIV viral tropism on diagnosis, or on virological failure to assess if maraviroc is a therapeutic option.

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A 45-year-old man attends with a skin rash after returning to the UK following a 5-month stay in Sudan. He explained that he sustained multiple fly bites during his travels and that some of the bites became raised red bumps that became scabby and painful, the majority of these have not healed.

On examination, there is a handful of atrophic scars over his cheeks, arms, and ankles. There are also raised scaly lesions with interspersed erythematous ulcerated papules, some of which are oozing pus in the same distribution.

What diagnostic tool would typically be used to confirm the likely diagnosis?

A. Blood cultures
B. Bone marrow aspiration
C. Punch biopsy of a lesion
D. Skin scraping of a lesion
E. Splenic aspirate

C. Punch biopsy of a lesion

Cutaneous leishmaniasis is typically diagnosed using a punch biopsy

The most likely diagnosis here is cutaneous leishmaniasis. This should be suspected in any patient with non-healing, scaling skin lesions, particularly if they have been in an endemic area.

Punch biopsy is correct. This is the typical method of diagnosing cutaneous leishmaniasis.

Blood cultures is incorrect. Leishmaniasis is a parasitic disease, blood culture is therefore not the typical diagnostic tool. In some cases, evidence of leishmaniasis can be seen on blood film.

Bone marrow aspiration is incorrect. This would be a diagnostic tool if we were concerned about visceral leishmaniasis. Fevers, substantial hepatomegaly, and blackening of the skin would raise suspicion of visceral leishmaniasis. None of these features are mentioned in the stem for this patient.

Skin scraping of a lesion is incorrect. Generally, leishmaniasis is diagnosed with PCR testing of a punch biopsy. This can also be performed on a sample obtained through skin scraping, but the diagnostic sensitivity is much poorer.

Splenic aspirate is incorrect. This would be a diagnostic tool if we were concerned about visceral leishmaniasis. Fevers, substantial hepatomegaly, and blackening of the skin would raise suspicion of visceral leishmaniasis. None of these features are mentioned in the stem for this patient.

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A 60-year-old man presented with history of excessive urination especially during night which disturbs his sleep, excessive thirst and tiredness for the past 2 months. He was a known HIV positive patient for which he was on highly active anti-retroviral therapy (HAART) since 6 months. Clinical examination was within normal limits. HIV related diabetes mellitus was suspected by the treating physician. His blood investigations are as follows

Hb	150 g/l
Platelets	150 * 10 /l9
WBC	7 * 10 /l9
Fasting blood glucose	10 mmol/l
Post prandial blood glucose	13.9 mmol/l
Serum creatinine	130 umol/L

He was planned to be started on metformin therapy.

Which of the following investigations is important to be checked before starting metformin in this patient?.

A. Liver enzymes
B. Venous lactate
C. Serum triglycerides
D. Serum albumin
E. Serum ammonia

B. Venous lactate

Patients with HIV are at increased risk of lactic acidosis and hence it is prudent to check lactate levels before starting metformin in such patients.Lactic acidaemia with no or mild symptoms was detected in 8% to 21% of patients receiving at least 1 nucleoside reverse transcriptase inhibitor, versus 0% to 1% of patients receiving no antiretroviral therapy. Symptomatic lactic acidaemia is less common (occurring in about 1.5%2.5%).Monitoring for lactic acidosis is recommended in the initial few months of metformin therapy. Patients with impaired renal function(serum creatinine>1.5 mg/dl) or with a venous lactate level more than twice the normal level prior to treatment should not be started on metformin.

This patient has an elevated serum creatinine but not >1.5mg/dl. So, he should get a venous lactate estimation before being started on metformin therapy considering the evidences so far.

Question 64 of 277

A 25-year-old man reports feeling unwell with fever, generalised muscle aches, headache and widespread rash 1 week after returning from Thailand. On admission his chest X-ray is unremarkable. His urine dip showed 1+ protein only and viral swabs are negative. Bloods are also normal aside from low platelet count. On day 2 of admission his blood pressure drops and repeat bloods show a further fall in platelets and reduction in haemoglobin count.

What is the most likely diagnosis?

A. Measles
B. Malaria
C. Dengue fever
D. Gonococcal septicaemia
E. Herpes simplex encephalitis

C. Dengue fever

Retro-orbital headache, fever, facial flushing, rash, thrombocytopenia in returning traveller → ?dengue

All plausible options and further history including prior vaccination, sexual history and malaria prophylaxis would be helpful. The history and deterioration are most likely due to dengue fever and subsequent 'dengue shock syndrome'. Measles would be unlikely at his age, malaria does not classically cause a widespread rash and herpes simplex would not cause a drop in the platelets. Gonorrhoea fits quite well and has a similar incubation period (4-7 days) . The patient would have normally have reported an isolated septic joint by this stage in the disease.

Question 65 of 277

A 68-year-old lady presents with lower abdominal pain, dysuria, and urinary frequency. A urine dip was positive for nitrites and leukocytes. She has a past medical history of recurrent C.difficile infection which has been treated with two tapering courses of vancomycin.

Microbiology results are as follows:

Urine culture	Vancomycin Resistant Enterococcus (VRE

How will you manage this patient?

A. Amoxicillin
B. Linzolide
C. Gentamicin
D. Penicillin
E. Erythromycin

B. Linzolide

Vancomycin Resistant Enterococcus (VRE) can be treated with linezolid, daptomycin and tigecycline

Enterococci are gram positive gamma-haemolytic Streptococci. Most important are E. faecalis and E. faecium. They are part of the normal flora of the human intestine, the female genital tract, and urinary tract. They are intrinsically of low pathogenicity however have a natural high resistance to antibiotics.

Vancomycin Resistant Enterococcus (VRE) are important nosocomial pathogens for which there are limited treatment options. Predisposing factors for VREs include previous treatment with antimicrobials (especially vancomycin, cephalosporins and broad-spectrum antibiotics).

Most VRE have high-level resistance to β-lactams and aminoglycosides.

Management of VRE may include removal of catheters and drainage of abscesses

If after clinical assessment and primary management, antimicrobial treatment of VRE is considered necessary, a drug should be selected based upon susceptibility testing of the organism involved. Nearly all strains of E. faecalis, including VRE strains, are susceptible to ampicillin. VREs also often remain sensitive to chloramphenicol, rifampicin and tetracyclines. A number of newer agents include linezolid, daptomycin and tigecycline.

Gentamicin can be used synergistically with another antibiotic, however it should not be used alone.

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A 26-year-old woman presents to the Emergency Department with a one week history of cough, fever and headache. She returned from Indonesia one week ago.

On examination she appears drowsy and a little muddled. She has no photophobia or focal neurological deficit. She has mild hepatosplenomegaly but no palpable lymphadenopathy. Her chest is clear and heart sounds are normal. She has a macular rash on both legs with a greyishblack scab-like lesion on her left shin.

Investigations results:

Chest x-ray: Bilateral patchy consolidation

Blood culture: Pending

Hb	120 g/l
Platelets	130 * 10 /l9
WBC	18 * 10 /l9
Blood film	Left shifted neutrophils in large numbers
Na +	142 mmol/l
K +	4.1 mmol/l
Urea	6 mmol/l
Creatinine	90 µmol/l
Bilirubin	23 µmol/l
ALP	170 u/l
ALT	50 u/l
Albumin	35 g/l

What is the most appropriate first line treatment?

A. Azithromycin
B. Chloroquine
C. Doxycycline
D. Mefloquine
E. Supportive medications

C. Doxycycline

Doxycycline is typically used in the management of typhus

This lady has a triad of fever, hepatosplenomegaly and encephalitis, which in a returning traveller could point towards a number of diseases including malaria and dengue fever. However, the lesion on her shin is an eschar, which points towards a diagnosis of rickettsia. Given her clinical signs and that she has been to Indonesia, this is likely to be scrub typhus (infection with Orienta Tsutsugamushi). Low platelets, raised LFTs and bilateral infiltrates on chest x-ray support this diagnosis.

First line treatment is with doxycycline for seven days, although a single dose of azithromycin may work in those for whom it is not possible to give doxycycline.

Mefloquine and Chloroquine are used in malaria. Supportive therapy is the recommended treatment in dengue fever.

Question 67 of 277

You are asked to review a patient on the ward who the nurse feels is looking very flushed. The nurse noticed the change in the patient whilst they were receiving their first dose of vancomycin. On examination the patient has no symptoms or signs of cardiorespiratory distress. Observations are as follows:

- Temperature 37.2 ºc
- Respiratory rate 18 breaths/min
- Saturations on air 97%
- Heart rate 70 beats/min
- Blood Pressure 136/72 mmHg

A blanching macular rash is evident on the patient's upper arms and upper thighs. No signs of urticaria or excoriations. The patient is penicillin allergic.

What is your next step?

A. Measure mast cell tryptase levels
B. Stop the infusion, give the patient piriton and hydrocortisone
C. Stop the infusion and give the patient piriton, hydrocortisone, adrenaline
D. Continue the infusion but slow it down and give the patient piriton
E. Stop the infusion, do not give any further treatment but discuss an alternative antibioticwith microbiology

D. Continue the infusion but slow it down and give the patient piriton

The most likely explanation for the patient's condition is a drug reaction called red man syndrome secondary to vancomycin infusion. This is not an allergic reaction as demonstrated by the lack of cardiorespiratory distress, subcutaneous oedema, wheals or urticaria. The reaction is more of a rate dependent infusion reaction. Treatment involves slowing down the infusion of vancomycin and administration of anti-histamines.

Question 68 of 277

A 75-year-old male with a long history of intravenous drug use is admitted with fevers, rigors and back pain. Three sets of blood cultures taken at admission grow positive for gram positive cocci in clusters. He is suspected of having *Staphylococcus aureus* bacteraemia and is commenced on intravenous vancomycin.

Half an hour after the infusion is commenced, he is noted by the nurse to be flushed. On examination, he is noted to have erythema over his neck, face and trunk but denies any significant distress or discomfort.

His observations are as follow: blood pressure 125/70 mmHg, heart rate 85/min, temperature of 36.8ºC, respiratory rate of 18/min and oxygen saturation of 98% on room air.

Which of the following is the most appropriate management?

A. Stopping the vancomycin infusion, administering 200mg of IV hydrocortisone and informing the patient that he is allergic to the medication
B. Stopping the vancomycin infusion and administering a single dose of 0.5mcgintramuscular adrenaline
C. Stopping the vancomycin infusion until symptoms resolve and then re-starting a slowerrate
D. Stopping the vancomycin infusion and prescribing topical 1% hydrocortisone cream toaffected areas
E. Continuing the vancomycin infusion and administering 1000 ml of 0.9% saline solutionover 1 hour

C. Stopping the vancomycin infusion until symptoms resolve and then re-starting a slowerrate

Red man syndrome is associated with rapid intravenous infusion vancomycin. It is a common adverse reaction of intravenous vancomycin use and is a distinct entity from anaphylaxis due to vancomycin use. Typical symptoms include redness, pruritus and a burning sensation, predominantly in the upper body (face, neck and upper chest). Severe cases can be associated with hypotension and chest pain.

The pathophysiology of red man syndrome is attributed to vancomycin-related activation of mast cells with release of histamine.

The management of red man syndrome involves cessation of the infusion, and when symptoms have resolved, recommencement at a slower rate. In patients who are more symptomatic antihistamines can be administered, and may require intravenous fluids if the syndrome is associated with hypotension.

Question 69 of 277

You are the medical basic specialist trainee on call and have been asked to see a gentleman who has just been admitted for a bad chest infection. According to the ward nurse, he has developed an allergic reaction after five minutes of vancomycin i.v infusion.

On arrival you proceed to examine this young man who is febrile, obviously unwell and receiving oxygen via nasal prongs. He claims he had not developed any new symptoms since admission but felt sudden warmth and flushing few minutes into his vancomycin drip. On examination he has crepitations in his left base.

Apart from generalised erythema he appears to be stable and there is no immediate danger to his airways. His blood pressure was 105/67mmHg, pulse was 99b/min and oxygen saturation on 4L oxygen was 95% (all observations are similar to previous readings). He has never taken vancomycin but states he is allergic to penicillin and ciprofloxacin.

Given the likely diagnosis, what is the next step in your management?

A. Immediately stop vancomycin
B. Continue vancomycin but at a slower rate
C. Continue vancomycin rate unchanged
D. Administer i.v chlorphenamine maleate and stop vancomycin
E. Urgent i.m adrenaline

B. Continue vancomycin but at a slower rate

Answer: continue vancomycin but at a slower rate.

This is typical red man syndrome caused by rapid infusion of vancomycin i.v. Vancomycin should be administered diluted and at a rate of 10mg/minutes for doses over 500mg i.v.

It is an IgE independent mast cell degranulation reaction that occurs up to 10 minutes into receiving vancomycin i.v at a fast rate. It is not a true allergic reaction thus vancomycin should be continued but at a lower rate of infusion.

Antihistamine prophylaxis, lower but frequent dosing and 2 hour infusion rate have been found to reduce the incidence of red man syndrome.

This gentleman is obviously unwell but his vitals and chest findings are more in keeping with his chest infection and possible sepsis.

Red-man syndrome after vancomycin: potential cross-reactivity with teicoplanin C Khurana, M A de Belder http://pmj.bmj.com/content/75/879/41.long

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A 36-year-old man is admitted with a painful right arm. On examination, there is mild erythema overlying the forearm but extreme tenderness to palpate. The arm is also tender beyond the area of erythema. Observations are as follows.

- Respiratory rate 32/min
- Oxygen saturation 94% on air
- Heart rate 134/min
- Blood pressure 72/44mmHg
- Temperature 38.4ºC

Over the next few hours, he deteriorates rapidly despite antibiotic therapy. The arm develops bullae and necrotic areas and ultimately requires amputation. His family arrive and ask how he could have developed this illness.

What recent infection could increase the risk of his current condition?

A. Chickenpox
B. Herpes simplex virus encephalitis
C. Influenza
D. Mumps
E. SARS-CoV2

A. Chickenpox

Chickenpox is a risk factor for invasive group A streptococcal soft tissue infections including necrotizing fasciitis

The correct answer is **chickenpox**. This patient has developed necrotising soft tissue infection, most commonly due to infection with group A streptococcus. Recent chickenpox is a recognised risk factor for invasive group A streptococcal (iGAS) infections such as this.

Herpes simplex virus encephalitis is incorrect. This is a severe central nervous system infection which does have complications including stroke and epilepsy. It is not known to increase the risk of iGAS.

Influenza is incorrect. The main bacterial infection seen in the aftermath of influenza is *Staphylococcus aureus* pneumonia.

Mumps is incorrect. It does have a range of complications including encephalitis and pancreatitis, but an increased risk of iGAS is not recognised.

SARS-CoV2 is incorrect. Infection with this virus can increase the risk of secondary bacterial respiratory tract infections. Some treatments also increase the risk of infections - for example, some areas saw increased rates of invasive fungal infections like mucormycosis related to highdose corticosteroid use. An association with iGAS, however, has not been described.

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A 27 week pregnant woman attends her GP with a 12 hour history of a rash. On examination, she has lesions consistent with a diagnosis of chickenpox. Her clinical observations are stable and she is otherwise well.

What would be the recommended management?

A. No treatment needed
B. Symptomatic treatment only (antihistamine & calamine lotion)
C. Oral aciclovir
D. Varicella-zoster immunoglobulin (VZIG)
E. Oral aciclovir & VZIG

C. Oral aciclovir

Pregnant women ≥ 20 weeks who develop chickenpox are generally treated with oral aciclovir if they present within 24 hours of the rash

The development of chickenpox in a pregnant woman is associated with increased morbidity, including pneumonia, hepatitis and encephalitis. Therefore, pregnant women who develop a chickenpox rash should see their GP immediately.

Oral aciclovir should be prescribed in pregnant women with chickenpox, if they present within 24 hours of the onset of the rash and are 20 weeks gestation or beyond. (Use before 20 weeks can be considered).

Aciclovir reduces the duration of fever and symptomatology of varicella, if commenced within 24 hours of developing the rash. Symptomatic treatment can be used alongside aciclovir.

Pregnant women with severe chickenpox should be referred to hospital for intravenous aciclovir. In addition, a referral to fetal medicine would be necessary (in the above scenario), due to the small risk of fetal varicella syndrome in the first 28 weeks of pregnancy.

It is important to advise anyone with chickenpox to avoid contact with other potentially susceptible people, such a pregnant women and neonates until the last lesions have crusted over (usually 5 days after the onset of the rash).

VZIG has no therapeutic benefit once the rash has started. RCOG Greentop guidelines, Chickepox in pregnancy.

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A 20-year-old law student had been backpacking around India for 2 months over her summer holiday. She spent time in a number of rural and coastal areas and volunteered at a wildlife conservation project for 2 weeks. She took malaria prophylaxis throughout.

A few days before her return to the UK she noticed a small nodule on her left forearm. The nodule has gradually enlarged and ulcerated over the last 4 weeks to form a 4cm painless ulcer.

What is the most appropriate investigation to confirm the diagnosis?

A. Biopsy of a draining lymph node
B. Montenegro skin test
C. Punch biopsy of the lesion for histology
D. Serology
E. Serum PCR

C. Punch biopsy of the lesion for histology

Cutaneous leishmaniasis is typically diagnosed using a punch biopsy

This patient has cutaneous leishmaniasis. This is a protozoal infection mainly caused by *Leishmania major*, *Leishmania tropica* and *Leishmania aethiopica* in Asia, Africa, the Middle East and the Mediterranean, and by *Leishmania braziliensis* and *Leishmania mexicana* in Central and South America. It presents classically with a slowly developing painless ulcer at the site of a sandfly bite.

The most appropriate investigation to confirm the diagnosis is **punch biopsy of the lesion for histology**. The biopsy should be taken from the outer border of the ulcer and should be full thickness. The specimen can be examined using Wright or Giemsa stains to look for amastigotes in the tissue. Culture or PCR can also be performed on the tissue sample.

Biopsy of a draining lymph node is incorrect. You are unlikely to see parasites in tissue samples outside of the ulcer itself in cutaneous leishmaniasis, as the infection is limited to the ulcer only.

Montenegro skin test is incorrect. This is a test in which killed promastigotes are injected under the skin, and the degree of induration at the site is read 48-72 hours later. It is used in some South American countries but is not recommended as the sensitivity is low and it cannot differentiate between past and current infections.

Serology is incorrect. This is not used in diagnosing cutaneous leishmaniasis because antibody levels are generally low, meaning that the sensitivity of the test is low. The specificity is also variable and there may be cross-reactivity with other infections.

Serum PCR is incorrect. PCR can be carried out on tissue samples from the lesion but is not carried out on serum as there would be minimal or no protozoal DNA in the circulating blood, so the sensitivity would be too low.

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A 43-year-old man presents to the emergency department with a fever, rash, and headache. He has recently returned from travelling around the Solomon Islands. He has no past medical history of note.

On examination, he has a diffuse morbilliform rash and a dark, scab-like lesion is noted on his leg. His temperature is 38.4ºC.

Given the likely diagnosis, what is the most appropriate management?

A. Atovaquone-proguanil
B. Ceftriaxone
C. Ciprofloxacin
D. Doxycycline
E. Supportive management

D. Doxycycline

Doxycycline is typically used in the management of typhus

Doxycycline is correct. The patient presents with a fever, headache, and rash. These are common features of many illnesses in the returning traveller. However, the dark scab-like lesion is more discriminating and in the context of a patient returning from the Solomon Islands, this likely represents the black eschar associated with scrub typhus infection. The Solomon Islands from the southern tip of the 'Tsutsugamushi Triangle' where scrub typhus is endemic. The treatment is with doxycycline.

Atovaquone-proguanil is incorrect. Also known as Malarone, this is an accepted oral treatment for uncomplicated malaria. Malaria is present in the Solomon Islands but a rash is actually uncommon in malaria and a black eschar is not a feature.

Ceftriaxone is incorrect. This would be an appropriate treatment for typhoid, which is a common cause of fever in returning travellers. A black eschar is not a feature of typhoid infection.

Ciprofloxacin is incorrect. This would be an appropriate treatment if we suspected anthrax. This is another cause of eschar. However, while a patient with anthrax may be systemically unwell, a diffuse morbilliform rash is not typical. Additionally, the eschar of anthrax may be associated with some perilesional oedema and blister which are not mentioned here.

Supportive management is incorrect. The mortality rate of scrub typhus is 6% untreated. Management is with doxycycline.

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A 46-year-old man telephones his GP for advice. Five days ago, he was in contact with his nephew, who was unwell and had a rash at the time. It has since been confirmed that his nephew has measles. The patient is concerned as he was not immunised against measles as a child. Furthermore, he takes methotrexate for rheumatoid arthritis.

He has no symptoms currently.

What is the most appropriate treatment?

A. Immunoglobulin
B. Measles, mumps and rubella (MMR) vaccine
C. Measles, mumps and rubella (MMR) vaccine and immunoglobulin
D. Measles-only vaccine
E. No treatment

A. Immunoglobulin

Those exposed to measles can be given immunoglobulin to reduce the risk of infection

The most appropriate course of action, for a patient who is immunosuppressed and does not have adequate immunity to measles, is to administer **immunoglobulin** following exposure. Ideally, this should be done within 72 hours, but can be done up until 6 days post-exposure.

For patients with normal immunity, who have not been fully immunised against measles, the **MMR vaccine** should be administered within 72 hours of exposure.

Administering both the **MMR vaccine and immunoglobulin** is inappropriate. This can invalidate the effect of the vaccine.

Giving the **measles-only vaccine** would also be inappropriate. This patient should be given immunoglobulin. For a patient with normal immunity, who is not fully immunised, the MMR vaccine should be given rather than a measles-only vaccine.

No treatment is incorrect. Given this patient's clear exposure and history of both immunosuppression and lack of immunisation against measles, immunoglobulin should be given.

Question 75 of 277

A 35-year-old man is referred to a tuberculosis (TB) clinic after his wife is confirmed to have fully sensitive pulmonary TB. There is no history of cough, fevers or weight loss. He has no medical problems, does not take any medication regularly and does not smoke or drink alcohol. His blood tests show:

HIV antibody test	negative
Hepatitis B surface antigen test	negative
Hepatitis B surface antibody test	positive
Hepatitis C antibody test	negative
Interferon-gamma release antibody	positive

What is the most appropriate treatment to offer initially?

A. 2 months of rifampicin, isoniazid, pyrazinamide, ethambutol and pyridoxine
B. 2 months of tenofovir
C. 3 months of rifampicin, isoniazid and pyridoxine
D. 48 weeks of peginterferon alfa-2a
E. 6 months of rifampicin only

C. 3 months of rifampicin, isoniazid and pyridoxine

Latent tuberculosis treatment options:

- 3 months of isoniazid (with pyridoxine) and rifampicin, or
- 6 months of isoniazid (with pyridoxine)

This patient has latent TB as evidenced by a positive interferon-gamma release antibody (IGRA) test and no other symptoms consistent with active TB. NICE advises that treatment for latent TB should be with either **3 months of rifampicin, isoniazid and pyridoxine** or 6 months of isoniazid and pyridoxine.

This patient has either been immunised against Hepatitis B or previously infected and therefore has a positive Hepatitis B surface antibody test but a negative antigen test. If they had chronic hepatitis B they would have a positive antigen test. Treatment of chronic hepatitis B includes onceweekly injections of **peginterferon alfa-2a, a recombinant interferon, for 48 weeks** or antivirals such as **tenofovir** or entecavir.

If this patient had a positive IGRA test and symptoms consistent with active TB, they would require treatment for active TB with **2 months of rifampicin, isoniazid, pyrazinamide, ethambutol and pyridoxine** followed by 4 months of rifampicin, isoniazid and pyridoxine.

6 months of rifampicin only can be used to treat patients with latent TB but is not recommended by NICE guidelines. It could also be used if this patient's wife had isoniazid-resistant pulmonary TB.

Question 76 of 277

A 38-year-old man presents to the emergency department with fever and shivering, occurring approximately every two days. He has returned from India three weeks ago, having spent four months travelling. While he was in India, he was treated for malaria with chloroquine. He has no other past medical history and does not take any regular medications.

Observations:

- Heart rate 88 beats/min
- Blood pressure 120/77 mmHg
- Respiratory rate 18 breaths/min
- Oxygen saturations 98% on room air T
- emperature 38.6ºC

On examination, the patient is diaphoretic. There is palpable splenomegaly. A thick and thin blood film confirms malaria.

What organism is most likely to be responsible?

A. Plasmodium falciparum
B. Plasmodium knowlesi
C. Plasmodium malariae
D. Plasmodium ovale
E. Plasmodium vivax

E. Plasmodium vivax

P. vivax and *ovale* have hypnozoite forms, which can cause clinical disease long after patients

leave the endemic area or may cause relapsing disease following treatment

Plasmodium vivax is the correct answer. The patient has a confirmed diagnosis of malaria, having previously been treated for malaria within the last six months. This suggests a relapse of disease. The two forms of malaria that cause relapsing disease are Plasmodium vivax and Plasmodium ovale. Plasmodium vivax is more common in India while Plasmodium ovale is more common in Africa. Chloroquine is the typical treatment in most countries. This medication acts on blood-stage parasites only and does not act on hypnozoites. The only available drug that can kill the hypnozoites is primaquine.

Plasmodium falciparum is incorrect. This type of malaria does not cause relapsing disease.

Plasmodium knowlesi is incorrect. This form of malaria, which is common in South-East Asia does not cause relapsing disease.

Plasmodium malariae is incorrect. This does not cause relapsing disease and is a less common form of malaria. Additionally, it causes fever every three days rather than every two days as in other forms of malaria.

Plasmodium ovale is incorrect. This can also cause relapsing disease. However, it does so far less commonly than *vivax* malaria and is more common in Africa rather than Asia.

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A 42-year-old woman visits the emergency department with a 4-day history of high fevers and myalgia. She has just landed in the UK from the Democratic Republic of the Congo. Her past medical history includes uterine fibroids and iron deficiency anaemia for which she takes regular ferrous sulfate.

Her observations are as follows:

- Temperature 39.1ºC
- Heart rate 122bpm
- Blood pressure 105/80mmHg
- Respiratory rate 15 breaths/min
- Oxygen saturations 99% on air

On examination, she is clammy to touch and complains of a headache. Her chest sounds are clear and her heart sounds are normal. She has a soft and non-tender abdomen. On closer examination, there are multiple enlarged and tender lymph nodes in her axillae and groin.

What is the most likely causative organism?

A. Burkholderia pseudomallei
B. Mycobacterium leprae
C. Treponema pallidum pertenue
D. Yersinia pestis
E. Zaire ebolavirus

D. Yersinia pestis

The plague is caused by the gram-negative bacterium *Yersinia pestis*. Three clinical presentations can occur: bubonic (most common), pneumonic and septicaemic

This patient has a diagnosis of the bubonic plague, caused by the gram-negative bacterium ***Yersinia pestis***. It is transmitted by fleas and is endemic to the Democratic Republic of the Congo, Madagascar, and Peru. Symptoms typically include flu-like symptoms from 3-7 days post-exposure followed by the eruption of inflamed, tense and painful lymph nodes. The disease carries a significantly high mortality rate if not treated and prompt recognition and antibiotic administration (i.e. Streptomycin) can reduce the mortality from 60% to 15%.

Melioidosis is a bacterial illness caused by ***Burkholderia pseudomallei***. It is a tropical disease seen in South-East Asia, Africa, the Middle East and the South Pacific. Some may present with a localised skin infection from exposure to the bacterium as it enters the skin (e.g. nodule or ulcer formation) whereas other patient's progress to septicaemia and abscess formation. Melioidosis is not associated with inflamed and painful lymph nodes making it an unlikely diagnosis.

Leprosy is a granulomatous disease caused by ***Mycobacterium leprae*** which affects the peripheral nerves and skin causing symptoms such as loss of temperature and light touch followed by loss of pain sensation. Associated cutaneous lesions follow later in the disease and appear as hypopigmented patches of skin. These symptoms are not seen in the above patient and leprosy is not associated with painful lymphadenopathy.

Yaws is a chronic skin infection caused by the bacterium ***Treponema pallidum pertenue***. There are several stages of illness with the disease initially presenting as a papule from the site of infection that later ulcerates. As the disease progresses, more widespread papules appear over the rest of the body. These lesions are not seen with this patient, making Yaws incorrect.

Ebola virus disease is an extremely deadly disease caused by multiple different viruses including **Zaire ebolavirus**. The disease causes flu-like symptoms including fever, myalgia and sore throat along with gastrointestinal symptoms such as abdominal pain and vomiting. Zaire ebolavirus causes a haemorrhagic illness and as the disease progresses, patients develop unexplained bleeding and bruising. Although this patient has flown from a high-risk area for the Ebola virus, she has no gastrointestinal symptoms or haemorrhagic symptoms.

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A 38 year-old woman presented to the medical outpatient clinic with an 8 month history of deteriorating vision. She also complained of a pruritic rash intermittently affecting her forearms and neck. She had recently migrated to the UK from Guinea, where she had lived since birth. She had suffered from malaria as a child but had been fit and well since. She did not smoke and drank 6-10 units of alcohol per week.

On examination, her temperature was 36.7ºC, pulse was 68 beats per minute and blood pressure was 124/80 mmHg. Her chest was clear on auscultation and heart sounds were normal. Fundoscopy was not possible due to clouding of both corneas. There was evidence of a mottled rash over the forearms and neck with a leopard print appearance.

Investigations:

Haemoglobin	141 g/L (130-180)
White cell count	6.7 x /L (4.0-11.0)$_{9}$
Neutrophil count	3.0 x /L (2.0-7.5)$_{9}$
Lymphocyte count	1.7 x /L (1.3-3.5)$_{9}$
Eosinophil count	0.6 X /L (0.1-0.4)$_{9}$
Platelets	260 x /L (150-400)$_{9}$
Sodium	138 mmol/L (135-145)
Potassium	4.3 mmol/L (3.5-5.0)
Urea	7.2 mmol/L (2.5-7.5)
Creatinine	61 mol/L (25-95)
Fasting plasma glucose	5.0 mmol/L (3.0-6.0)
Giemsa-stained blood film	No abnormality detected

Given the likely diagnosis, what is the most appropriate treatment?

A. Diethylcarbamazine (DEC)
B. Ivermectin
C. Benzinidazole
D. Albendazole
E. Praziquantel

B. Ivermectin

This patient has presented with features of onchocerciasis, a disease caused by the filarial nematode Onchocerca volvulus. The parasite is transmitted by blackflies of the genus Simulium, which deposit the parasites larva onto the skin when biting to extract blood. The adult worm lives in subcutaneous nodules and produces larvae, which become deposited in the skin and eyes. Disease manifests when the parasite dies in tissues and releases Wolbachia endosymbiotic bacteria into tissues, provoking a host immune response. This above patients disease has manifested as river blindness, where the worms die in the cornea and provoke chronic inflammation, eventually leading to clouding of the cornea. Inflammation in subcutaneous tissues leads to a pruritic rash and formation of a leopard-skin appearance over time. The worms can be visualised by microscopy of skin snip biopsy. The treatment of choice is ivermectin; DEC should not be used because rapid death of the worms can exacerbate damage to surround tissues and even cause complete blindness.

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A 29-year-old lady comes to see you for advice having seen news of the recent Zika virus outbreak. She and her husband are planning on starting a family, but she has only arrived back from Brazil last week after a business trip. She has not experienced any fever or worrying symptoms, either during travel or since arrival back in the UK.

What would be the most appropriate advice to give her?

A. Start 5mg folic acid daily
B. If no fever can become pregnant
C. Need to check Zika virus serology before becoming pregnant
D. Should avoid becoming pregnant for at least 2 weeks after travel
E. Should avoid becoming pregnant for at least 8 weeks after travel

E. Should avoid becoming pregnant for at least 8 weeks after travel

The latest advice as per Public Health England is that women who are considering becoming pregnant should avoid becoming pregnant while travelling in an area with active Zika virus transmission, and for 8 weeks after their return.

Whilst folic acid is recommended to women trying for pregnancy, the usual dose is 400 micrograms daily and there is currently no recommendation to increase this after Zika exposure. The test for Zika serology is only recommended for patients with a relevant travel history and current symptoms.

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A 27-year-old man who recently immigrated to the UK from Ethiopia complains of a chronic cough with night sweats for five weeks. On direct questioning, he also admits coughing up small volumes of bright red blood a few times. This has made him especially concerned as he his father died from lung related problems following chronic coughs.

Following a chest X-ray and blood tests he is diagnosed with TB and contact tracing is started. His 25-year-old male partner lives with him and is identified as being at high risk of contracting TB. The partner undergoes Mantoux testing and has a 2mm area of induration. He has never had BCG vaccination as far as he is aware and has no vaccination scar.

What is the most appropriate management that should be offered to the partner?

A. BCG vaccination
B. HIV testing and if negative then BCG vaccination
C. Prophylactic anti-TB treatment
D. Repeat TB screening in six weeks
E. No further management needed

B. HIV testing and if negative then BCG vaccination

The correct answer is HIV testing and if negative then BCG vaccination. The low area of response to the Mantoux testing indicates that he is unlikely to have TB and has not been vaccinated. However, the test can be a false negative result in an immunosuppressed patient. As the partner is at increased risk of HIV, NICE advises that an HIV test should be done prior to vaccination. BCG vaccination is a live vaccination and therefore is contraindicated in an immunosuppressed patient. Of note, previous vaccination can lead to a false positive result.

NICE advises that once a diagnosis of pulmonary TB has been made then close contacts should be managed as follow:

- If asymptomatic and younger than 65 years then test for latent TB. If Mantoux-negative and unvaccinated then offer vaccination. If at risk of HIV then test for HIV first.
- If asymptomatic and older than 65 years then assess with a chest X-ray.

Treatment for TB should be considered if TB is confirmed and not used prophylactically. Repeat screening is generally not used.

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A 35-year-old man is referred to the medical assessment unit by his general practitioner. He reports two months of episodic swellings of the soft tissue near various joints that are hot and occasionally pruritic. He has no past medical history and is not on any regular medications. He has recently returned from spending six months in Equatorial Guinea.

On examination, there is a large, 2 x 3cm non-pitting swelling of his dorsal forearm that is hot but not erythematous.

Blood tests:

Hb	136 g/L	Male: (135-180)Female: (115 - 160)
Platelets	189 * 10 /L 9	(150 - 400)
WBC	14.2 * 10 /L 9	(4.0 - 11.0)
Neuts	6.5 * 10 /L 9	(2.0 - 7.0)
Lymphs	2.2 * 10 /L 9	(1.0 - 3.5)
Mono	0.3 * 10 /L 9	(0.2 - 0.8)
Eosin	5.2 * 10 /L 9	(0.0 - 0.4)

What is the likely diagnosis?

A. Cutaneous larva migrans
B. Cutaneous leishmaniasis
C. Loiasis
D. Schistosomiasis
E. Syphilis

C. Loiasis

Calabar swellings: transient, non-erythematous, hot swelling of soft-tissue around joints → loiasis

Loiasis is the correct answer. The patient presents with transient, non-erythematous, hot swellings of the soft tissues around joints. This is a description of so-called Calabar swellings, which are associated with loiasis. This condition is a filarial infection caused by the nematode Loa Loa. The Calabar swellings are caused by an immune reaction to the worms. Eosinophilia is common in this condition and it is endemic to Equatorial Guinea.

Cutaneous larva migrans is incorrect. This can cause skin lesions and eosinophilia. However, the lesions in this condition (which is caused by a hookworm infection) are classically serpiginous in outline and therefore a 2x3cm lesion would not be characteristic.

Cutaneous leishmaniasis is incorrect. This condition is transmitted by sandflies. It characteristically causes an ulcerative lesion, which is not present in this case as there is no mention of skin erosion. Additionally, the lesions tend not to be transient and persist without treatment.

Schistosomiasis is incorrect. An acute schistosomiasis infection is typically characterized by fever, blotchy rash, myalgia, arthralgia and diarrhoea. So while it is endemic in Equatorial Guinea and associated with blood eosinophilia, the presentation is not characteristic of the disease.

Syphilis is incorrect. This can cause a skin lesion called a chancre, which is a painless ulcer. However, this typically occurs on the genitals and occurs once rather than episodically. Additionally, eosinophilia is not typical of this condition.

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A 25-year-old man is seen in the walk-in travellers clinic. He returned from a holiday in Brazil 5 days ago and has a 1 day history of fever, headache and myalgia. He is usually fit and well. He tells you he took regular malaria prophylaxis while away.

On examination he has a heart rate of 110 beats per minute and a blood pressure of 102/72 mmHg. His temperature is 38.1 ºC and he has dry mucous membranes. Examination of cardiovascular, respiratory and gastrointestinal systems is unremarkable but he has multiple mosquito bites over his arms and legs.

His blood tests are as follows:

Hb	132 g/l	Na +	144 mmol/l	Bilirubin	8 µmol/l
Platelets	91 * 10 /l 9	K +	3.5 mmol/l	ALP	98 u/l
WBC	13 * 10 /l 9	Urea	9.7 mmol/l	ALT	22 u/l
Neuts	11 * 10 /l 9	Creatinine	111 µmol/l	γGT	14 u/l
Lymphs	0.6 * 10 /l 9	CRP	78 mg/l	Albumin	40 g/l

He is admitted for broad spectrum antibiotics, fluids and analgesia but his fever continues. His malaria films are negative but he has a positive PCR for dengue virus. Antibiotics are stopped but fluids are continued. His fever and headache settle though he does developed some mild ankle oedema.

Which of the following is true regarding his discharge?

A. Dengue shock is unlikely to occur after 24 hours post-fever
B. Dengue shock is unlikely to occur once platelets improving
C. Dengue shock is unlikely to occur once renal function improving
D. Platelets above 20 * 109/l are considered safe for discharge
E. Stable haematocrit is essential for discharge

E. Stable haematocrit is essential for discharge

This gentleman is admitted in the febrile phase of his dengue infection. The plasma leak phase may occur anytime within the first 48 hours after the fever has broken and manifests with leakage into pleural and peritoneal spaces. It may be accompanied by shock and in some cases haemorrhage. Platelets and renal function may be falsely reassuring as they tend to improve after the febrile phase but rising haematocrit is a sensitive sign of plasma leak. It is therefore essential to monitor patients for a full 48 hours after the febrile phase and ensure haematocrit is stable prior to discharge. Platelets should be at least 50 * 109/l and rising prior to discharge.

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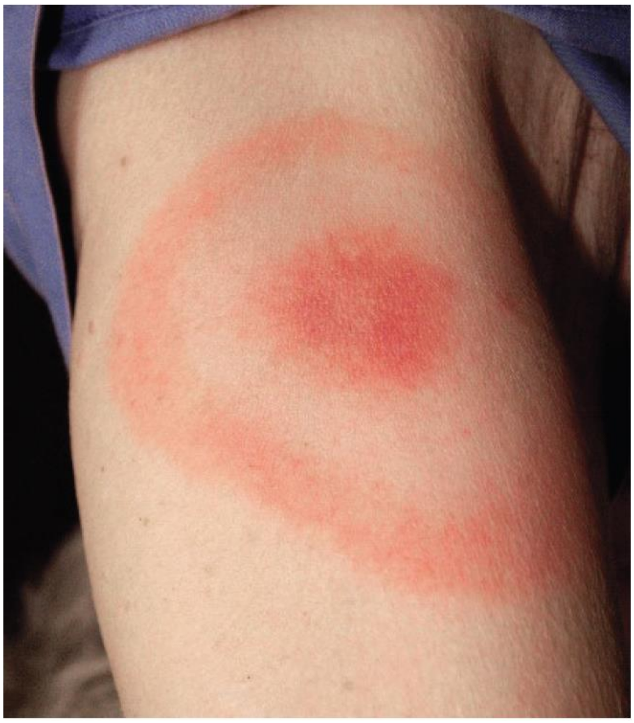
A 28-year-old woman presents to the dermatology clinic with a rash on her right upper arm. She says it has been enlarging over the past 2 weeks. There is no history of pain or itch. Her past medical history includes mild gastritis and she is 14-weeks pregnant. She has no known allergies.

On examination, her observations are within normal limits. Her rash is examined, as shown below:



What is the recommended management for this patient?

A. Amoxicillin
B. Clotrimazole
C. Doxycycline
D. Phenoxymethylpenicillin
E. Prednisolone

A. Amoxicillin

This patient's rash is typical of erythema migrans given the 'bulls-eye' appearance of a circular erythematous centre with a clear border and outer erythematous border. Erythema migrans is typically associated with Lyme disease, a zoonotic infection secondary to the spirochete *Borellia burgdorferi*. It is spread by the bites of infected ticks. Associated symptoms include fever, myalgia and fatigue. The rash can appear 1-2 weeks following a tick bite and can enlarge in size over time with central clearing. The lesions are not typically painful or itchy.

Amoxicillin is correct. The typical first-line treatment for Lyme disease is doxycycline. However, as this patient is pregnant, doxycycline is contraindicated and amoxicillin is the next best treatment of choice.

Clotrimazole is incorrect. This is used in the treatment of fungal infections including tinea corporis. Tinea corporis can present similarly with a circular lesion and an erythematous outer border.

However, the lesions with ringworm are usually itchy with a scaly outer edge which is not seen here.

Doxycycline is incorrect. This is the first-line management for lyme disease. However, doxycycline is contraindicated in pregnancy due to the risk of bone and teeth deformity. Therefore, the most appropriate option would be amoxicillin.

Phenoxymethylpenicillin is incorrect. This is typically used in the management of infections caused secondary to Streptococcal species (e.g. *Streptococcus pyogenes*). Group A Streptococci such as *Streptococcus pyogenes* have the potential to cause infections such as tonsillitis and rheumatic fever. Rheumatic fever is associated with erythema marginatum, an annular erythematous rash that appears on the trunk, upper arms and legs that spread outwards in a circular shape with central clearing. However, this is very rare in adults and in developed countries.

Prednisolone is incorrect. Corticosteroids can be used for several inflammatory skin conditions including eczema and psoriasis. However, these lesions are often multiple, exfoliative, scaly and itchy which is not seen in this patient's presentation. Therefore, an alternative diagnosis should be considered.

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A 45-year-old female presents with a rash. She states that she initially felt feverish and had a runny nose. This progressed to a rash over her arms, trunk and legs. She recently immigrated to the UK from Poland. She lived in a travellers community in Poland and is not aware of any childhood vaccinations. She has a past medical history of asthma and hereditary spherocytosis. On examination you note a erythematous maculopapular rash over the the upper limbs, thorax and lower limbs.

The following investigations were performed:

Hb	95 g/l	Na +	138 mmol/l
Platelets	110 * 10 /l 9	K +	3.8 mmol/l
WBC	3.8 * 10 /l 9	Urea	6.5 mmol/l
Neuts	1.8 * 10 /l 9	Creatinine	58 µmol/l
Lymphs	1.6 * 10 /l 9	CRP	48 mg/l

What is the most likely diagnosis?

A. Measles
B. Mumps
C. Rubella
D. Parvovirus B19
E. Scabies

D. Parvovirus B19

Parvovirus B19 can cause fever, rash and patients with predisposing haematological conditions, pancytopenia

The patient has presented with a febrile rash. The differential is wide. The immunisation history should alert you to possible measles or rubella. However the presence of pancytopenia favours a diagnosis of parvovirus B19 infection which is associated with an aplastic crisis.

Although most patients have a decrease of hematopoiesis during parvovirus infection, it is most dangerous in patients with pre-existing bone marrow stress (e.g. sickle cell anaemia or hereditary spherocytosis) as these disorders are heavily dependent on erythropoiesis due to the reduced lifespan of the red cells.

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A 42-year-old man presents with a fever and shortness of breath. Of note, he is a cattle farmer. Observations are as follows: heart rate 82 beats per minute, blood pressure 130/85 mmHg, temperature 38.2ºC, respiratory rate 20 breaths per minute, and SpO2 95% on air.

Blood results are as follows:

Hb	136 g/L	Male: (135-180)Female: (115 - 160)
Platelets	162 * 10 /L 9	(150 - 400)
WBC	14.4 * 10 /L 9	(4.0 - 11.0)
Na +	136 mmol/L	(135 - 145)
K +	3.8 mmol/L	(3.5 - 5.0)
Urea	4.2 mmol/L	(2.0 - 7.0)
Creatinine	66 µmol/L	(55 - 120)
CRP	44 mg/L	(< 5)
Bilirubin	16 µmol/L	(3 - 17)
ALP	104 u/L	(30 - 100)
ALT	146 u/L	(3 - 40)
Î³GT	68 u/L	(8 - 60)
Albumin	36 g/L	(35 - 50)

A chest x-ray is as follows:

Chest x-ray	Perihilar patchy reticulonodular opacities

What treatment is indicated?

A. Amoxicillin
B. Clarithromycin
C. Co-amoxiclav
D. Doxycycline
E. Levofloxacin

D. Doxycycline

Doxycycline should be used to treat Q fever

Doxycycline is correct. The chest x-ray appearances are suggestive of atypical pneumonia. Atypical pneumonia associated with fever, hepatitis, and exposure to cattle is highly suggestive of Q fever. Doxycycline is the antibiotic of choice for Q fever.

Amoxicillin is incorrect. Penicillins are ineffective against Q fever since the bacteria is intracellular.

Clarithromycin is incorrect. Whilst macrolides have activity against some intracellular bacteria, they are not considered the first-line treatment choice for Q fever due to a limited evidence base.

Co-amoxiclav is incorrect. Penicillins are ineffective against Q fever since the bacteria is intracellular.

Levofloxacin is incorrect. Q fever can be treated with fluoroquinolones however these agents are not first-line since they have a much broader spectrum.

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A 55-year-old man was admitted with a fever, pain, and erythema across his abdomen. He had been discharged 8 days prior following an elective repair of an umbilical hernia. He has a background of type 2 diabetes mellitus and a raised body mass index of 32kg/m² (normal range 18.5 - 24.9kg/m²).

He had been recovering from his operation at home and had felt well up until that morning. He had noticed a small area of erythema and tenderness around the incision site near his umbilicus. This area of erythema seemed to grow and become more painful throughout the day, therefore, he was brought to the Emergency Department by his wife.

On examination, he looked unwell. His observations were as follows:

- temperature 38.6ºC
- respiratory rate 23 breaths/min
- oxygen saturation 97% on air
- heart rate 125bpm
- blood pressure 88/61mmHg

He felt nauseous and had vomited once, complaining of severe abdominal pain. There was a large area of erythema across the lower half of his abdomen which was extremely tender to palpation. Examination of the erythematous area also revealed impaired sensation to light touch.

His blood results:

WBC	17 * 10 /L 9	(4.0 - 11.0)
CRP	460 mg/L	(< 5)

What is the most likely cause of this patient's infection?

A. Polymicrobial
B. Streptococcus pyogenes
C. Aeromonas hydrophila
D. Staphylococcus epidermidis
E. Bacillus anthracis

A. Polymicrobial

Type 1, or polymicrobial, necrotising fasciitis is the most common

This patient is systemically unwell with a rapidly progressive soft tissue infection. Diabetes mellitus is a risk factor for necrotising fasciitis and his recent operation served as a cutaneous portal of entry for causative organisms. A rapidly expanding area of cellulitis in a systemically unwell patient should alert you to the possible diagnosis of necrotising fasciitis. Furthermore, the presence of anaesthesia of the area and pain out of keeping with the visible skin changes is also characteristic of this condition. Whilst necrotising fasciitis is often seen in the extremities, it can also occur in the perineum, head, neck, and trunk.

The most common cause of necrotising fasciitis is type 1, caused by polymicrobial infection of subcutaneous tissue (organisms present include *Bacteroides*, *Peptostreptococcus*, *E. coli*, *Enterobacter*, etc). Bacteria are introduced into the skin via minor skin trauma or surgery and track along fascial planes, extending beyond the area of overlying cellulitis.

Type 2 necrotising fasciitis is a monomicrobial infection most commonly caused by *Streptococcus pyogenes*.

Aeromonas hydrophila is a less common cause of necrotising fasciitis, typically associated with freshwater exposure.

Staphylococcus epidermidis is a commensal organism present on the skin and the most common cause of infections from indwelling medical devices.

Bacillus anthracis, a gram-positive bacteria, causes cutaneous anthrax. There is typically a history of exposure to animals/animal products or IV drug use. A papule forms following exposure which develops into a black eschar with surrounding oedema.

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A 48-year-old man presented to the emergency department with a headache, confusion, and slurred speech. His past medical history, family history, and medication history were unremarkable. He had returned to the UK three weeks ago from Germany where he had been camping in a forest with his family. On the last day of his trip, he noticed a tick on his right leg which he carefully removed with tweezers. One week later, he began to develop fevers, muscle aches, and fatigue so was prescribed doxycycline by his GP. After about 7 days these symptoms resolved but over the past 24 hours, he began to feel worse. His family is in good health and he denied any unwell contacts.

On examination, he was confused with a score of 14/15 on the Glasgow Coma Scale and a noticeable tremor. He was globally weak and looked lethargic. His observations were as follows:

- temperature 38.5ºC
- respiratory rate 19/min
- heart rate 92bpm
- blood pressure 128/84mmHg
- oxygen saturations 98% on room air

Examination of his chest and abdomen was unremarkable and no rashes could be seen. A CT head scan was unremarkable.

A lumbar puncture was performed and the patient was started on empirical treatment with IV aciclovir and ceftriaxone. The CSF results were as follows:

Polynuclear WBCs	3 cells/µL	(0-5 cells/µL)
Mononuclear WBCs	71 cells/µL	(0-5 cells/µL)
Red blood cells	8 cells/µL	(0-10 cells)
Glucose	4.5 mmol/L	(>70% serum glucose)
Protein	0.89 g/L	(0.15-0.45 g/L)
Cytomegalovirus CSF PCR	negative	
Enterovirus CSF PCR	negative	
Epstein-barr virus CSF PCR	negative	
Herpes simplex virus 1 and 2 CSF PCR	negative	
Varicella zoster virus CSF PCR	negative	

Blood test results were as follows:

Serum glucose	5.1 mmol/L	(4 - 11.1 mmol/L)

Borrelia burgdorferi IgG	negative
Borrelia burgdorferi IgM	negative
Treponema pallidum screening serology	negative
Tuberculosis IGRA	negative
HIV screening serology	negative

What is the most appropriate management of this patient given the likely diagnosis?

A. A further course of doxycycline
B. Continue aciclovir
C. Supportive management
D. IV immunoglobulin
E. Praziquantel

C. Supportive management

investigations for HSV-1 and HSV-2 and

Treatment for tick-borne encephalitis is supportive

This patient presents with a history typical of tick-borne encephalitis. Tick-borne encephalitis (TBE) presents as meningoencephalitis, caused by a flavivirus infection of the central nervous system. This virus is transmitted by the *Ixodes* tick, the activity of which is usually highest in the summer months. The disease is endemic in Central and Eastern Europe and the Far East (Russia, China, northern Japan). The disease starts 4-28 days after being bitten by the infected tick and presents in a biphasic fashion. After a few days of fever, fatigue, and myalgia, the patient will have a symptomfree interval before progressing to central nervous system dysfunction (meningitis/encephalitis/cranial nerve palsies).

Diagnosis is generally by CSF/serum serology for TBE-specific antibody and management is supportive - aciclovir is given initially as an empirical treatment but can be stopped when HSV/VZV is ruled out.

In this case, whilst the specific serology confirming the infection is not given, the other investigations and history of a tick bite point towards this diagnosis. He has a predominantly lymphocytic pleocytosis with a raised CSF protein which is typical for viral CNS infections.

Whilst doxycycline appeared to help this patient initially, this coincided with the symptom-free interval commonly seen in the biphasic illness of TBE. Negative serology for *Borrelia burgdorferi* makes a diagnosis of Lyme disease very unlikely. Therefore, another course of doxycycline is not appropriate.

Continuing aciclovir is not necessary given that the CSF VZV are negative.

IV immunoglobulin can be used for autoimmune/paraneoplastic encephalitis but there is nothing in the stem to suggest that this is the underlying pathological process.

Praziquantel is a drug used in the treatment of parasitic infections such as schistosomiasis.

The management of TBE is supportive. Whilst it would be important to identify the cause by performing further serological testing, this is not an option.

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A 55-year-old woman is referred for an urgent assessment by the infectious diseases team after presenting to her local out of hours GP. The patient stated that she was concerned about having caught Lyme disease during a recent day out in the New Forest in Hampshire, UK. The patient was particularly concerned about this diagnosis as she had a close friend who had been forced to give up work after suffering from neuroborreliosis.

On closer questioning, the patient reported that 2 days previously she had gone on a walk in the New Forest. When she had arrived home that evening she had found two engorged ticks attached to her right lower leg. Her husband had removed the ticks by applying nail polish to them and then pulling them off with tweezers.

The patient denied suffering from any symptoms at the time of presentation though admitted she was very anxious. In particular, the patient had not experienced any neurological or cardiovascular symptoms and had not observed any skin rashes.

The patient was receiving treatment from her GP for hypercholesterolaemia and mild asthma. The patient had no known drug allergies and was employed as a legal secretary at a local firm.

A full examination of the patient's skin was performed and no abnormal rashes or other skin changes were identified. No focal neurology was identified on examination of the peripheral and central nervous systems. The assessment did not identify any signs of meningism. The patient was afebrile.

What is the appropriate management of this patient's tick bites?

A. Treat with amoxicillin for 14 days
B. Immediate serological testing for pathogenic Borrelia species
C. Serological testing for pathogenic Borrelia species in 4 weeks (to allow for seroconversion)
D. No investigation or treatment indicated at present time, carefully observe for onset ofsymptoms
E. Treat with doxycycline for 14 days

D. No investigation or treatment indicated at present time, carefully observe for onset ofsymptoms

There is no need for prophylactic antibiotics for Lyme disease in asymptomatic patients bitten by a tick

This patient has a convincing history for the attachment of ticks from a region of the UK at high risk of Lyme borreliosis. When assessing the risk of tick bites it is important to clarify for how long the tick was attached and if it became engorged. Transmission of pathogenic *Borrelia* species is unlikely if ticks are attached for less than 24 hours or do not become engorged. Ticks should be removed gently using tweezers to minimise irritation to the skin and the risk of rupturing the tick which can increase the risk of infection.

Asymptomatic patients who report a tick bite should not undergo serological testing or receive treatment. Testing can lead to false positives secondary to previous infection or cross-reactive antibodies. Prophylactic treatment of tick bites may be required if the patient is immunosuppressed or in certain regions of the USA where the prevalence of infected ticks is particularly high.

When serological testing is indicated paired blood samples should be taken at four week intervals to allow seroconversion to take place. Treatment with doxycycline or amoxicillin for 14 days is the appropriate treatment for a patient presenting with erythema migrans after a tick bite.

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A 14-year-old, unvaccinated child is brought into the emergency department after possible exposure to measles. The child has juvenile arthritis well controlled on methotrexate but is otherwise well with no other medical issues. His mother explains that the child attended a large party 3 days previously and she received a call this morning informing them that another child who attended the party has just been diagnosed with measles. She has no further information regarding the other child or as to whether they were symptomatic etc.

The 14-year-old reports no symptoms currently and appears well in the emergency department. His examination is unremarkable and observations are as follows:

- Heart rate 100 beats per minute
- Blood pressure 120/80 mmHg
- Respiratory rate 20 breaths per minute
- Oxygen saturations 98%
- Temperature 36.2 ºC

What is the most appropriate management option?

A. No treatment is indicated
B. Provide aciclovir immediately
C. Provide immunoglobulin immediately
D. Test for IgM antibodies and if present start aciclovir
E. Test for serum measles DNA and if present provide immunoglobulin

C. Provide immunoglobulin immediately

Those exposed to measles can be given immunoglobulin to reduce the risk of infection

Providing immunoglobulin (Ig) is correct as the child is unvaccinated, immunocompromised due to methotrexate and has had exposure to measles. The MMR vaccination can also be offered to patients who are unvaccinated and have exposure as vaccine-induced measles antibody develops more rapidly than that following natural infection.

No treatment is incorrect. The child is unvaccinated, immunocompromised and most likely had significant measles exposure therefore immunoglobulin should be provided as a minimum.

Aciclovir is incorrect as it does not play a role in post-exposure prophylaxis or the treatment of active measles.

Testing for serum measles DNA is incorrect as measles is an RNA paramyxovirus and also this will have no impact on management.

IgM antibodies can be tested for, and are normally present within a few days of rash onset however aciclovir is not indicated in measle post-exposure prophylaxis.

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A 30-year-old homeless, intravenous drug user is brought into the emergency department by his concerned friends. The patient has difficulty speaking clearly but his friends report he had been complaining of progressive weakness, especially of his face with difficulty swallowing and some double vision.

On examination, the patient has a broad gait, with flaccid weakness globally. Cranial nerve examination relieves ptosis, ocular and facial muscle weakness. The patient exam is otherwise normal including a normal GCS.

On further questioning, the group who brought the patient in reports another one of their friends was admitted with similar symptoms a few days ago. On review of the hospital computer system, it is noted that this friend's blood cultures grew a gram-positive, anaerobic bacillus.

What should be provided to the patient urgently?

A. Intubation and ventilation
B. Broad spectrum antibiotics
C. Plasma exchange
D. Antitoxin
E. Specific vaccination

D. Antitoxin

The treatment of botulism - supportive care and botulism antitoxin

This patient is suffering from botulism which, although rare, normally presents following consumption of contaminated food (e.g. tinned) or as in this case IV drug use. The patient has the typical presentation of flaccid paralysis with bulbar and ocular involvement, whilst maintaining full consciousness and no sensory disturbance. The mainstay of treatment is supportive with early antitoxin use which can neutralise non-bound, circulating botulinum toxin.

This patient has most likely a diagnosis of botulism. Although this can progress to cause respiratory issues the patient does not currently have evidence of respiratory failure. Antitoxin should be given as a priority and respiratory function monitored.

Broad-spectrum antibiotics can be considered if there is evidence of wound infection with *Clostridium botulinum* otherwise there use is non-beneficial and specific botulism antitoxin with supportive care are the only management that will affect the course of the disease.

Plasma exchange is incorrect as this has no evidence for its use in botulism.

To date, no effective vaccination to assist in the management of botulism has been approved for use.

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A 23-year-old woman attends with fever, myalgia, rash, and headache 4 days after returning from Sao Paulo. She noted that fevers and generalised abdominal pain started the day before she boarded her flight home and that the rash had come up in the last 24 hours.

On examination, the woman has petechiae and a maculopapular rash over her wrists, ankles, and palms as well as a single itchy, erythematous raised papule with a central punctum on her right calf.

Given the likely diagnosis, what is the most appropriate treatment?

A. Albendazole
B. Artemether with lumefantrine
C. Doxycycline
D. Metronidazole
E. Praziquantel

C. Doxycycline

Doxycycline is typically used in the management of typhus

The most likely diagnosis here is Rocky Mountain spotted fever, one of the Rickettsial diseases. Rocky Mountain spotted fever is transmitted via tick bites in endemic areas such as South America - the single, itchy, erythematous, raised papule is descriptive of a tick bite. It presents initially with general symptoms of malaise, fever, and abdominal pain. The classic vasculitic rash then develops after 3-5 days.

Doxycycline is the treatment of choice for Rocky Mountain spotted fever.

Albendazole is incorrect. This would be more appropriate for helminth infections such as roundworm and hookworm.

Artemether with lumefantrine is incorrect. This would be the appropriate treatment for uncomplicated *P. falciparum* malaria. *P. falciparum* malaria is most prevalent in Africa rather than in South America. It can present with similar general symptoms to Rocky Mountain spotted fever

with fevers, headache, and myalgias but is also often associated with hepatomegaly and splenomegaly. It is not typically associated with the vasculitic rash seen in this patient.

Metronidazole is incorrect. It has good action against anaerobic bacteria and protozoa but would not be the choice for Rickettsial infections.

Praziquantel is incorrect. This is used to treat parasitic worm infections such as schistosomiasis. It would not be the choice for Rickettsial infections.

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A 25-year-old gentleman was referred to the outpatient infectious diseases clinic by his GP. He had recently immigrated from Pakistan a few months ago with his extended family. A couple of weeks ago his mother who lived in the same house as him was diagnosed with respiratory tuberculosis and he was accordingly advised to get 'checked up' by his GP. He felt well in himself, stating that he recovered from a cold a few weeks ago having had symptoms of nasal congestion, sore throat and a cough lasting a few days. He has since felt well, denying the presence of any further symptoms. Of note he has not lost weight, not had a fever, and did not suffer from a cough or night sweats. He was unsure what immunisations he had whilst in Pakistan. As a precautionary measure, the GP referred him to the infectious diseases clinic given the close proximity with his mother.

Examination revealed a well looking 25-year-old gentleman. His temperature was 36.6ºC, heart rate 78 bpm, respiratory rate 16/min and oxygen saturations 99% on air. He had a BCG scar on his left arm. Examination of his cardiovascular, respiratory, gastrointestinal and neurological systems were all entirely unremarkable.

Initial investigations revealed the following:

Blood film:negative; including negative malaria screen

Blood culture: negative culture; awaiting prolonged acid-fast bacilli culture result

Quantaferon blood test: positive Mantoux test: positive

Chest x-ray: normal appearances of heart and lung fields, no abnormality was seen

Based upon the above information, what is the next best management step?

A. Commence treatment with isoniazid, pyridoxine, ethambutol, rifampicin and pyrazinamide for 2 months
B. Discharge from clinic with no further action to take
C. Refer for diagnostic bronchoscopy alveolar lavage
D. Commence isoniazid and pyridoxine for 6 months
E. Repeat Quantaferon blood test weekly for the next month - treat if remains positive

D. Commence isoniazid and pyridoxine for 6 months

Latent tuberculosis treatment options:

3 months of isoniazid (with pyridoxine) and rifampicin, or

6 months of isoniazid (with pyridoxine)

This gentleman has latent TB. He has had close (and presumably prolonged) contact with an active TB carrier but other than the presence of transient symptoms of an upper respiratory tract infection has been asymptomatic. He has had a positive Mantoux test, which can be positive with both active and latent TB but can also by a previous BCG vaccination. Quantiferon testing, on the other hand, is not influenced by BCG vaccination status but cannot distinguish between active and latent TB. However in the absence of significant symptoms and with normal investigations including a normal CXR the most likely diagnosis is latent TB and therefore he requires monotherapy with isoniazid (and pyridoxine) for 6 months.

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A 28-year-old man presents to the Emergency Department with a four-day history of recurrent headaches. He reports no significant medical history but mentions a recent trip to Bolivia, where he recalls sustaining a tick bite. He also describes a rash that started on his extremities.

On examination, you observe a maculopapular rash involving his extremities, face, and trunk. His vital signs are as follows: blood pressure 110/80 mmHg, pulse rate 78 beats per minute, and temperature 39.6 ºC.

The following laboratory results are available:

Hb	140 g/L	Male: (135-180)
Platelets	105 * 10 /L 9	(150 - 400)
WBC	3.1 * 10 /L 9	(4.0 - 11.0)
ALT	68 u/L	(3 - 40)

What is the most likely diagnosis?

A. Babesiosis
B. Dengue Fever
C. Lyme disease
D. Rocky Mountain Spotted Fever
E. Tularemia

D. Rocky Mountain Spotted Fever

Headache, fever, rash on the peripheries before spreading centrally, tick bite → Rocky Mountain spotted fever (*Rickettsia ricketsii*)

Rocky Mountain Spotted Fever is the correct diagnosis. The clinical presentation of fever, headache, and a rash that started on the extremities and subsequently spread to the face and trunk, coupled with a travel history to South America (specifically Bolivia) and a tick bite, should prompt consideration of Rocky Mountain spotted fever. This disease involves the proliferation of intracellular bacteria *Rickettsia rickettsii* at the site of the tick bite, predominantly affecting the endothelium of small vessels. Laboratory findings such as thrombocytopenia, leukopenia, and raised liver enzymes further support this diagnosis.

Babesiosis represents a tick-borne disease caused by protozoan parasites of the Babesia genus. Transmission is primarily through Ixode ticks. While it shares common features with Rocky Mountain spotted fever, including fever, headache, thrombocytopenia, and increased liver enzyme levels, Babesiosis characteristically does not present with a rash and mainly infects erythrocytes.

Dengue Fever is an arboviral infection endemic in the Americas transmitted by mosquitoes carrying dengue virus. Clinical manifestations typically include fever, retro-orbital pain, severe myalgia, and a distinctive rash which commences with facial flushing before progressing to a second maculopapular rash on the back of hands and feet extending to the torso. However, this type of rash differs from that described in the scenario provided.

Lyme disease, another tick-borne illness caused by spirochetes of *Borrelia burgdorferi*, is transmitted via hard ticks. It typically presents with an expanding area of localised erythema with central clearing (erythema migrans), often resembling a bull's-eye pattern at the tick bite location. The rash does not match Lyme disease but suggests Rocky Mountain spotted fever.

Tularemia is an uncommon zoonotic infection caused by Francisella tularensis bacteria and transmitted by ticks. It can cause symptoms such as fever, painful ulcerative lesions at inoculation sites with associated regional lymphadenopathy, hepatosplenomegaly, and potentially community-acquired pneumonia. The patient's clinical features do not correspond with those commonly associated with tularemia.

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A 32 year old man presents with left lower limb weakness for the past three weeks. Over the past week his condition has got much worse. He has lost his appetite and about six kilograms in the last two months. In the same period he reported recurrent bleeding from the nose.

With the help of three months of nicotine transdermal patches and vareniciline he is now no longer actively smoking. He is married and works as a salesman in a pharmaceutical company. For the last year he has been to many destinations around the world as part of his job.

On examination he appeared ill. He was oriented in time, place, and person, but was mentally slow in understanding commands during examination. The power in the left leg was grade 2 for all muscle groups. Tone, sensation, coordination and reflexes were all normal. The right leg was normal. No abnormality detected in other systems. The following investigation were ordered:

Hb	10g/dl
Platelets	10 * 10^9/l

WBC	4* 10^9/l
Neutrophils	60%
Lymphocytes	34%
Na+	135 mmol/l
K+	4 mmol/l
Creatinine	95 µmol/l
Urea	4 mmol/l

Urine analysis: Clear

MRI brain showed bilateral multiple hyperintense demyelinating lesions involving sub cortical areas without any mass effect.

What is the most appropriate thing to do?

A. CSF analysis for oligoclonal bands
B. Brain biopsy
C. CSF analysis for herpes simplex virus (HSV)
D. HIV test
E. Stop vareniciline

D. HIV test

HIV, neuro symptoms, widespread demyelination - progressive multifocal leukoencephalopathy

The progressive neurological symptoms with demyelination on MRI points to progressive multifocal leukoencephalopathy (PML).

PML is a demyelinating disease of the CNS due to the JC virus. It occurs almost exclusively in immunosuppressed individuals with the majority of cases being secondary to HIV infection. Patients typically experience insidious onset of focal symptoms that include mainly behavior, cognitive, speech, and motor functions. Polymerase chain reaction (PCR) of the CSF has a high sensitivity and specificity for the detection of JC virus in patients with PML.

Neither the symptoms nor the signs are compatible with either multiple sclerosis or HSV.

Vareniciline could cause low platelets and appetite changes with psychiatric manifestation. But it would not explain the motor deterioration nor the cognitive decline for this patient.

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A 21-year-old man presents to the emergency department with five days of feeling tired, cough, fevers and general lethargy. He becomes quite tearful during the consultation as he has never felt like this.

On examination, he has tenderness over both trapezium muscles, his temperature is 38.6 ºC and his throat is red without any collections of pus.

A rapid point of care test shows that he is influenza positive. \

What is your next management step?

A. Oseltamivir
B. Zanamivir
C. Admit for observation
D. Discharge with reassurance
E. Oral amoxicillin

D. Discharge with reassurance

Low-risk patients with influenza who present 48 hours after symptoms are recommended for supportive treatment

This is a simple case of influenza of which most people who are low risk will recover with supportive treatment. In high risk groups e.g. immunosuppression or pregnancy, oseltamivir or zanamivir can be considered. The duration of symptoms is important as those who present within 48 hours of symptoms are more likely to derive benefit. Oral antibiotics are not usually recommended though bacterial superinfection can occur in influenza.

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A 27 year-old Lithuanian male presents to the Emergency Department with a short history productive cough, fever and breathlessness. The chest X-Ray shows left upper zone consolidation. His white cell count and CRP are elevated and IV antibiotics are commenced for a community acquired pneumonia. He makes some improvement after 48 hours but remains on 4L of oxygen to maintain adequate saturations. An interferon gamma release assay for mycobacterium is positive.

What would be the correct course of action?

A. Send three morning sputum samples for acid fast bacilli
B. Arrange a bronchoscopy and washings for acid fast bacilli
C. No action, continue antibiotics, repeat CXR in 6 weeks time
D. Treat for latent pulmonary tuberculosis with two agents
E. Treat for active pulmonary tuberculosis with quadruple therapy

A. Send three morning sputum samples for acid fast bacilli

This young gentleman's positive interferon release assay shows he has been exposed to tuberculosis in the past. It however does not give any information regarding this active infection which could represent pulmonary tuberculosis or bacterial pneumonia. A wise course of action would include isolating the patient and sending three morning sputum samples to look for acid fast bacilli. He is not currently fit for bronchoscopy given his oxygen requirements.

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A 24-year-old lady presents 16 weeks pregnant. She complains of myalgia, sore throat and coryza and is noted to have a pyrexia of 38.1. She works in a nursing home which is currently closed due to an influenza A outbreak. On further questioning, you note that she has been vomiting every morning for the past 8 weeks and is no longer able to take oral medication. On examination her chest is clear but she is obviously coryzal with an erythematous oropharynx (no exudate). Chest Xray is clear.

Apart from informing the obstetrics team, how would you manage this patient?

A. Admit, isolate and commence oseltamivir
B. Admit and observe
C. Oral antibiotics
D. Admit, isolate and commence zanamivir
E. Admit, isolate and await nose and throat swabs before commencing antiviral therapy

D. Admit, isolate and commence zanamivir

This patient has had known exposure to influenza A. She should be admitted for observation and immediately commenced on empirical treatment. NICE recommends oseltamivir treatment as first line for suspected or confirmed influenza A in pregnancy. However, given that this patient has been vomiting, zanamivir (intranasal/intravenous) should be used in preference to oral Tamiflu (oseltamivir). Empirical treatment should be commenced immediately and not withheld until the results of swabs are available.

There may be a role for antibiotics if there is a co-existing infection or evidence of pneumonia on chest X-ray.

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A 35 year-old Brazilian accountant presented to the medical outpatient clinic with progressive leg swelling over the last 18 months. His mobility had become increasingly restricted due to both the swelling and shortness of breath on exertion. His father and paternal grandfather had both died from ischaemic heart disease in their 60s. His past medical history was unremarkable. He had smoked 15 cigarettes a day for 15 years and drank 15 units of alcohol per week.

On examination, his temperature was 36.5ºC, blood pressure was 95/50 mmHg and heart rate was 75 beats per minute. His JVP was elevated to the angle of the jaw. His chest was clear on auscultation, but a third heart sound and pan-systolic murmur were audible on auscultation of the precordium. Bilateral pitting oedema to mid-thigh level was present.

Investigations:

Haemoglobin	131 g/L (130-180)
White cell count	6.9 x 10 /L (4.0-11.0)9
Neutrophil count	3.1 x 10 /L (2.0-7.5)9
Lymphocyte count	1.9 x 10 /L (1.3-3.5)9
Eosinophil count	1.1 X 10 /L (0.1-0.4)9
Platelets	260 x 10 /L (150-400)9

Sodium	132 mmol/L (135-145)
Potassium	3.6 mmol/L (3.5-5.0)
Urea	8.0 mmol/L (2.5-7.5)
Creatinine	101 mol/L (20-90)
Fasting plasma glucose	7.2 mmol/L (3.0-6.0)

Echocardiogram	Moderately impaired left ventricular systolic function Dilated left ventricle with moderate mitral regurgitation

What is the most appropriate treatment for the underlying condition?

A. Sulfadiazine and pyrimethamine
B. Doxycycline
C. Artesunate
D. Benznidazole
E. Mebendazole

D. Benznidazole :

This patient has developed chronic infection with tropical trypanosomiasis (Chagas disease). The parasite is endemic in South America, and is transmitted by the triatomine bug. Initial infection may present with transient fever and/or a swelling around the site of inoculation. In this case, chronic infection has developed; this may consist of dilated cardiomyopathy, arrhythmias or oesophageal dysmotility mimicking achalasia. Though this patient has some risk factors for ischaemic heart disease, the chances of this affecting him at this age are relatively small. The current treatment of choice for tropical trypanosomiasis is benznidazole.

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A 65-year-old man is undergoing treatment in the intensive care unit. He had presented 8 days previously after suffering a seizure at home. He had been unable to give a coherent history at the time of presentation but his family had reported he had been unwell for about two weeks. Initial symptoms included a fever, myalgia, anorexia and an intense itching of his left arm. Subsequently, the patient had developed progressive confusion and agitation and was incoherent by the time of presentation to hospital. The patient was a long-term resident of the UK, but had made a trip back to his native India around 6 months previously. During that trip, his wife recalled the patient being bitten by a dog in his family village, although the wound had been minor and healed without incident after basic first aid. Medical history was otherwise fairly unremarkable, including hypertension, tablet-controlled type 2 diabetes and gout.

At presentation, the patient had been noted to been highly agitated and disorientated. Hypersalivation was noted and patient had been refusing to eat or drink. Examination had been limited by the patient's agitation but no overt focal neurological symptoms were noted in the cranial nerve or peripheral nervous examination. After the patient had been sedated, it was noted that striking a large muscle group with a tendon hammer led to few seconds of mounding of the muscle.

CSF protein	457 g / dL
CSF Microscopy	NAD
CSF rabies virus neutralising antibodies	Not detected
Serum rabies virus neutralising antibodies	Detected

At the present time, the patient is receiving end-of-life care. He is sedated and appears comfortable secondary to midazolam delivered by a syringe driver. His family wish to be present at his bedside.

What is the appropriate personal protection for healthcare workers and family members to use while in contact with the patient?

A. High isolation
B. Standard precautions
C. Respiratory isolation
D. Reverse isolation
E. Biocontainment precautions

B. Standard precautions

The patient is unfortunately suffering from rabies. His presentation is consistent with the classical form of 'furious rabies', with fever, agitation, confusion and seizures. The response of the patient's muscles to being struck with a tendon hammer described in the question is called myoedema and is suggestive of rabies. Neutralising serum antibodies may be detected 7-8 days after the onset of symptoms but are only occasionally found in CSF. An alternative form of the disease ('paralytic rabies') is also known, featuring an ascending paralysis or symmetric quadriparesis.

The patient presumably contracted the disease on his previous trip to India when he was bitten by a dog. An incubation period of 6 months is not uncommon (mean incubation time 274 days in one study) with cases reported with incubation time up to 10 years. By the time neurological symptoms have taken hold, death is inevitable in unimmunised patients. This highlights the need for appropriate vaccination for individuals travelling to high-risk areas, as well as post-exposure vaccination and immunoglobulin administration.

Lyssaviruses such as rabies cannot cross intact skin and humans are regarded as an end-host (outside of transplantation-associated transmission). Therefore, only standard infection-prevention precautions such as gloves and gowns are required.

High isolation refers to the extreme precautions taken with high-transmissible and dangerous infections such as Ebola. Respiratory isolation requires the use of a mask to prevent droplet transmission by the respiratory route. Reverse isolation refers to techniques required to prevent external pathogens causing harm to an immunosuppressed patient. Biocontainment refers to laboratory biosafety techniques required when handling highly pathogenic organisms.

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A 65 year-old gentleman with type 2 diabetes mellitus and alcoholism presents with fever, headache and neck stiffness. He has a history of previous anaphylactic reaction to penicillin.

On examination the temperature is 37.9ºC, respiratory rate is 20 breaths/min and heart rate is 105 beats per minute. Nutritional state and dental hygiene is poor. The chest is clear to auscultation. The abdomen is soft and non-tender. The Glasgow coma scale score is 15, the patient is uncomfortable during pen-torch pupillary examination and neck flexion is limited by pain.

CSF examination reveals:

White Cells	560 per mm³ (85% polymorphs)
Red cells	8 per mm³
Protein	0.9g/L
Glucose	3.3mmol/L (serum glucose 9.2mmol/L
Gram stain	Gram-negative coccobacilli

Which antimicrobial agent should be initiated?

A. Vancomycin
B. Ceftriaxone
C. Chloramphenicol
D. Rifampicin
E. Gentamicin

C. Chloramphenicol

This patient has meningitis caused by Haemophilus influenzae. Immunocompromised patients including those with alcoholism or diabetes are at risk.

Other gram -ve coccobacilli include Gardnerella vaginalis, Chlamydia trachomatis, Bordetella pertussis, Coxiella burnetti and Haemophilus ducreyi however these would not classically cause meningitis.

Cefotaxime or ceftriaxone would be the first-line treatment for haemophilus meningitis however for those with a history of anaphylaxis to penicillin, chloramphenicol is the most appropriate choice.

Chloramphenicol would also be the appropriate choice in meningococcal meningitis in a penicillinallergic patient.

The area of induration described in the vignette constitutes a positive Mantoux test. The area of surrounding erythema is not taken into account.

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A 54-year-old oil businessman who frequently visits the Gambia and is usually careful with his malaria prophylaxis has been admitted with general malaise and relapsing/remitting fevers which seem to occur every third day. He returned from the Gambia about one week ago and on this occasion, he did not take his malaria prophylaxis as he has never caught it before. He has no past medical history of note and no regular medication. Malarial parasites are seen on the thick and thin films and it is confirmed as *Plasmodium vivax* by the Malaria Reference Laboratory.

What is the most appropriate management according to current UK guidelines?

A. Chloroquine
B. Atovaquone-proguanil
C. Chloroquine and primaquine
D. Artemether with lumefantrine
E. Intravenous artesunate + primaquine

C. Chloroquine and primaquine

Plasmodium vivax is the second most commonly seen cause of malaria by UK statistics after *Plasmodium falciparum* malaria. It is characteristically a cause of benign tertian malaria where fevers occur every three days. Current UK guidelines (Health Protection Association(are that nonfalciparum malaria should be treated with chloroquine but *P. vivax* and *P. ovale* are unusual in that dormant parasites (or hypnozoites) can exist within the liver.

The only currently available treatment for hypnozoites is primaquine and vivax malaria is more effectively treated with a higher dose than ovale (30mg). Therefore the correct answer is chloroquine and high-dose primaquine. Anyone commencing treatment with primaquine should have their glucose-6-phosphate dehydrogenase status tested as primaquine could precipitate severe haemolysis in deficient patients.

Artesunate, atovaquone-proguanil and artemether with lumefantrine are treatments for falciparum malaria.

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A 35-year-old man presents to the genitourinary medicine clinic with a 1-week history of an ulcer on his penis. It is painless and he feels it might be increasing in size, though there are no other ulcers. He has never had any ulcers before.

He is HIV positive and remains sexually active with one regular male partner. They do not usually use condoms as he strictly adheres to his antiretroviral medication schedule and has an undetectable viral load.

He had gonorrhoea aged 18 which was treated with antibiotics at the time and he has no history of any other sexually transmitted infections.

He has no other past medical history and no drug allergies. He takes no medications other than his antiretrovirals.

On examination, he has a single shallow ulcer 1 x 2 cm on the dorsal surface of the penis with a small rim of surrounding erythema. He has no other rashes or lymphadenopathy. The systemic examination is otherwise unremarkable with no focal neurology.

Swabs from the ulcer are negative for herpes simplex virus 1 and 2. Penile swabs are negative for chlamydia and gonorrhoea.

Syphilis serology result: Enzyme immunoassay (EIA) +

Treponema Pallidum particle agglutination assay (TPPA) + Rapid plasma reagin (RPR) -

What is the best immediate management option?

A. Intramuscular penicillin G and check repeat syphilis serology
B. Intramuscular penicillin G and check syphilis PCR
C. Check syphilis PCR
D. Oral doxycycline and check repeat syphilis serology
E. Oral doxycycline and check syphilis PCR

B. Intramuscular penicillin G and check syphilis PCR

This gentleman has a painless single penile ulcer which given negative testing for HSV, should be investigated for syphilis.

His syphilis serology shows a positive screening EIA and follow up TPPA (both treponemalspecific). However, his RPR titre is undetectable. This could indicate either past infection only or early acute infection without seroconversion on the RPR.

This gentleman is having unprotected intercourse and is HIV positive, so high risk for syphilis even if he gives a history of a single long-term partner. He also gives no history of past infection and so he should be treated for syphilis with antibiotics. Given he has no allergies, the first choice should penicillin G.

However, for clarification regarding diagnosis, he requires further testing, the gold standard being syphilis PCR.

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A 35-year-old female returned from Tanzania 2 weeks ago. She had consulted her GP after feeling unwell over the previous 3 days with intermittent symptoms of fever, headache and generalised arthralgia. She had taken doxycycline as an antimalarial and denies missing any of the prescribed doses and had been fully vaccinated prior to her travel.

She is referred to the medical take where you retake the history. She admits to getting mosquito bites regularly whilst away, one of them she seemed to react severely to and this has left a mark on her shoulder.

On examination she appears drowsy, she has mild heptosplenomegally and there is a well circumscribed, rubbery, painful, red lesion approximately 5cm in diameter above her left shoulder.

What is the likely diagnosis?

A. Plasmodium falciparum
B. Trypanosomiasis brucei rhodesiensi
C. Trypanosomiasis brucei cruzi
D. Trypanosomiasis brucei gambiensi
E. Toxoplasmosis

B. Trypanosomiasis brucei rhodesiensi

Human African trypanosomiasis (HAT), also known as sleeping sickness, is caused by protozoan parasites. There are two forms of the disease, an acute form occurring mainly in East Africa caused by *Trypanosoma brucei rhodesiense*, and a more chronic form occurring in West and Central Africa, caused by *Trypanosoma brucei gambiense*. *Trypanosomiasis brucei cruzi* is endemic in south america where it causes Chagas disease.

Trypanosomiasis rhodesiense is transmitted by the tsetse fly which are classically found on safari in Tanzania. *T. rhodesiense* will often form a chancre at the site of inoculation and can often be the first and only sign of disease where it is rapidly progressive.

Given the story we are immediately highlighted to the potential of malaria. However given that she has taken adequate prophylaxis we should keep an open mind to other potential diagnoses. Furthermore her illness started 3 days prior to her admission to hospital making *P.falciparum* less likely as this would have resulted in her being very unwell by this point.

As explained *T.Cruzi* is endemic in south america and can therefore be discounted early on. *T.gambiense* is likely to present with a much more chronic picture. Toxoplasmosis is very unlikely in an immunocompetant host and her travel to Tanzania would not necessarily increase her risk of acquiring the condition. As a result the most likely diagnosis is *T.rhodesiense*. Diagnosis can be made by demonstration of the parasite in the blood, lymph node/chancre aspirate or on CSF examination. The condition is fatal in the absence of treatment.

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A 59 year-old man attends his general practitioner with a 10 day history of watery diarrhoea. His stools are unformed, sometimes associated with fresh red blood. He also has associated abdominal cramps for which he has been taking paracetamol. He estimates a 5kg weight loss during this period, and has struggled to tolerate food or fluids due to significant pain on swallowing. He also has an intermittent sensation of needing to defecate, but being unable to.

He has been diagnosed with HIV 8 years ago, and admits that he has not been attending follow up, or taking his medications. He is unsure what his last CD4 cell count was. He has had 5 episodes of pneumonia over the last 12 months, the last one was 4 months ago.

On examination, he has dry mucous membranes with reduced skin turgor. He has generalised abdominal tenderness, but no features of peritonism, or organomegaly.

Observations

- Respiratory rate- 18 breaths per minute
- Heart rate- 110 beats per minute
- Blood pressure- 104/60 mmHg
- Temperature 37.7ºC

What is the most likely pathogen causing his symptoms?

A. Cryptosporidiosis
B. Clostridium difficile
C. Giardia
D. E Coli 0157
E. Cytomegalovirus colitis

E. Cytomegalovirus colitis

Cytomegalovirus (CMV) colitis is usually seen in patients who are immunocompromised. It is a member of the herpes virus, for which around 50-80% of the general population are seropositive. The virus lays dormant until the patient is sufficiently immunocompromised.

Approximately 30% of all patients with AIDS will develop a gastrointestinal CMV infection. This usually occurs when the CD4+ counts drop to below 100 mm3.

CMV can affect the gastrointestinal tract from the oesophagus to the rectum, and symptoms depend on the site of infection. CMV oesophagitis causes odynophagia and retrosternal chest pain. CMV enterocolitis commonly presents with diarrhoea, fever, weight loss and per rectal bleeding. Tenesmus can occur if there is rectal involvement.

Cryptosporidiosis is a protozoal infection caused by cryptosporidium parvum. Immunocompromised individuals are particularly susceptible. The incubation period is between 2- 10 days (average 7) and symptoms usually last about 1 to 2 weeks, although can occur for longer. The most common symptom is watery diarrhoea. Other symptoms include stomach cramps, dehydration, nausea and vomiting and fever.

E Coli 0157 is a relatively rare cause of infective diarrhoea in England. Symptoms range from mild gastroenteritis through to severe bloody diarrhoea. It can be associated with haemolytic uraemic syndrome and thrombotic thrombocytopaenic purpura.

Giardia is causes by the protozoon parasite Giardia lamblia. Symptoms include diarrhoea, foul smelling flatulence and abdominal cramps. Symptoms typically last between 2-4 weeks, but chronic diarrhoea and malabsorption can occur.

The patient has likely got a very low CD4+ count, due to his non-compliance with medication and follow up and history of recurrent pneumonia. CMV colitis with rectal and oesophageal involvement would explain the tenesmus, odynophagia, along with the bloody diarrhoea, abdominal cramps and weight loss.

Cryptosporidiosis and giardia do not tend to cause bloody diarrhoea. Clostridium difficile is an important cause of diarrhoea in immunocompromised patients. However, neither clostridium difficule nor E Coli 0157 this would not explain the odynophagia or tenesmus.

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A 47-year-old man presented with a headache to the emergency department a few days ago. His partner accompanied him and reported that he has been more confused over the last few days. He had also been febrile and complained of swollen glands in his neck. He had a self-terminating seizure whilst awaiting transfer to the medical ward.

His past medical history is significant for HIV. His partner reported he is not compliant with his anti-retroviral treatment and last year was hospitalised for severe pneumonia.

A CT head showed multiple ring-enhancing lesions. The infectious diseases team reviewed him and suggested additional blood tests, results of which are shown below:

Hb	140 g/L	(135-180)
Platelets	167 * 10 /L 9	(150 - 400)
WBC	3.8 * 10 /L 9	(4.0 - 11.0)
Na^{+}	137 mmol/L	(135 - 145)
K^{+}	4.5 mmol/L	(3.5 - 5.0)
Urea	8.5 mmol/L	(2.0 - 7.0)
Creatinine	137 µmol/L	(55 - 120)
CRP	50 mg/L	(< 5)
Toxoplasmosis serology	positive	
Cryptococcal serology	negative	
β-d-Glucan	normal	
Galactomannan	normal	

What is the most appropriate treatment to give this patient?

A. Co-trimoxazole and prednisolone
B. Fluconazole
C. Pyrimethamine and sulphadiazine
D. Amphotericin B
E. Pentamidine and prednisolone

C. Pyrimethamine and sulphadiazine

Immunocompromised patients with toxoplasmosis are treated with pyrimethamine plus sulphadiazine

This man has a background of HIV and is not compliant with treatment, therefore is immunocompromised and at risk of opportunistic infections. Toxoplasmosis serology is positive and the multiple ring-enhancing lesions on CT support this diagnosis. Management of toxoplasmosis in immunocompromised patients is with pyrimethamine and sulphadiazine.

Co-trimoxazole and prednisolone is used in the management of *Pneumocystis jirovecii* pneumonia. There are no chest symptoms to suggest this is the cause of this patient's presentation.

Fluconazole can be used to treat fungal infections such as candidiasis.

Amphotericin B is used in the management of cryptococcal infection. Cryptococcus serology is negative, and the CT head would show cerebral oedema and meningeal enhancement if this was the diagnosis.

Pentamidine and prednisolone is an alternative treatment regimen for *Pneumocystis jirovecii* pneumonia.

Question 6 of 277

A 28-year-old man presents to the clinic with a rash on his foot. He states that he first noticed it two weeks after returning from Thailand. The rash is very itchy and has been keeping him awake at night. He has no past medical history and takes no regular medicines. On examination he has a erythematous, oedematous, serpiginous track on the superior aspect of his right foot.

What is the treatment of choice for this condition?

A. Clotrimazole
B. Terbinafine
C. Albendazole
D. Flucloxacillin
E. Doxycycline

C. Albendazole

Albendazole is the treatment of choice for cutaneous larva migrans

The clinical history is suggestive of cutaneous larva migrans which is caused by infection of the dog hookworm Ancylostoma braziliense. Management is with albendazole or ivermectin.

Flucloxacillin and doxycycline are indicated for cellulitis.

Clotrimazole and terbinafine are used in the treatment of fungal infections such as ringworm.

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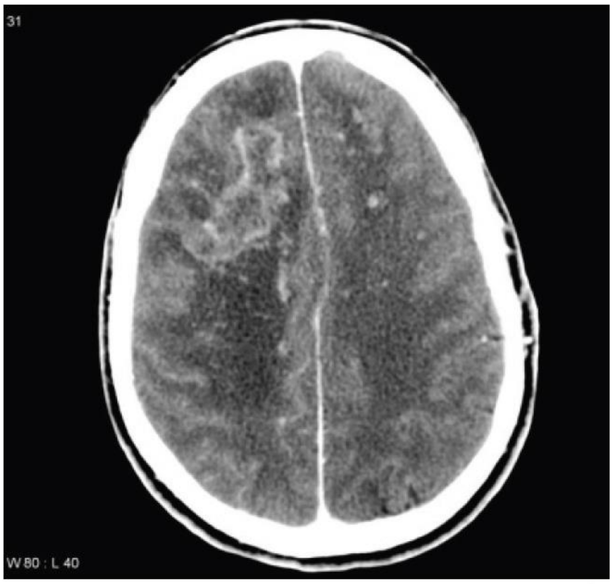
A 33-year-old man who is HIV positive is admitted to the Emergency Department with confusion and drowsiness. He has been complaining of headaches for a number of days. On examination heart rate is 90/min, blood pressure 104/78 mmHg and temperature is 37.2ºC. He is confused giving a Glasgow Coma Scale (GCS) score of 14. There is no photophobia or neck stiffness.

His infectious diseases consultant reports that he is prescribed highly active antiretroviral treatment (HAART) but his compliance is poor and he often misses clinic appointments.

A CT scan is requested: .



What is the most likely diagnosis?

A. Cerebral toxoplasmosis
B. Tuberculosis
C. CMV encephalitis
D. Progressive multifocal leukoencephalopathy
E. Cryptococcal infection

A. Cerebral toxoplasmosis

HIV, neuro symptoms, multiple brain lesions with ring enhancement - toxoplasmosis

The CT scan shows multiple ring enhancing lesions consistent with cerebral toxoplasmosis. He is at risk of this due to his poor compliance with medication.

Cerebral toxoplasmosis is the most common neurological infection seen in HIV, occurring in up to 10% of patients.

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A 25-year-old man presents to the genitourinary medicine clinic with a penile lesion which has been present for the last three weeks but is now starting to reduce in size. He has had two sexual partners in the last 2 months, who were both men. There is no history of travel outside of the UK in the last 3 years.

The lesion is a shallow ulcer with well-defined margins, which is not tender to palpation. He has some non-tender enlarged lymph nodes on both sides in the inguinal region.

What is the most likely diagnosis?

A. Chancroid
B. Donovanosis
C. Lymphogranuloma venereum
D. M-pox
E. Primary syphilis

E. Primary syphilis

Primary syphilis is associated with **painless** ulceration

The correct answer is **primary syphilis** , which is caused by *Treponema pallidum*. This classically presents with a chancre at the site of entry of infection. This usually starts as a painless papule, which evolves into a painless ulcer. The lesion is usually single but multiple lesions can occur. It generally heals over a few weeks.

Chancroid is incorrect. This is a sexually transmitted disease caused by *Haemophilus ducreyi*. However, unlike in this patient, the ulcers are commonly multiple and very painful. The inguinal lymphadenopathy in chancroid is also generally painful. Chancroid is rare in the UK, and most cases are acquired abroad in Africa or South East Asia.

Donovanosis is incorrect. This condition is also called granuloma inguinale and is caused by *Klebsiella granulomatis*. The genital ulcers in this condition are painless with a classically 'beefy red' base. This diagnosis is less likely in this patient because the ulcers would not start to heal without treatment but would continue to enlarge, and because it is rarely seen in the UK. It is most commonly seen in tropical regions.

Lymphogranuloma venereum is incorrect. This is caused by serovars L1, L2 and L3 of *Chlamydia trachomatis* . It is more common in tropical areas but does occur in the UK, mainly in men who have sex with men. It can cause painless ulceration in the primary stage, but unlike in this scenario, the ulcers are small and heal within 1-3 days so this stage is generally not noticed by patients. The secondary stage is characterised by painful swollen inguinal lymph nodes or anal symptoms in patients who have acquired an anal/rectal infection.

M-pox is incorrect. The M-pox virus classically causes painful vesicular lesions, which usually start at the site of inoculation and are generally multiple. The lesions may then spread to involve other areas of the body.

Question 109 of 277

A 35-year-old man presents to his general practitioner with two months of dysphagia and odynophagia. He has a past medical history of HIV. He is not compliant with anti-retroviral treatment.

Observations:

- Heart rate 88 beats per minute
- Blood pressure 120/72 mmHg
- Respiratory rate 18/minute
- Oxygen saturations 98% on room air
- Temperature 37ºC

On examination, there are white patches on the gums, tongue and extending past the pharynx. The examination is otherwise unremarkable.

What is the most appropriate treatment?

A. Amphotericin
B. Fluconazole
C. Lansoprazole
D. Nystatin
E. Surgical excision

B. Fluconazole

Candidiasis in immunocompromised patients should be treated using high dose fluconazole

Fluconazole is the correct answer. The description of the lesions is typical of candidiasis. Given the patient has oesophageal symptoms in the form of dysphagia and odynophagia, we can assume that the oesophagus is involved, in addition to the oro-pharynx. The patient is not compliant with treatment for HIV and is therefore at risk of developing AIDS. Oesophageal candidiasis is an AIDSdefining illness. In immunocompromised patients, the treatment of candidiasis is with high dose fluconazole.

Amphotericin is incorrect. This is an intravenous treatment associated with significant toxicity. It is reserved for life-threatening, disseminated fungal infections.

Lansoprazole is incorrect. The dysphagia and odynophagia are not likely a result of gastrooesophageal reflux.

Nystatin is incorrect. This would be suitable to treat oral thrush in an immunocompetent patient but would not be sufficient in this case.

Surgical excision is incorrect. A differential for a single white patch in the mouth or tongue might be oral leucoplakia, which is a pre-malignant condition and which could be surgically excised. However, in this context, multiple white patches in multiple locations is more likely to reflect candida.

Question 110 of 277

A 65-year-old man presents to the emergency department complaining of a 1 day history of fever, new rash and severe pain in his leg. His past medical history includes type 2 diabetes and peripheral vascular disease. His medications include metformin and clopidogrel. He smokes twenty cigarettes daily and lives alone.

His observations are heart rate 110 beats per minute, blood pressure 88/51 mmHg, respiratory rate 21/minute, oxygen saturations 96% on room air and temperature 38.4ºC.

On examination, he appears unwell. He is clammy and diaphoretic. There is an area of erythema extending from his right calf to his right thigh. It is warm to touch and in the region of the thigh, is diffusely tender. The degree of tenderness seems out of proportion to the severity of the rash. Cardiovascular, respiratory and abdominal examinations are unremarkable.

Blood tests:

Hb	111 g/L	Male: (135-180)Female: (115 - 160)
Platelets	444 * 10 /L 9	(150 - 400)
WBC	14.4 * 10 /L 9	(4.0 - 11.0)
Na +	131 mmol/L	(135 - 145)
K +	4.2 mmol/L	(3.5 - 5.0)
Urea	9.8 mmol/L	(2.0 - 7.0)
Creatinine	180 µmol/L	(55 - 120)
CRP	176 mg/L	(< 5)
Lactate	3.4 mmol/L	(0.5 - 2.2)
Creatine kinase	1200 U/L	(40-320)

Despite antibiotics and aggressive fluid resuscitation with 3 litres of normal saline, he remains hypotensive with a blood pressure of 85/52 mmHg.

Given the likely diagnosis, what combination of organisms is likely to be responsible?

A. Bacteroides fragilis and Clostridium perfringens
B. Pseudomonas aeruginosa and Escherichia coli
C. Staphylococcus aureus , Streptococcus pyogenes and Enterococcus faecalis
D. Streptococcus pyogenes , Escherichia coli and Clostridium perfringens
E. Vibrio vulnificus and Pseudomonas aeruginosa

D. Streptococcus pyogenes , Escherichia coli and Clostridium perfringens

Type 1, or polymicrobial, necrotising fasciitis is the most common

Streptococcus pyogenes , *Escherichia coli* and *Clostridium perfringens* is the correct answer. The diagnosis is necrotizing fasciitis, as evidenced by a severe soft tissue infection with pain and tenderness out of proportion to the clinical signs. Further supportive of evidence of necrotizing fasciitis versus a simple skin and soft tissue infection is the associated evidence of septic shock and organ dysfunction (acute kidney injury and raised lactate) with a raised creatine kinase. The most common type of necrotizing fasciitis is 'Type 1', which is caused by a combination of gram positive, gram negative and anaerobic bacteria. Furthermore, this type is more common in an older population with type 2 diabetes and peripheral vascular disease. It is the only option listed that is polymicrobial. Management is with antibiotics and aggressive surgical debridement.

Pseudomonas aeruginosa and *Escherichia coli* is the wrong answer. While both of these organisms can cause necrotizing fasciitis, they are both gram negative rods. They are less likely to cause necrotizing fasciitis either alone or in combination when compared to a polymicrobial combination.

Staphylococcus aureus , *Streptococcus pyogenes* and *Enterococcus faecalis* is incorrect. All of these organisms can cause necrotizing fasciitis. However, they are a less likely combination than the polymicrobial subtype and are all gram positive cocci. Type 2 necrotizing fasciitis tends to be caused by *Streptococcus pyogenes* alone and is more common in a younger population.

Bacteroides fragilis and *Clostridium perfringens* is the wrong answer. These are anaerobic bacteria that can cause necrotizing fasciitis but they are less likely to cause necrotizing fasciitis either alone or in combination than the polymicrobial subtype.

Vibrio vulnificus and *Pseudomonas aeruginosa* is the wrong answer. These are both gram negative rods rather than a polymicrobial combination. *Vibrio vulnificus* has been implicated in the development of necrotizing fasciitis and is a bacterium found in saltwater but this is a rare cause.

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A 33-year-old woman is brought to the emergency department by ambulance. She was found by her sister at home, confused and complaining of headache and fever. She has no past medical history and is on no regular medications. She had returned seven days previously from a trip on the Trans-Siberian Railway where she had stopped at a number of locations to do some hiking with friends. She had a brief flu-like illness towards the end of the holiday, which had resolved.

Her observations are heart rate 93 beats per minute, blood pressure 128/84 mmHg, respiratory rate 19/minute, oxygen saturations 96% on room air and temperature 38.4ºC.

On examination, her Glasgow coma scale is 12/15 (eyes 3 verbal 4 motor 5). There is evidence of nuchal rigidity and when she opens her eyes she is photophobic. Examination is otherwise unremarkable and there is no evidence of rash.

Urinalysis is normal.

An ECG demonstrates sinus rhythm.

Plain radiography of the chest demonstrates clear lung fields. Blood tests:

Hb	137 g/L	Male: (135-180)Female: (115 - 160)
Platelets	149 * 10 /L 9	(150 - 400)
WBC	11.8 * 10 /L 9	(4.0 - 11.0)
Na +	137 mmol/L	(135 - 145)
K +	4.2 mmol/L	(3.5 - 5.0)
Urea	6.4 mmol/L	(2.0 - 7.0)
Creatinine	101 µmol/L	(55 - 120)
Glucose	4 mmol/L	(4-11)
CRP	38 mg/L	(< 5)

She is treated empirically with ceftriaxone and aciclovir for presumed meningo-encephalitis and a lumbar puncture is performed.

Results:

Appearance	clear	
WCC	80 cells/µL	(<5)
Cell type	lymphocytes	92%
Glucose	3.6 mmol/L	(2.8-4.2)
Protein	.65 g/L	(0.15 -0.45)

Subsequent testing reveals specific IgM antibodies in the CSF against a flavivirus.

What is the appropriate next step in management?

A. Continue aciclovir and ceftriaxone
B. Continue aciclovir and stop ceftriaxone
C. Continue ceftriaxone and stop aciclovir
D. Stop ceftriaxone and aciclovir and manage supportively
E. Stop ceftriaxone and aciclovir and start ganciclovir

D. Stop ceftriaxone and aciclovir and manage supportively

Treatment for tick-borne encephalitis is supportive

Stop ceftriaxone and aciclovir and manage supportively is the correct answer. The patient has tickborne encephalitis. This is evidenced by the fact that she has returned from a region where the disease is endemic (Siberia) and has presented with a biphasic illness characterized by flu-like symptoms in the first stage and neurological dysfunction in the form of meningoencephalitis in the second stage. Her lumbar puncture results suggest a viral meningitis and her CSF testing reveal specific antibodies against a flavivirus, which is the cause of tick-borne encephalitis. There is no specific antiviral treatment for tick-borne encephalitis. Patients need admission to hospital if neurological symptoms are present and require supportive care in the form of analgesia, antipyretics, fluid and electrolyte management and intensive care input if appropriate.

Continue aciclovir and ceftriaxone is an incorrect answer. We have confirmed a diagnosis of tickborne encephalitis. Ceftriaxone can be stopped as the CSF results do not indicate a bacterial infection and aciclovir can be stopped as we have a confirmed diagnosis that does not benefit from specific anti-viral treatment. Aciclovir is indicated in cases of meningo-encephalitis caused by herpes zoster and herpes simplex.

Continue aciclovir and stop ceftriaxone is incorrect. Aciclovir has no benefit in the management of tick-borne encephalitis.

Continue ceftriaxone and stop aciclovir is incorrect. The CSF results demonstrate a lymphocytic pleocytosis with a normal glucose, indicating a viral meningitis. Ceftriaxone has no role in the management of viral infections.

Stop ceftriaxone and aciclovir and start ganciclovir is incorrect. Ganciclovir is predominantly used in the treatment of severe cytomegalovirus infection in immunocompromised patients. It has no role in the treatment of tick-borne encephalitis.

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A 65-year-old man presents to the emergency department with three days of fever, leg pain and rash. He has a past medical history of type 2 diabetes, peripheral vascular disease and hypertension. He is on regular clopidogrel, amlodipine, metformin, gliclazide and insulin. He is independent and a retired shoe-maker.

His observations are, heart rate 121 beats per minute, blood pressure 84/56 mmHg, respiratory rate 24/minute, oxygen saturation 95% on room air and temperature 39.1ºC.

On examination, he appears diaphoretic and unwell. His capillary refill time is four seconds and he is cool to touch. His pulse is regular and thready. Chest auscultation is normal. His abdomen is soft and non-tender. Heart sounds are normal and there are no murmurs. His left thigh is erythematous and painful to touch. The pain appears out of proportion to the clinical signs and the leg feels wooden and hard.

Blood tests:

Hb	108 g/L	Male: (135-180)Female: (115 - 160)
Platelets	555 * 10 /L 9	(150 - 400)
WBC	21.1 * 10 /L 9	(4.0 - 11.0)
Na +	131 mmol/L	(135 - 145)
K +	4.8 mmol/L	(3.5 - 5.0)
Urea	14.2 mmol/L	(2.0 - 7.0)
Creatinine	181 µmol/L	(55 - 120)
CRP	321 mg/L	(< 5)

What is the appropriate initial pharmacological management?

A. Benzyl-penicillin and Flucloxacillin
B. Flucloxacillin
C. Intravenous immunoglobulin
D. Tazocin and Clindamycin
E. Vancomycin and Metronidazole

D. Tazocin and Clindamycin

Type 1, or polymicrobial, necrotising fasciitis is the most common

Tazocin and clindamycin is the correct answer. This man has necrotizing fasciitis as evidenced by an erythematous rash with pain out of proportion to the clinical signs, fever, a tense thigh and shock with organ dysfunction (acute kidney injury). The most common type of necrotizing fasciitis is type 1, which is polymicrobial. We, therefore, need an antibiotic combination that provides adequate coverage against gram-positive, gram-negative and anaerobic bacteria. Tazocin and clindamycin is the only option listed that fulfils these criteria. Clindamycin has the additional benefit of theoretically inhibiting toxin production in severe streptococcal infections. The polymicrobial type is in particular, more common in diabetic patients.

Benzyl-penicillin and Flucloxacillin are not correct. This will only provide adequate coverage for streptococcal and staphylococcal skin and soft tissue infections (gram-positive) but will not adequately treat gram-negative and anaerobic infections.

Flucloxacillin is incorrect. Again, this has activity predominantly against gram-positive bacteria.

Intravenous immunoglobulin is incorrect. This treatment can be used as an adjunct in severe skin and soft tissue infections but it does not replace antibiotic treatment.

Vancomycin and metronidazole are incorrect. Vancomycin provides coverage for gram-positive organisms and metronidazole for anaerobic bacteria but there is not sufficient gram-negative coverage in this combination.

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A 32-year-old man presents with his left eyelid closing and dribbling from the side of his mouth. This arose three weeks ago and he has also felt tired and fatigued. He denies any fevers and has no weakness in any other parts of his body. He has a dry cough which comes and goes and he put down to hay fever. His left ear has reduced hearing and has been sore.

In addition, there is a red rash on his legs which has now faded. He has no past medical history, has not travelled abroad and is regularly sexually active with a single male partner. He has had shingles three months ago.

On examination, he has a clear left sided facial droop with normal sensation and no sparing of the forehead. He has normal power in all four limbs with no sensory loss. There is a faint red circle on the right leg surrounding a central red area within. Scattered crepitations are present in the lungs. He has pain in his left ear with an effusion behind the tympanic membrane.

Hb	156 g/l	Na +	135 mmol/l
Platelets	345 * 10 /l 9	K +	4.5 mmol/l
WBC	12 * 10 /l 9	Urea	4.2 mmol/l
Neuts	9 * 10 /l 9	Creatinine	94 µmol/l
Lymphs	1 * 10 /l 9	CRP	34 mg/l
Eosin	0.2 * 10 /l9		
Corrected Calcium	2.2mmol/l		

ECG	sinus, PR interval 202ms
Chest x-ray	no abnormalities seen

What is the likely diagnosis?

A. Ramsay hunt syndrome
B. Lyme disease
C. Sarcoidosis
D. Bell's palsy
E. Syphilis

B. Lyme disease

Lyme's disease can present with a facial nerve palsy

This gentleman has a facial nerve palsy without sparing of the forehead. Ramsay hunt syndrome occurs following shingles in the facial nerve and has vesicles in the ear canal but this is visible and it does not cause an effusion. Sarcoidosis is possible but the calcium is normal and there is no hilar lymphadenopathy. Bell's palsy is idiopathic or even possibly viral and should be diagnosed once all other causes are excluded. Syphilis is possible and can have a rash however the rash here is described as a red circle with a central part which should prompt thoughts of erythema migrans in Lyme disease.

Question 114 of 277

You are working in the liver clinic. A 28-year-old pregnant lady attends the clinic. She has been diagnosed with hepatitis C. She has no other co-morbidities and is not taking any regular medications. Hepatitis B and HIV have been excluded. She asks you about the risk to her baby.

What is the risk of vertical transmission?

A. 30%
B. 6%
C. 80%
D. 50%
E. No risk

B. 6%

Risk of vertical transmission for hepatitis C is 6%

Risk of vertical transmission with hepatitis C is generally low and up to 6%. The risk of transmission appears to be high in women with HIV co-infection or high viral load.

Question 115 of 277

A 55-year-old patient is admitted to the acute medical unit with a 10-day history of a dry cough and progressive shortness of breath, particularly on exertion. He also complains of intermittent fevers and a general loss of appetite. His past medical history includes HIV and he is noncompliant with his regular medications.

His observations are as follows:

- Temperature 38.3ºC
- Heart rate 112 beats/min
- Blood pressure 123/84mmHg
- Respiratory rate 25 breaths/min
- Oxygen saturations 94% on air, 83% on exertion

On examination, he appears pale and cachectic. There are audible crackles bilaterally. His heart rate is regular and heart sounds are normal. He has a soft and non-tender abdomen.

Laboratory tests:

CD4 count	< 200/mm³

Which of the following complications is most commonly associated with the likely underlying diagnosis?

A. Encephalitis
B. Hepatitis
C. Pancreatitis
D. Pleural effusion
E. Pneumothorax

E. Pneumothorax

Pneumocystis jiroveci pneumonia - pneumothorax is a common complication

This patient has a diagnosis of pneumocystis jirovecii pneumonia (or PCP), a fungal pneumonia that is most commonly seen in immunocompromised individuals, particularly those with HIV. Symptoms of infection include fever, dry cough and shortness of breath (especially on exertion) as

seen in this patient. Given his lack of compliance with his medications, most importantly the highly active antiretroviral therapy, combined with the CD4 count < 200/mm³, this patient is at high risk of developing PCP. The most common complication associated with PCP is a pneumothorax secondary to rupture of pulmonary cysts.

Extrapulmonary manifestations of PCP include hepatosplenomegaly and lymphadenopathy. However, these are rare and less likely. Encephalitis is a complication of several viral infections including measles, herpes simplex and varicella-zoster virus. It is not a complication of PCP.

Hepatitis has multiple causes such as inflammatory, ischaemic and infective (e.g. hepatitis A,B,C,D and E). PCP is not a cause of hepatitis.

Pancreatitis is a complication of mumps and coxsackie B virus, but not PCP.

Along with pneumothorax, other respiratory complications of PCP include acute respiratory distress syndrome. However, a pleural effusion is a less likely complication compared to a pneumothorax, making it incorrect.

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A 28-year-old man presents with cough and fevers. He states that the cough has been going on for approximately 6 months. He states that there is occasional blood in the sputum. He has also lost approximately 10 kg over the past 2 months. He immigrated to the UK from India approximately 1 year ago. He is not aware of being in contact with anyone with tuberculosis.

You suspect a diagnosis of tuberculosis and isolate him into a negative pressure room.

What investigation is most likely to yield a positive diagnosis?

A. Bronchoalveolar lavage (BAL) with staining, culture and sensitivity
B. Induced sputum with staining, culture and sensitivity
C. Interferon-Gamma Release Assays
D. Mantoux test
E. Chest x-ray

A. Bronchoalveolar lavage (BAL) with staining, culture and sensitivity

In order to confirm the diagnosis of TB, it is necessary to culture *Mycobacterium tuberculosis* from a respiratory specimen. Bronchoscopy will produce the best respiratory specimen for acid-fast staining and mycobacterial culture

Patients who are suspected to have pulmonary tuberculosis should have multiple respiratory samples (3 deep cough sputum samples, preferably including 1 early morning sample) for TB microscopy and culture. Patients that are unable to produce sputum spontaneously or have smear-negative sputum present a diagnostic challenge. NICE recommend that clinicians should perform induction of sputum or bronchoscopy and lavage in adults in this situation.

The investigation with the greatest diagnostic yield is bronchoscopy with lavage making this the best answer.

The Mantoux and Interferon-Gamma Release Assays tests are useful for diagnosing latent TB infection.

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A 38 year-old intravenous drug user presented to accident and emergency with a 10-day history of severe watery diarrhoea and abdominal cramps. Her past medical history included treatment for a groin abscess 3 years previously. She could not remember ingesting any possible causative food and had not travelled abroad. She smoked 15 cigarettes per day and drank 30-40 units of alcohol per week.

On examination, her temperature was 36.8ºC, heart rate 100 beats per minute, blood pressure 92/58 mmHg, respiratory rate 18 breaths per minute and oxygen saturations 100% on room air. Her tongue appeared dry with white patches evident on the hard palate and throat. The JVP was not visible. Her chest was clear on auscultation and heart sounds were normal. The abdomen was soft on palpation with some mild central abdominal tenderness.

Investigations:

Haemoglobin	170 g/L
White cell count	4.5 * 10 /l9
Neutrophil count	3.0 * 10 /l9
Lymphocyte count	0.1 * 10 /l9
Eosinophil count	0.4 * 10 /l9
Platelets	423 * 10 /l9

Sodium	132 mmol/l
Potassium	3.0 mmol/L
Urea	13.0 mmol/L
Creatinine	110 mol/L
Alkaline phosphatase	57 IU/L
Alanine aminotransferase	60 IU/L
Gamma-glutyl transferase	67 IU/L
Bilirubin	19 mol/L
Albumin	29 g/L

Abdominal x-ray: No abnormality detected Stool culture: No growth at 48 hours

What additional staining technique should be used for analysing the stool sample?

A. Ziehl-Neelson
B. Romanowsky
C. Periodic acid-Schiff
D. Sudan
E. Silver

A. Ziehl-Neelson

This patient is likely to have *Cryptosporidium parvum* infection. In the immunocompetent, the protozoan causes a self-limiting diarrhoeal illness lasting 7-10 days or may even be asymptomatic. However, in this case there is likely to be co-existing immunosuppression from human immunodeficiency virus infection, which can result in a severe cholera-like diarrhoea syndrome. The most appropriate staining technique in this case is a modified acid-fast stain such as ZiehlNeelson, which will stain *C. parvum* oocysts red. Periodic acid-schiff is used on jejunal biopsy specimens to diagnose Whipples disease. Silver staining can pick up *Pneumocystis jiroveci* on sputum samples. Sudan stain is used to diagnose steatorrhoea, and Romanowsky is a blood staining method which preceded Giemsa staining.

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A 23-year-old man attends the emergency department after arriving back in the UK from a oneyear trip travelling in South America. He denies any acute medical problems but reports that he is concerned about a dog bite he had received in Brazil about three months previously. The patient had been on a rain-forest expedition when a dog from the village he was staying in became aggressive and bit him on the left hand, leaving a open wound. The local inhabitants of the village had then chased the dog away. The patient had received first aid from his tour guide and been provided with a course of oral antibiotics. The wound had then healed over the following two weeks. It was only when the patient had been speaking with fellow travellers shortly before he returned to the UK did he realise the possible risk for rabies exposure.

The patient was well in himself and denied any neurological or other symptoms. He had no significant past medical history and took no regular medications. He admitted that he had no adequately researched his travel health needs before leaving the UK and so had not had any vaccinations prior to his trip.

Examination of the patients wound demonstrated fully healed scars consistent with a bite marks across the palm and dorsal surface of the wrist. The limb had normal neurological and vascular examination and the patient reported normal function of the hand.

What is the correct statement regarding rabies post-exposure prophylaxis for this patient?

A. The patient should receive rabies vaccination but not rabies immunoglobulin as his wound has now healed
B. The patient does not require post-exposure prophylaxis as he remains free of symptomsfollowing the incubation period of rabies
C. The patient should receive rabies immunoglobulin and full rabies vaccination schedule
D. The patient does not require rabies post-exposure prophylaxis as he did not receive hisinjury in a high-risk region
E. The patient should receive rabies immunoglobulin but not rabies vaccination as this isonly of benefit if given pre-exposure

C. The patient should receive rabies immunoglobulin and full rabies vaccination schedule

The patient requires immediate (if belated) rabies post-exposure prophylaxis. Penetrating wounds are considered high risk-exposures, especially those to highly innervated areas such as the hands. The history of an unprovoked animal biting is also of concern.

Rabies immunoglobulin should be given no matter how long has elapsed since the exposure. As much as possible of the immunoglobulin dose should be infiltrated around the wound, with the remainder given as an IM gluteal injection. This should be combined with a full course of rabies vaccination (typically a 5-dose schedule with doses at day 0, 3, 7, 14 and 28). Such post-exposure prophylaxis is highly effective in preventing the rabies virus reaching the nervous system.

Large parts of South America and Central America (including Brazil) are considered high-risk rabies regions. The virus is also endemic in Africa, the Middle East, Eastern Europe and most of Asia.

The incubation period of rabies can be prolonged and the patient is certainly still at risk of contracting the disease. One study found a mean incubation time of 274 days (range 12 days to 10 years).

Question 119 of 277

A 35-year-old woman attends gastroenterology clinic following a referral from her general practitioner. The patient is suffering from ongoing gastrointestinal symptoms following a recent diagnosis of giardiasis, despite receiving appropriate first line treatment. The patient reports a 3- month history of variable bowel habit, consisting of periods of normal motions interspersed with episodes of frequent diarrhoea. During the course of her illness, the patient reports having lost approximately 7 kg in weight. The patient denied every experiencing fever or bloody diarrhoea, but did report unusual episodes of burping with an unpleasant taste, described by the patient as tasting like 'rotten eggs'.

After her initial symptoms, the patient attended her GP, with a subsequent stool microscopy test demonstrating evidence of infection with *Giardia lamblia*. The patient had not undertaken any recent overseas travel but was employed as a nursery nurse, which was suspected of being the source of her infection. Her employer had moved the patient to a different job role, not including direct contact with children after the patient disclosed her condition.

After the diagnosis of giardiasis, the patient was treated with a 5-day course of metronidazole consistent with British National Formulary guidelines. Following treatment, the patient reported some improvement but had not gained full resolution of her symptoms.

The patient was normally fit and well and her only significant past medical history was a salpingectomy performed 5 years previously due to an ectopic pregnancy. She had never previously experienced problematic gastrointestinal symptoms. The patient took no regular medications and had no known drug allergies.

Examination of the patient's abdomen was unremarkable and she appeared to be in generally good physical condition. A urinary pregnancy test was negative.

Following her review in the clinic, a decision was made to prescribe the patient a single dose of tinidazole as a second-line treatment for giardiasis.

What advice should be given to the patient alongside this treatment?

A. Avoid consuming legumes for 2-6 weeks
B. Avoid consuming dairy products for 2-6 weeks
C. Avoid consuming eggs for 2-6 weeks
D. Her ongoing symptoms are likely secondary to irritable bowel syndrome not giardiasis
E. Avoid consuming gluten for 2-6 weeks

B. Avoid consuming dairy products for 2-6 weeks

The patient has typical symptoms of giardiasis. While giardiasis is the most common protozoal infection in returning travellers, research suggests that the majority of cases in the UK are acquired through domestic exposure such as child-care, sexual contact or consumption of unprocessed foods.

Following unsuccessful first line treatment, a repeat course of antibiotic therapy (either metronidazole or tinidazole) should be given. The patient has already taken steps to avoid reinfection by temporarily changing her job role away from direct contact with children. Giardiasis can cause temporary lactose intolerance, which can lead to ongoing gastrointestinal symptoms after the infection has been eradicated. To exclude this possibility, patients should be advised to avoid dairy products for 2-6 weeks.

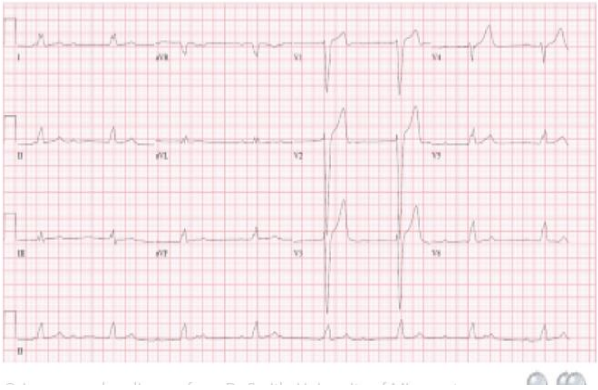
There is an overlap between the symptoms of irritable bowel syndrome and giardiasis. Therefore, in an individual with symptoms suggestive of irritable bowel syndrome but with risk factors for *Giardia* infection, faecal testing for ova and parasites should be considered to exclude giardiasis. However, in this patient, the absence of gastrointestinal symptoms prior to the current episode would make a diagnosis of irritable bowel syndrome unlikely.

Question 120 of 277

A 31-year-old man presents to the emergency department with an episode of collapse. He reports that for the past week, he has been having fevers, headaches, and generally feels faint. He has recently returned from a two-month holiday to the North-Eastern coast of the United States of America (USA). He has no past medical history.

Blood results are as follows:

Hb	141 g/L	135-180
Platelets	231 * 10 /L ^{9}	(150 - 400)
WBC	19.3 * 10 /L ^{9}	(4.0 - 11.0)
Na $^{+}$	138 mmol/L	(135 - 145)
K $^{+}$	4.1 mmol/L	(3.5 - 5.0)
Urea	5.1 mmol/L	(2.0 - 7.0)
Creatinine	91 µmol/L	(55 - 120)
CRP	74 mg/L	(< 5)



Which infectious cause is most likely to be responsible?

A. American trypanosomiasis
B. Coxsackie A
C. Coxsackie B
D. Group A streptococcus
E. Lyme disease

E. Lyme disease

This man's ECG shows a complete heart block with complete dissociation of the QRS complexes from the p waves. The most common infectious cause for this is Lyme disease which is the correct answer here. Ticks that transmit Lyme disease can be found in the United Kingdom as well as the USA.

American trypanosomiasis causes Chagas' disease which can manifest as myocarditis and, ultimately, arrhythmias including complete atrioventricular block. Despite the name, this disease is mainly found in Southern and Central America, whereas the main source of transmission throughout the USA is via immigration and blood transfusions.

Coxsackie A is not associated with ECG changes and therefore this answer is incorrect.

Coxsackie B can cause viral myocarditis for which the most common ECG change is sinus tachycardia. Fulminant Coxsackie myocarditis can result in complete heart block however this would likely present with gastrointestinal symptoms and more severe signs of cardiogenic shock.

Group A streptococci can cause rheumatic fever which in turn can cause first-degree heart block however complete heart block is a far rarer observation.

Question 121 of 277

A 28 year-old man presents to his doctor with a rash and bone pain. Radiographs of the limbs revealed numerous osteolytic lesions. He was successfully treated for secondary syphilis.

Which of the following tests is likely to remain positive in this patient despite treatment?

A. Blood culture
B. Treponema pallidum particle agglutination (TPPA)
C. Venereal disease reference laboratory (VDRL)
D. Rapid plasmin reagin (RPR)
E. Wasserman test

B. Treponema pallidum particle agglutination (TPPA)

Syphilis is a disease caused by infection with the spirochete Treponema pallidum. The infection is systemic and the disease is characterized by periods of latency. These features, together with the fact that Treponema pallidum cannot be isolated in culture, mean that serologic techniques play a major role in the diagnosis and follow-up of treatment for syphilis.

The Treponema pallidum particle agglutination assay (also called TPPA test) is an indirect agglutination assay used for detection and titration of antibodies against Treponema pallidum.

IgG antibodies to syphilis can remain elevated despite appropriate antimicrobial treatment and a reactive result does not distinguish between recent or past infection.

Question 122 of 277

A 20-year-old woman who is 16 weeks pregnant presents with pain passing urine and an irritating rash. On examination, she has a tender, red, vesicular rash on her vulva. A urine dipstick shows both blood and white cells.

What is the best treatment?

A. Clotrimazole
B. Cefalexin
C. Oral aciclovir
D. Topical aciclovir
E. Fluconazole

C. Oral aciclovir

This patient has genital herpes simplex virus (HSV). The guidelines recommend treatment with oral (or intravenous) aciclovir at any stage in pregnancy. Aciclovir is not licensed in pregnancy but is considered safe and not associated with birth defects. It is well tolerated in pregnancy. Paracetamol and topical lidocaine 2% gel can be used for symptomatic relief.

The primary purpose of treatment is to reduce the risk of transmission to the neonate at birth. The risk is much more considerable with primary genital herpes simplex within the final six weeks of pregnancy. Caesarian section should be the recommended mode of delivery for all women developing the first episode of genital HSV in the third trimester.

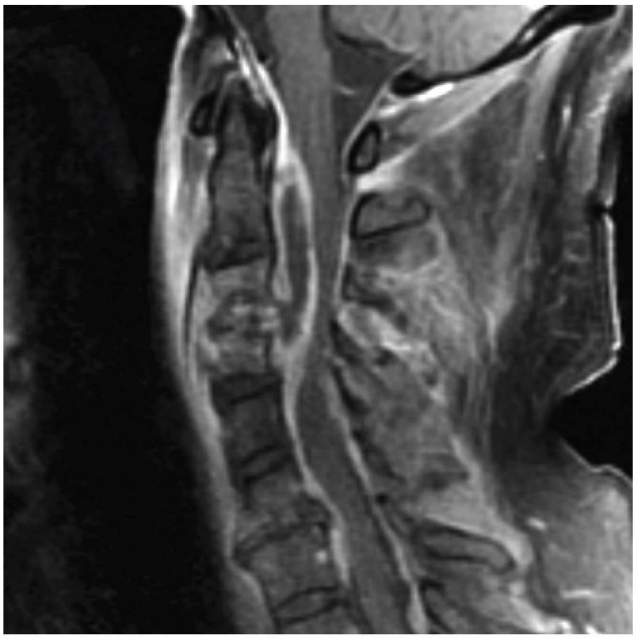
Question 123 of 277

A 54-year-old man presents with neck pain and feeling generally unwell. This has been getting progressively worse over the past two weeks and is now 'unbearable'. He feels hot and also complains of headaches.

He emigrated from Pakistan 30 years ago. He smokes 20 cigarattes/day and does not drink alcohol.

On examination pulse is 102/min, blood pressure 124/74 mmHg and temperature 37.9ºC. He has weakness in both arms

MRI of his cervical spine is shown below:



What is the most likely diagnosis?

A. Cervical disc prolapse
B. Syringomyelia
C. Cervical epidural abscess
D. Meningitis
E. Tuberculosis

C. Cervical epidural abscess

MRI demonstrates an epidural collection with peripheral contrast enhancement. The cord is displaced posteriorly and to the right. Features are consistent with an epidural abscess. There are associated changes at the C3/4 level consistent with advanced discitis osteomyelitis.

Question 124 of 277

A 29-year-old woman who is a former intravenous drug user is seen in the hepatology clinic. She is 12 weeks pregnant and has been referred as she is known to have chronic hepatitis C. Twelve months ago she was treated with pegylated interferon-alpha, ribavirin and a protease inhibitor which failed to result in her clearing the hepatitis C virus.

What is the most appropriate way, if any, to reduce the risk of vertical transmission?

A. Caesarean section
B. Further course of antiviral therapy during pregnancy
C. Advise against breastfeeding
D. Antiviral therapy for the neonate for the first 4 weeks
E. None of the above interventions is recommended

E. None of the above interventions is recommended

Question 125 of 277

A 45-year-old female presents with a history of headache and generalised malaise for the last two months. She describes her headaches as being a tight band like sensation which is present almost throughout the day and causes significant difficulty in sleeping at night. She also mentions occasional episodes of vomiting along with low-grade fever and weight loss of about 7 kg over the same duration of time. She suffers from generalised anxiety disorder and takes 0.5mg alprazolam TDS. She returned from Dubai 9 months ago where she had been spending her holidays with her family.

On examination, she has a fever of 37.5°C and a pulse of 105bpm. She appears slightly disoriented with a tendency to speak out of context but is otherwise cooperative.

There is diplopia on right sided gaze and mild neck stiffness, but the remaining clinical examination is essentially unremarkable.

Lab reports reveal:

Hb	115 g/l
Platelets	340 * 10 /l9
WBC	9.0 * 10 /l9

Na +	137 mmol/l
K +	4.2 mmol/l
Urea	5.9 mmol/l
Creatinine	102 µmol/l
Glucose	7.0 mmol/l
ESR	87 mm/hr

MRI shows meningeal enhancement but no evidence of any parenchymal lesions. CSF examination reveals:

Opening Pressure	Normal
Appearance	Turbid
Protein	3.2g/L (0.2 0.4 g/L)
Glucose	2.7 mmol/l
Lymphocytes	$371/mm^3$
Neutrophils	$42/mm^3$
ZN staining	No acid fast bacilli detected

Which of the following is the most appropriate treatment option?

A. IV acyclovir
B. IV ceftriaxone
C. Isoniazid, rifampicin, pyrazinamide and dexamethasone
D. IV benzylpenicillin
E. Artemether/lumefantrine

C. Isoniazid, rifampicin, pyrazinamide and dexamethasone

The 2-month history of headache, generalised malaise, low-grade fever and weight loss all go in favour of a diagnosis of tuberculous meningitis. This diagnosis is also supported by the examination findings of diplopia neck stiffness.

The complete blood count does not favour a bacterial infection as one would expect a leucocytosis. The raised ESR is also supportive of TBM.

The CSF shows a predominant lymphocytosis with greatly raised protein and significantly low glucose which once again goes with tuberculosis.

TBM typically commences with symptoms of a vague headache, lassitude, anorexia and vomiting. Acute meningitis may occur but it is uncommon. The development of meningitic signs often takes weeks. Common signs include diplopia, papilloedema and hemiparesis and seizures are also often commonly seen. Treatment is generally commenced on a presumptive basis since multiple CSF cultures are often required and it may take weeks before culture reports are available. Staining with ZN staining is often negative and PCR for mycobacterium tuberculosis is helpful but is often negative too and repeat PCR is often needed.

Treatment is with isoniazid, rifampicin, pyrazinamide and steroids. Ethambutol should be avoided due to eye complications.

Question 126 of 277

A 44-year-old woman with a background of asthma who has required frequent use of oral corticosteroids is inquiring about vaccinations available to her.

Which one of the following should be recommended?

A. 23-valent unconjugated pneumococcal polysaccharide vaccine and influenza vaccine
B. 13-valent conjugated pneumococcal polysaccharide vaccine only
C. 23-valent unconjugated pneumococcal polysaccharide vaccine only
D. 13-valent conjugated pneumococcal polysaccharide vaccine and influenza vaccine
E. Influenza vaccine only

A. 23-valent unconjugated pneumococcal polysaccharide vaccine and influenza vaccine

Patients with chronic respiratory conditions including asthma requiring frequent use of oral steroids should be offered both the 23-valent unconjugated pneumococcal polysaccharide vaccine and the influenza vaccine. In this population, the pneumococcal vaccine is usually given as a once off but may be required 5 yearly depending on the underlying condition and the age of the patient. The influenza vaccine, however, is given annually.

The 13-valent conjugated pneumococcal polysaccharide vaccine is routinely given to infants as it has been found to be more effective given their less mature immune systems.

Question 127 of 277

A 39-year-old female with end-stage renal failure secondary to IgA nephropathy is admitted to hospital. She was discharged 3 days earlier following an admission due to collapse in the context of hyperkalaemia (Potassium 7.8 mmol/l). She has been transferred to the medical assessment unit having developed rigors on dialysis earlier today.

Bloods taken at the end of dialysis reveal:

White cell count	15.2 *109/l
Sodium	134 mmol/l
Potassium	2.1 mmol/l
Urea	10.6 mmol/l
Creatinine	400 µmol/l
C-reactive protein (CRP)	119 mg/dL

What is the immediate priority in her management?

A. Replace potassium (K+)
B. Replace magnesium (Mg2+)
C. Intravenous antibiotics
D. Intravenous calcium gluconate
E. Insulin/dextrose infusion

C. Intravenous antibiotics

The patient is septic and infection is her most imminent threat. Post-dialysis bloods are often misleading in terms of electrolytes as it takes 30 minutes for intracellular and extracellular compartments to equilibrate. Assuming that the patients pre-dialysis bloods showed a highnormal K+ (which would fit with her recent hyperkalaemia presentation) then her total body K+ will be much higher than the post-dialysis sample suggests. The bloods should be repeated to confirm this but over-zealous correction of K+ in this situation can be dangerous.

Magnesium is important to correct in the presence of hypokalaemia, however, we do not think this patient genuinely has low potassium. Intravenous calcium gluconate and insulin/dextrose infusion are both treatments for hyperkalaemia and therefore are not appropriate.

Question 128 of 277

A 23-year-old student presented to his GP with fever and epistaxis. The fever had started 6 days earlier. It had been associated with a headache and malaise. Following clinical examination his doctor prescribed him amoxicillin which he took for 3 days but without any improvement. He later became so ill that he refused to eat and started to vomit.

His history included mumps at the age of nine. His father had been diagnosed with liver cirrhosis 2 years ago due to excessive alcohol consumption. He is a regular smoker for the last 10 years and lives in the dorm. Two weeks earlier he had been in rural Central Africa as a reporter covering the conflict there. He denied being vaccinated for any disease before going there but did take antimalarial tablets.

On examination he appeared ill. His temperature was 38ºC. He was jaundiced. Eye examination revealed conjunctival hemorrhages on both eyes. All other systems were normal.

The following investigations had been requested:

Hb	13 g/dl
platelets	170 * 10^9/l
WBC	4 * 10^9/l
MCV	85 fl
MCH	0.4 fmol/cell
MCHC	20 mmol/l
Na+	135 mmol/l
K+	4 mmol/l
Creatinine	80 µmol/l
Urea	3 mmol/l
ESR	60 mm/hr
Alkaline phosphatase	100 IU/l
Alanine transaminase	400 IU/l
Aspartate transaminase	200 IU/l
Bilirubin	25 µmol/l (direct 18 umol/l)
Serum albumin	40 g/l
Prothrombin time	prolonged
Partial thrompoblastin time	prolonged
Urine analysis	Albumin + ,acetone ++ ,bile pigment ++,urinary urobilinogen +

Thick Blood film for malaria	negative

What is the most likely diagnosis?

A. Yellow fever
B. Primary sclerosing cholangitis
C. Leptospirosis
D. Falciparum malaria
E. Dengue fever

A. Yellow fever

Yellow fever is endemic around the equator mainly in Africa. It is transmitted by the Aedes mosquitoes there. Characteristically the patient presents with fever and constitutional symptoms. Jaundice is a main clinical sign associated most of the times with conjunctival injection. Other important clinical signs are relative bradycardia, and hemorrhagic diathesis. The cause of death is due to multi-organ failure.

Blood tests generally show leucopenia with predominant neutropenia but this is not specific. Liver enzymes are elevated most of the time. Diagnosis depends on a high degree of suspicion for the disease and but also more important on serology which is highly specific for this lethal condition.

Treatment is mainly supportive.

Primary sclerosing cholangitis could present with fever and jaundice. This would happen in the context of ascending cholangitis (fever + pain + jaundice) with a long history of abdominal discomfort. Liver profile would reflect obstructive changes.

Malaria should be excluded in any patient who had traveled to an endemic area and developed fever. The picture in our patient was not typical and the blood film had been negative for malaria parasites. However one has to keep it in mind if the clinical picture fits. In that case one needs to repeat the blood film and to order serology.

Leptospirosis is a consideration here. But it usually responds to antibiotics. Liver enzymes would generally be more mildly elevated.

Dengue fever mainly presents with headache, fever and rash. It is mainly endemic in South East Asia.

Question 129 of 277

A 60 year old male who is a malnourished alcoholic presents with a chronic cough for the past 6 weeks associated with a low grade fever. The cough is productive of purulent sputum.

Six months previously he had been diagnosed with early stage non-Hodgkin's lymphoma, which had responded well to chemotherapy (doxorubicin, bleomycin, vinblastine, and prednisolone).

On examination his temperature is 37.8ºC, blood pressure 140/80 mmHg, and his pulse is 96/minute and regular. Auscultation of the chest reveals absence of breath sounds over the left middle lung field. Chest x-ray confirms left upper lobar consolidation.

The following investigations were ordered:

Hb	12 g/dl
Platelets	180 * 10^9/l
WBC	7 * 10^9/l
MCV	85 fl
Na+	140 mmol/l
K+	5 mmol/l
Creatinine	90 µmol/l
Urea	5 mmol/l
CRP	50 mg/l

Sputum stains partially acid fast bacilli with branching rods

What is the most suitable initial management of this patient?

A. Metronidazole + ampicillin
B. Clarithromycin
C. Ceftriaxone + clarithromycin
D. Trimethoprim/sulfamethoxazole + amikacin + ceftriaxone
E. Isoniazid + rifampin + pyrazinamide + ethambutol

D. Trimethoprim/sulfamethoxazole + amikacin + ceftriaxone

Nocardiosis typically occurs in immunocompromised patients with pulmonary involvement and in most cases with CNS or skin dissemination.

The clue here is the acid-fast stain which shows partially acid fast branching rods organism in contrast to mycobacterium which does fully stain.

Treatment of immunocompromised patients consists of multiple regimens. But mainly all of them involve Trimethoprim/sulfamethoxazole.

Nocardia species are ubiquitous soil organisms that often infect patients who are immunosuppressed, have pulmonary disease, or have a history of surgery or trauma. It is a Gram positive, acid fast bacilli.

Question 130 of 277

A 27 year old Caucasian man has just returned from a 12 week trip to Ivory Coast where he was working as a missionary. He had not consulted a travel clinic for immunisations and had taken no malaria prophylaxis. During his trip he slept in a hammock outside and he took a daily wash in the river near the church where he was working. For the last few days of his trip he felt very unwell. He developed fevers, muscle aches and a headache, however this was improving and he had been feeling much better when he arrived home in the UK.

He has been home for 24 hours. Today he felt very much worse and presented to A and E.

On admission:

- Temperature: 39.1C
- Blood pressure: 89/61mmHg
- Pulse: 68 / min
- Respiratory rate: 26 / min
- Oxygen saturations: 97% on room air.

His initial investigations show:

Hb	11.1 g/dl
Platelets	61 * 10 /l9
WBC	3.3 * 10 /l9
Neutrophils	2.6 * 10 /l9
Lymphocytes	0.6 * 10 /l9

Na +	146 mmol/l
K +	4.1 mmol/l
Bicarbonate	16 mmol/l
Urea	17.4 mmol/l
Creatinine	321 mol/l

Bilirubin	88 mol/l
ALP	322 u/l
ALT	821 u/l
γGT	421 u/l
Albumin	29 g/l

Following discussion with the ID consultant on call, he is isolated while a blood sample is sent to Public Health Englands imported fever service. He received empirical therapy for Malaria with Artesunate and for sepsis with Piperacillin/Tazobactam and Gentamicin, however he rapidly deteriorated and died within 24 hours of admission. Following discussion with Public Health England, who confirm that there was no evidence of a nosocomially transmitted Viral Haemorrhagic Fever, he has a post mortem examination which reveals Councilman bodies in his liver.

What would likely have prevented his death?

A. Sleeping under an insecticide treated bed net
B. Avoiding bathing in the river
C. Live attenuated Yellow Fever vaccine
D. Prophylaxis with doxycycline
E. Avoiding unprotected sex

C. Live attenuated Yellow Fever vaccine

This is yellow fever as suggested by:

- Travel to endemic area (West Africa and Central America)
- Fever, with initial resolution
- Progression to jaundice and renal failure

For exam purposes Councilman bodies (eosinophilic inclusion in the liver on post mortem) are diagnostic of yellow fever, although they can occasionally be seen in other Viral Haemorrhagic Fevers such as Crimean Congo Haemorrhagic Fever, (but this is nosocomially spread and therefore not the case in this example.

As such the vaccination is the only intervention which could have prevented his death.

Question 131 of 277

A 37 year old Caucasian male attends an outpatient clinic. He had recently spent three months in Jamaica on a beach-side yoga retreat. He only drank bottled water and ate well cooked vegetarian food. He ensured that any raw vegetables were peeled. He returned home three weeks ago.

During his final week in Jamaica he noticed an itchy spot develop on the side of his ankle. The rash has gradually spread from the original spot slowly 'like a snake up the side of his foot'. The lesion is currently 11cm long and is a raised itchy serpiginous linear lesion.

He is otherwise well.

What is the causative organism?

A. Strongyloides stercoralis
B. Ancylostoma braziliense
C. Necator americanus
D. Ascaris lumbricoides
E. Schistosomiasis mansoni \

B. Ancylostoma braziliense

The correct answer in this case is ***Ancylostoma braziliense***. The patient's presentation of a raised, itchy serpiginous linear lesion that has gradually spread 'like a snake' up the side of his foot is characteristic of cutaneous larva migrans (CLM), also known as creeping eruption. CLM is caused by the larval form of hookworms, most commonly *Ancylostoma braziliense*, which are found in tropical and subtropical regions such as Jamaica. The larvae penetrate the skin, usually through contact with contaminated soil or sand, and migrate within the epidermis, causing an intensely pruritic inflammatory reaction.

Strongyloides stercoralis is an incorrect option because this nematode causes strongyloidiasis, which typically presents with gastrointestinal symptoms such as abdominal pain and diarrhea. Skin involvement in strongyloidiasis can occur but usually manifests as pruritic papules or urticaria rather than the serpiginous linear lesion seen in this case.

Necator americanus is another species of hookworm, but it primarily causes human infection through intestinal penetration rather than cutaneous manifestations like CLM. Infection with *Necator americanus* can lead to iron-deficiency anemia due to blood loss from attachment sites in the small intestine.

Ascaris lumbricoides is a roundworm that causes ascariasis, one of the most common helminthic infections worldwide. Ascariasis typically presents with gastrointestinal symptoms such as abdominal pain and bloating; however, during the larval migration phase through the lungs, patients may experience coughing and wheezing. Cutaneous manifestations are not typical in ascariasis.

Schistosomiasis mansoni is a blood fluke that causes schistosomiasis, which is characterized by granulomatous inflammation in response to the eggs of the parasite. The primary manifestation of schistosomiasis is hepatosplenic involvement, with symptoms such as abdominal pain and hepatomegaly. Cutaneous manifestations can occur during the cercarial penetration phase (swimmer's itch), but this is typically a localized pruritic papular rash rather than the serpiginous linear lesion seen in this case.

Question 132 of 277

A 24-year-old Somali woman attends her booking appointment in the UK for her first pregnancy. Screening tests reveal that she is HIV positive. She is asymptomatic.

Her viral load is 150 000 copies/ml and her CD4 count is 523 cells/mm³. No viral resistance is detected. Her hepatitis serology is negative. Her husband consequently tests negative for HIV.

She is commenced on triple antiretroviral therapy (ART) with zidovudine, lamivudine and lopinavir/ritonavir. By 36 weeks her viral load is undetectable at <20 copies/ml.

Which of the following statements is true regarding her ongoing management?

A. She should have an elective caesarean section and continue to take her ART whilst breastfeeding.
B. She should have a vaginal delivery and continue to take ART whilst breast feeding.
C. She should have an elective caesarean section. ART should be continued.
D. She should have a vaginal delivery and formula feed. ART can be discontinued.
E. She should have a vaginal delivery and formula feed. ART should be continued.

E. She should have a vaginal delivery and formula feed. ART should be continued.

In this case, as her viral load is less than 50 copies/ml she may have a vaginal delivery. Formula feeding is recommended in the UK to all babies with HIV +ve mothers. In this case although her CD4 count is >350 cells/mm³ her ART should be continued to reduce the risk of onward transmission to her husband.

Question 133 of 277

A 48-year-old woman attends with a one-week history of a productive cough and shortness of breath. She has never smoked and has no past medical history. Observations are as follows: heart rate 105 beats per minute, blood pressure 125/70 mmHg, temperature 38.4ºC, respiratory rate 20 breaths per minute, oxygen saturation 92% on air.

Blood results are as follows:

Hb	122 g/L	Male: (135-180)Female: (115 - 160)
Platelets	246 * 10 /L 9	(150 - 400)
WBC	14.2 * 10 /L 9	(4.0 - 11.0)
Na +	128 mmol/L	(135 - 145)
K +	3.8 mmol/L	(3.5 - 5.0)
Urea	7.2 mmol/L	(2.0 - 7.0)
Creatinine	105 µmol/L	(55 - 120)
CRP	96 mg/L	(< 5)

Bilirubin	22 µmol/L	(3 - 17)
ALP	115 u/L	(30 - 100)
ALT	262 u/L	(3 - 40)
Î³GT	114 u/L	(8 - 60)
Albumin	32 g/L	(35 - 50)

A chest x-ray was performed:

Chest x-ray	Bilateral patchy consolidation

What treatment is indicated?

A. Co-amoxiclav
B. Co-trimoxazole
C. Co-trimoxazole and prednisolone
D. Erythromycin
E. Piperacillin/tazobactam

D. Erythromycin

Macrolides such as clarithromycin are used to treat *Legionella*

Erythromycin is correct. The clinical features are suggestive of *Legionella* infection. The patient has an atypical pneumonia with bilateral patchy consolidation, hyponatraemia, and deranged liver function tests, all suggestive of *Legionella* infection. Most guidelines recommend either a fluoroquinolone or a macrolide as first-line treatment for Legionella pneumonia, often with the addition of a penicillin for a synergistic effect.

Co-amoxiclav is incorrect. This antibiotic is effective for typical organisms such as *Streptococci* and *Haemophilus*.

Co-trimoxazole is incorrect. This drug can be used to treat pneumocystis pneumonia (PCP) which is a serious infection caused by the fungus *Pneumocystis jirovecii*. Although this can present similarly, it is a condition which tends to only develop in patients with weak immune systems. The patient in this clinical case is immunocompetent making PCP an unlikely diagnosis.

Co-trimoxazole and prednisolone is incorrect. This regime of drugs can be used to treat severe PCP infections characterised by a PaO2 < 8kPa.

Piperacillin/tazobactam is incorrect. This drug has a similar spectrum to co-amoxiclav with the addition of activity against *Pseudomonas*.

Question 134 of 277

A 29-year-old woman is brought to the emergency department by her husband with drowsiness. Over the last day she had been complaining of headaches, nausea and fevers. About a week ago she had a period where she felt unwell with fever, headache and myalgia, but this self-resolved. She had returned from Poland 2 weeks ago where she had been on a camping holiday with friends. Her past medical history consists only of asthma, for which she takes salbutamol as required.

Examination reveals a Glasgow Coma Scale (GCS) of 14 with neck stiffness but no focal neurology. Her lungs are clear and her abdomen is soft and non-tender. There is no rash.

A lumbar puncture is performed which reveals mildly elevated white blood cells. An HIV test is negative. The patient is then started on ceftriaxone, dexamethasone and aciclovir.

The CSF culture and PCR come back negative. A sample had also been sent away to the infectious disease laboratory, which after serological testing comes back as positive for *flavivirus* antibodies.

Given the most likely diagnosis, what is the most appropriate management?

A. IV amphotericin B
B. IV aciclovir
C. IV pyrimethamine/sulfadiazine and folinic acid
D. IV quinine dihydrochloride
E. Supportive management

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E. Supportive management

Treatment for tick-borne encephalitis is supportive

A presentation of headache, neck stiffness, fever and reduced GCS is suggestive of meningoencephalitis. The question revolves around determining what is the most likely causative agent and therefore the most appropriate treatment.

The history of recent travel should raise your suspicion for a potentially uncommon aetiology. The camping history combined with a biphasic illness is characteristic for tick-borne encephalitis. Ultimately, the finding of a *flavivirus* in the CSF confirms the diagnosis. Management for this condition is supportive.

Amphotericin B may be used in the treatment of primary amoebic meningoencephalitis. This is a very rare cause of meningoencephalitis caused by *naegleria fowleri.*

Aciclovir is used in the management of herpes simplex virus (HSV) encephalitis. HSV, along with varicella-zoster virus and enteroviruses make up 90% of cases of viral encephalitis. In this patient, the travel history and biphasic illness suggest an alternate, atypical cause.

IV pyrimethamine/sulfadiazine and folinic acid are used in the management of cerebral toxoplasmosis. Toxoplasmosis is usually asymptomatic or mild in immunocompetent patients. The patient only has a past medical history of asthma and is HIV negative, therefore this is a less likely diagnosis.

IV quinine dihydrochloride may be used in severe or complicated falciparum malaria. The patient has not come from a malaria-risk country and does not have other features suggestive of malaria, such as jaundice.

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A 40-year-old woman is referred to the infectious diseases team after attending her GP practice requesting advice regarding antibiotic prophylaxis for Lyme disease. The patient explains that she is planning on a 2 week long walking holiday in the Lake District National Park in Cumbria, United Kingdom and that she is concerned about the risk associated with receiving tick bites during this period. Lyme disease is a particular concern for the patient as her younger sister had suffered serious neurological complications from the condition secondary to a tick bite in the Scottish Highlands. Concern about contracting Lyme disease herself had prevented her from partaking in her hobby of cross country walking for the past 2 years but she had now decided to confront her fear by undertaking the planned trip. After undertaking research on the Internet, the patient was keen to receive prophylactic antibiotics against *Borrelia* species during her trip.

The patient's past medical history included a diagnosis of breast cancer seven years previously. Following this diagnosis, the patient had undergone a wide local excision of the tumour with postoperative chemotherapy. The patient had been given the all clear from her oncologists after five years and had been subsequently been discharged from follow-up. In addition, the patient had experienced long-standing and, at times, troubling symptoms of irritable bowel syndrome.

The patient took regular hyoscine butylbromide as treatment of her gastrointestinal symptoms. The patient stated that she was allergic to penicillin-based antibiotics. Close questioning on this topic revealed that her adverse reaction to a previous course of penicillin V had been a protracted episode of diarrhoea.

The patient lives with her husband and three teenage children. She was employed full-time as a music teacher. The patient did not smoke cigarettes and consumed approximately 10 units of alcohol per week.

What is appropriate management following the patient's request for antibiotic prophylaxis against Lyme disease during her walking holiday?

A. Prescribe amoxicillin for the duration of the patient's holiday
B. Prescribe amoxicillin for immediate use if she receives a tick bite
C. Prescribe doxycycline for the duration of the patient's holiday continued for an additional10 days after her return home
D. Advice on the prevention and management of tick bites only
E. Prescribe doxycycline for immediate use if she receives a tick bite

D. Advice on the prevention and management of tick bites only

There is no need for prophylactic antibiotics for Lyme disease in asymptomatic patients bitten by a tick

There is no role for antibiotic prophylaxis against Lyme disease. Simple advice on prevention of tick bites includes wearing clothing that adequately covers the patient's body and the use of insect repellent chemicals. Ticks that have attached themselves should be gently removed using tweezers to minimise the risk of rupturing the tick and potentially releasing infected material.

Individuals showing symptoms of Lyme disease - for example, erythema migrans - following potential exposure to tick bites should be treated with a 14-21 day course of doxycycline or amoxicillin immediately.

Individuals with a history of tick bite but without symptoms of Lyme disease do not normally require either treatment or serological testing. Treatment of asymptomatic individuals who have received a tick bite may be appropriate in certain situations; for example, in individuals who are immunosuppressed or who received tick bites in high-risk regions of the USA.

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A 35-year-old male attends clinic with a new diagnosis of chronic hepatitis B infection. He gives a history of unprotected sex with multiple sexual partners in the two years. His blood tests show him to be HIV, HCV antibody and HBeAg negative. The most recent liver function tests are shown below. On examination, he has no evidence of decompensated liver disease.

Bilirubin	18µmol/L
ALT	52iu/L
AST	38iu/L
ALP	190iu/L
Albumin	30g/L
Protein	93g/L

HBV viral load	2,000IU/mL

Which one of the following treatment options should be considered first line?

A. Peginterferon alpha 2a and entecavir
B. Entecavir
C. Peginterferon alpha 2a
D. Tenofovir disoproxil
E. Entecavir and tenofovir disoproxil

C. Peginterferon alpha 2a

Peginterferon alpha 2a is the first line treatment for adults with HBeAg-negative chronic hepatitis B with compensated liver disease. Tenofovir disoproxil or entecavir are second line treatment in those who have detectable HBV DNA after treatment with peginterferon alpha 2a.

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A 43-year-old gentleman presents with bloody diarrhoea. He is HIV positive and has a CD4 count of 150 cells/µL.

He had been on a trip to Zimbabwe 10 months previously. Over the past 2 weeks, he had symptoms of anorexia and mild abdominal discomfort. These had progressed on to severe abdominal cramps and bloody diarrhoea.

He has a temperature of 37.8ºC, a blood pressure of 100/60 and a pulse of 111/min. On examination, he looks unwell, is emaciated and has generalised tenderness on examination of his abdomen. There is no guarding or peritonitis.

What is the most likely causative organism?

A. Giardia lamblia
B. Toxoplasma gondii
C. Entamoeba histolytica
D. Cytomegalovirus
E. Strongyloides stercoralis

C. Entamoeba histolytica

Entamoeba histolytica is the only pathogen here which typically causes bloody diarrhoea in an immunocompromised individual and hence is the correct answer. Giardia typically causes steatorrhoea, *Toxoplasma gondii* is more likely to cause a central nervous system infection, and while CMV can cause diarrhoea it is rarely a presenting feature of the infection. While *Strongyloides stercoralis* can cause diarrhoea, it is usually watery rather than bloody.

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A 25-year-old woman returns from a holiday in Thailand with diarrhoea. She has no past medical history and takes no regular tablets apart from the oral contraceptive pill. She has no allergies apart from peanuts. She has a been suffering from diarrhoea for three days with mild abdominal cramps which started five days after returning from her trip. There has been a small amount of blood in her stool. Her observations are within normal range. She has no postural symptoms. Stool samples are requested and sent to the microbiology lab.

What is the most appropriate treatment?

A. Ciprofloxacin
B. Tetracycline
C. Oral rehydration solution
D. Metronidazole
E. Co-amoxiclav

C. Oral rehydration solution

The correct answer is oral rehydration solution. Empirical antibiotics are only indicated if systemically unwell, immunosuppressed or if elderly, even if the patient has bloody diarrhoea. CIprofloxacin is used to treat Salmonella, Shigella and Campylobacter, tetracycline can be used for cholera, metronidazole in parasitic infections or other bowel problems, and finally, co-amoxiclav is seldom used in enteric infections.

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A 25-year-old man presents to gastroenterology with a 6-month history of unexplained chronic diarrhoea and unexplained chronic weight loss. An HIV test is sent as part of his work-up and returns positive. His CD4 count is 150. He is started on anti-retroviral therapy.

Round oocysts measuring between 4 and 6 µm are seen during microscopy of the patient's stool sample. They stain red using the modified acid-fast stain. A diagnosis of Cryptosporidium is made. The first-line treatment of nitazoxanide is not available.

What other medication could be a potential treatment option for this patient?

A. Ciprofloxacin
B. Metronidazole
C. Nitrofurantoin
D. Prednisolone
E. Rifaximin

E. Rifaximin

Rifaxamin is licensed for the treatment of cryptosporidiosis

The oocysts described on stool microscopy are typical of *Cryptosporidium*. *Cyclospora* and *Isospora* oocysts will also stain with the modified acid-fast stain. The oocysts of *Cyclospora* look similar to those of *Cryptosporidium* but are larger at 8-10 µm. *Isospora* oocysts are oval shaped and are larger again at 10-20 x 20-30 µm.

Cryptosporidiosis is a protozoal infection that generally causes mild, watery diarrhoea in immunecompetent patients, although symptoms can persist for several weeks. It can cause chronic diarrhoea in immune suppressed patients, especially those with HIV infection. Treatment is not always required as the symptoms often improve once the immune system starts to recover. If treatment is indicated the first line option is nitazoxanide, followed by rifaximin.

Ciprofloxacin can be used for some causes of infectious diarrhoea, such as *Campylobacter* and *Salmonella* but does not treat *Cryptosporidium*.

Metronidazole is effective against anaerobic bacteria and some protozoa (including *Giardia*) but is not active against *Cryptosporidium*.

Nitrofurantoin is mainly used for the treatment of lower urinary tract infections due to Gram negative rods and will not treat *Cryptosporidium*.

Prednisolone may worsen the patient's symptoms by increasing his level of immune suppression so would not be a good treatment option in this case.

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A 38-year-old man from Nigeria presents to a gastroenterology clinic. He is a businessman, working for an iron and steel trading company and makes regular trips to Swaziland to visit mining executives.

For the previous four months, he has been troubled by diarrhoea, up to 8 episodes a day, with abdominal bloating and cramping. There is no blood visible in the stool. He has previously been treated for tuberculosis as a child.

A stool sample is sent to the lab, and Modified Ziehl-Neelson stain reveals multiple red staining round objects measuring 5 microns in diameter.

What will be the most appropriate treatment?

A. Highly active anti-retroviral therapy
B. Co-trimoxazole
C. Rifampicin, isoniazid, ethambutol, pyrazinamide
D. Metronidazole
E. Ciprofloxacin

A. Highly active anti-retroviral therapy

The diagnosis is cryptosporidium infection. It is the most common cause of chronic diarrhoea in HIV patients. Diagnosis is made by visualisation with modified Zeihl-Neelson stain of stool. You would very rarely expect to see *Mycobacterium tuberculosis* on a faecal sample.

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Mrs Stevens is a 60-year-old lady with pulmonary fibrosis secondary to dermatomyositis. She returns to your clinic seven weeks after being commenced on high dose steroids due to disease progression. During the review Mrs Stevens describes increasing shortness of breath, which is worse when she exerts herself. She has not noticed any fever and has not lost any weight. She has a chronic cough productive of white sputum. This remains stable.

On examination she looks comfortable at rest and auscultation of her chest is surprisingly clear. At rest her oxygen Sats are 97%. You ask her to walk to the end of the corridor and back. You notice that on returning her Sats have fallen to 82%. You request an urgent chest radiograph, which is reported as showing bilateral patchy infiltrates.

What is the most appropriate management option?

A. Fluconazole
B. Ambisome
C. Co-trimoxazole
D. Ribavirin
E. Stop steroids

C. Co-trimoxazole

Pneumocystis jiroveci penumonia is treated with co-trimoxazole, which is a mix of trimethoprim and sulfamethoxazole

The patient has PCP pneumonia, caused by *Pneumocystis jirovecii* - a yeast-like fungus which is an opportunistic pathogen. The treatment for this is co-trimoxazole.

All patients with dermatomyositis or polymyositis with pulmonary fibrosis who are treated with glucocorticoids are advised to receive PCP prophylaxis with co-trimoxazole. Stopping steroids would not be appropriate in this scenario.

It is important to note that not all patients taking glucocorticoids for rheumatological conditions require PCP prophylaxis. However, there is evidence that in non-HIV cohorts, the greatest risk for developing PCP is glucocorticoid use and deficiencies in innate and acquired immune components. Therefore, although there are currently no formal guidelines it is recommended that if an individual satisfies both these criteria in rheumatological conditions, they should be considered for PCP prophylaxis.

For example the following all should be considered for prophylaxis:

- A patient on the equivalent of 20 mg prednisolone or greater for one month plus the concomitant use of another immunosuppressive drug
- Dermatomyositis on high dose prednisolone for greater than one month and pulmonary fibrosis
- Allogenic or autogenic stem cell transplant or solid organ transplantation

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A 43-year-old man presents to his local sexual health clinic with an indurated well-circumscribed painless ulcer on the glans of his penis. Further examination demonstrates inguinal lymphadenopathy. He has had unprotected vaginal intercourse with a causal female partner within the last 14 days. He identifies as heterosexual, was born in London. He consents to a full sexual health screening and is prescribed a 14-day course of doxycycline as he reports a previous urticarial rash associated with penicillin.

The following day, the patient returns to the clinic with a high fever and widespread rash. He is concerned that he may have an allergy to doxycycline and would like advice regarding an alternate treatment. He reports no respiratory distress, tongue swelling or headache and consents to a set of monitoring blood tests, the results of which are within normal limits.

Given the likely diagnosis, which of the following would be the most appropriate course of action?

A. Advise the patient that his rash is likely due to sun exposure with concomitant doxycycline use
B. Give a cephalosporin based treatment regime instead
C. Reassure the patient that this reaction with treatment of syphilis and advice paracetamolwith good oral hydration
D. Admit immediately to hospital
E. Take a p24 antigen test and counsel the patient about possible HIV seroconversion

C. Reassure the patient that this reaction with treatment of syphilis and advice paracetamolwith good oral hydration

The Jarisch-Herxheimer reaction is a known phenomenon following syphilis treatment that does not require any specific treatment or investigations other antipyretics

This patient has syphilis and is experiencing a Jarisch-Herxheimer reaction, the mechanism of which remains poorly understood, but is felt to be related to the immune response triggered by the killed organisms.

Doxycycline is the treatment of choice in patients with a penicillin allergy and, given the reported allergy described by the patient, a cephalosporin would not be advised.

The Jarisch-Herxheimer reaction is usually self-limiting and best managed conservatively with antipyretics and good hydration unless there is evidence of organ compromise. Hospital admission is unnecessary unless there is evidence of organ compromise.

HIV seroconversion is possible given the recent unprotected intercourse, but this patient would be considered low risk given his exposure and a Jarisch-Herxheimer reaction is much more likely.

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A 57-year-old man who just returned from a cruise to the Bahamas comes to the emergency department with fever, non-productive cough and shortness of breath for the past 2 days. He has also had a headache, abdominal pain and diarrhoea since yesterday. The patient has a history of type 2 diabetes mellitus, and a smoking history of 20 pack-years. He does not use alcohol or illicit drugs. His temperature is 39.2ºC, blood pressure is 110/65 mmHg, pulse is 80/min and respirations are 18/min. Pulse oximetry shows 97% on room air.

Lung examination demonstrates crackles bilaterally. The abdomen is soft and non-tender. The patient seems confused at times during the examination. His serum glucose is normal; serum sodium is 128 mmol/l. Chest x-ray demonstrates bilateral interstitial infiltrates. He is admitted to the ward.

Which of the following antibiotics should be given to this patient?

A. Co-amoxiclav
B. Ceftriaxone
C. Clindamycin
D. Levofloxacin
E. Vancomycin

D. Levofloxacin

This patient has bilateral lung infiltrates, confusion and diarrhoea following travel. Legionella pneumonia (Legionnaires' disease) must be considered. Travel-associated Legionnaires' disease due to contaminated water supplies is a well-documented condition linked to cruise ships and hotels. *Legionella pneumophila* is a Gram-negative bacilli that stains poorly as it is primarily intracellular; hence Gram stain showing many neutrophils but no organisms is characteristic.

Legionnaires' disease is treated with antibiotics that achieve high intracellular concentration, particularly macrolides or fluoroquinolones (such as levofloxacin). The latter is favoured when the illness is severe enough to warrant admission.

Choice 1: Co-amoxiclav is typically used in some cases of CAP, but not Legionnaires' disease. It has activity against beta-lactamase-producing *Haemophilus* and *Moraxella* species.

Choice 2: Ceftriaxone has good activity against pneumococcus as well as *Haemophilus* and *Moraxella*. It does not have activity against *Legionella* or other causes of atypical pneumonia.

Choice 3: Clindamycin is used primarily to treat infections with anaerobic organisms; *Legionella* has aerobic activity.

Choice 5: Vancomycin has no activity against *Legionella* or most other causes of atypical pneumonia.

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A 64-year-old lady with a background of Alzheimer's dementia presents with a 3 day history of increased confusion, lower abdominal pain and foul smelling urine. Blood tests on admission show:

Hb	119 g/l	Na^+	136 mmol/l
Platelets	425 * 10 /l 9	K^+	3.7 mmol/l
WBC	15.9 * 10 /l 9	Urea	7.2 mmol/l
Neuts	13 * 10 /l 9	Creatinine	78 µmol/l
Lymphs	2 * 10 /l 9	CRP	140 mg/l
Eosin	0.02 * 10 /l9		

She is started on treatment for a urinary tract infection. 2 days later her blood tests show the following:

Hb	115 g/l	Na^+	141 mmol/l
Platelets	360 * 10 /l 9	K^+	5.2 mmol/l
WBC	10.2* 10 /l 9	Urea	8 mmol/l
Neuts	7.5 * 10 /l 9	Creatinine	105 µmol/l
Lymphs	1.5 * 10 /l 9	CRP	43 mg/l
Eosin	0.001* 10 /l9		

Which antibiotic was she likely to have been treated with?

A. Trimethoprim
B. Erythromycin
C. Co-amoxiclav
D. Nitrofurantoin
E. Ciprofloxacin

A. Trimethoprim

The correct answer is **Trimethoprim**. This is evidenced by the rise in serum potassium from 3.7 mmol/L to 5.2 mmol/L over two days of treatment. Trimethoprim is known to cause hyperkalaemia

through its action on the distal tubule of the kidney where it blocks the sodium channel ENaC, leading to reduced potassium excretion. This effect is particularly notable in elderly patients and those with reduced renal function. The patient's other blood results show an appropriate response to antibiotic therapy with falling WBC and CRP.

Erythromycin is not a first-line treatment for urinary tract infections in the UK according to NICE guidelines. While it can cause various side effects, significant changes in potassium levels are not typically associated with its use. Additionally, it has poor urinary penetration making it an unsuitable choice for UTIs.

Co-amoxiclav, while commonly used for UTIs, does not typically cause hyperkalaemia. The blood results show improving inflammatory markers which could be consistent with co-amoxiclav use, but the marked rise in potassium would not be expected with this antibiotic.

Nitrofurantoin is a common first-line treatment for uncomplicated UTIs, but it would be contraindicated in this patient due to her reduced renal function (eGFR likely <45 ml/min based on her age and creatinine). Furthermore, nitrofurantoin does not typically cause electrolyte disturbances.

Ciprofloxacin, while effective against urinary pathogens, does not cause significant changes in potassium levels. While it could effectively treat this infection, the characteristic rise in potassium seen in this case would not be expected with ciprofloxacin therapy.

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A 19-year-old man returning from Zimbabwe is admitted to the emergency department. He arrived back in the UK one week ago but for the last three days has complained of a severe headache and rigors. He has no significant past medical history. He smokes 10 cigarettes per day and does not drink alcohol.

On examination, the patient is sweaty and appears anxious. Pulse rate is 108/min, blood pressure 91/58 mmHg, temperature 39ºC and the respiratory rate 22/min. His chest is clear and heart sounds normal. A brief neurological examination is normal. An urgent blood film reveals malarial parasites.

Which of the following would suggest severe malaria infection?

A. Blood sugar > 14 mmol/l

B. Bicarbonate < 14mmol/l
C. CRP > 250 mg/l
D. Temperature > 38.5ºC
E. Infection despite use of appropriate antimalarial prophylaxis

B. Bicarbonate < 14mmol/l

The World Health Organisation states that the early recognition and treatment of severe malaria is vital to prevent rapid deterioration in the patient's condition and the onset of life-threatening complications.

Complications indicating severe malaria include:

- hypoglycaemia
- acidosis
- coma
- convulsions
- severe anaemia
- pulmonary oedema

Both coma and convulsions may represent cerebral malaria. In addition the parasite involved and percentage parasitaemia is important in the prognosis and management of malaria

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A 30-year-old woman with rheumatoid arthritis has been diagnosed in the community with a prolonged urinary tract infection. For her rheumatoid arthritis she has been stable on azathioprine for nearly a year and besides paracetamol she takes no other regular medications.

The GP has requested a follow-up blood test which is shown below. Baseline bloods were taken 2 months ago:

Hb	120 g/l
Platelets	310 * 10 /l9
WBC	4.5 * 10 /l9

Yesterday the bloods showed the following:

Hb	101 g/l
Platelets	296 * 10 /l9
WBC	1.9 * 10 /l9

Which of the following drugs is most likely responsible?

A. Trimethoprim
B. Nitrofurantoin
C. Cephalexin
D. Amoxicillin
E. Co-amoxiclav

A. Trimethoprim

It is likely she has received a protracted course of antibiotics, possibly more than one different antibiotic even.

An important side effect of azathioprine is cytopaenia. Azathioprine is metabolised to 6- mercaptopurine (6-MP) and then inactivated either by oxidation or methylation. Oxidation is catalysed by xanthine oxidase and methylation is catalysed by the enzyme TPMT. Factors that can lead to elevated levels of azathioprine are:

- Drugs that inhibit xanthine oxidase e.g. allopurinol
- Drugs that can also cause myelosuppression e.g.sulphonamides, trimethoprim
- Reduced activity of TPMT

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A 70-year-old female was admitted to the emergency department from a nursing home due to a progressive decline in her level of consciousness over the past two days. Her carer mentioned that she had been complaining of a burning sensation while urinating associated with a low-grade fever for the last week. Blood investigations showed:

Na +	130 mmol/l
K +	3.6 mmol/l
Urea	13 mmol/l
Creatinine	130 µmol/l

Urine dipstick showed increase leukocytes and nitrites. Urine culture showed a growth of extended-spectrum B-lactamase (ESBL) - producing *Escherichia coli*.

What is the first line treatment?

A. Ciprofloxacin
B. Ceftriaxone
C. Meropenem
D. Methoprim
E. Aztreonam

C. Meropenem

The regular use of B-lactam antibiotics for urinary tract infection has resulted in the emergence of new strains of bacteria that are resistant to these antibiotics. ESBL is one of the most problematic strains that is usually difficult to treat.

First line antibiotics are still carbapenems (imipenem, meropenem, ertapenem, doripenem).

Giving these antibiotics may further increase resistance so the advice is not to give antibiotics unless there are clinical signs and symptoms of a urinary tract infection.

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A 27-year-old nurse attends the emergency department and reports she has just suffered a significant needle-stick injury while caring for her patient on the intensive care unit. While providing her patient personal care with her colleague, she was injured in the hand by a wide bore stylet needle previously used to introduce an IV cannula to her patient that had not been placed in an appropriate sharps bin. The needle had been visibly blood stained. The nurse had been wearing gloves and had followed correct first aid procedure for needle-stick injuries.

The nurse was very concerned as the patient who had been the needle-stick donor was receiving treatment for the acute neurological phase of rabies. Review of the patients clinical notes (with the consent of the patients next of kin) indicated that he had contracted rabies following a dog bite two months previously in Pakistan. The diagnosis of rabies encephalitis had been made following clinical review by infectious disease experts and the detection of neutralising serum antibodies.

The nurse was previously fit and healthy with no significant past medical history. She did not use any regular medications and had no allergies. She was uncertain if she had had any previous vaccination against rabies, although did recall having a course of injections 10 years previously before she visited family in rural India.

What is the correct management of the needle-stick injury to prevent transmission of rabies?

A. Rabies immunoglobulin and full course rabies vaccination
B. Booster course rabies vaccination
C. Rabies immunoglobulin

D. Full course rabies vaccination
E. No action required

A. Rabies immunoglobulin and full course rabies vaccination

This situation represents a difficult clinical decision. Humans are considered to be an end-host for the rabies virus as inter-human transmission is not documented outside of transplantation cases. For that reason, only standard infection control precautions are recommended for health-care workers when in contact with individuals with rabies. However, the needle-stick injury described could foreseeably lead to transmission of the virus and given the severe consequences of rabies should be considered as possible rabies exposure.

The patient does not have a clear history of previous immunisation (the history stated could also be consistent with hepatitis B vaccination for example). Rabies immunoglobulin should be given to provide passive antibodies at the site of exposure prior to the development of an immune response. As far as possible, the dose (20 IU / Kg) should be administered locally around the wound. In addition, post-exposure vaccination should be provided, with the WHO currently recommending a four dose schedule (given on days 0, 3, 7 and 14).

If an individual has been previously vaccinated then immunoglobulin is not required and a two dose vaccination schedule (given on days 0 and 3) should be given.

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A 65-year-old man is referred to gastroenterology clinic for advice prior to planned overseas travel. The patient states that he is planning to visit relatives in Brazil in two months time and is very concerned about the possibility of contracting travellers' diarrhoea in light of his previous and current medical history.

The patient had undergone a total colectomy with ileostomy formation twenty years previously, as curative treatment for severe ulcerative colitis. He had subsequently learnt to manage his ileostomy well and had declined reconstructive surgery. Ten years previously, the patient had contracted diarrhoea while travelling in the Caribbean, which he reported had ruined his holiday and complicated his ileostomy management for weeks.

Of more pressing concern was a recent diagnosis of small-cell lung cancer, for which the patient was undergoing chemotherapy with cisplatin and etoposide. The patient's chemotherapy regime was due to be completed four weeks before his proposed travel.

Aside from the issues outlined above, the patient's only other past medical history included hypertension for which he took ramipril. The patient denied any drug allergies or intolerances.

Standard precautions regarding the avoidance of travellers' diarrhoea were discussed with the patient, who fully acknowledged the risks of travelling so soon after completing chemotherapy.

What is appropriate management to help prevent travellers' diarrhoea in this patient?

A. Vibrio cholerae vaccination
B. Standard advice regarding hygiene precautions only
C. Co-amoxiclav prophylaxis while overseas
D. Ciprofloxacin prophylaxis while overseas
E. Low-dose loperamide

D. Ciprofloxacin prophylaxis while overseas

Travellers' diarrhoea is most commonly caused by *Escherichia coli* species and *Campylobacter jejuni*. Less common causative pathogens include *Salmonella* and *Shigella* species, parasitic infections such as *Giardia lamblia*, and viruses such as norovirus.

In the majority of individuals, diarrhoea is annoying and unpleasant but does not lead to longterm sequelae. However, some groups of individuals may be unable to tolerate the consequences of dehydration from diarrhoea, or be vulnerable to invasive complications such as bacteraemia.

The patient presented in this question will be immunosuppressed secondary to his chemotherapy and has an under-lying bowel condition due to his ileostomy. Therefore, he should be considered for antibiotic prophylaxis to prevent travellers' diarrhoea, following discussion of the risks of antibiotic associated diarrhoea and other side effects.When antibiotic prophylaxis is used, typical choices of agent are ciprofloxacin, norfloxacin or rifaximin.

Anti-motility agents such as loperamide can be helpful in limiting diarrhoea in individuals unable to tolerate dehydration, but does not have a role in preventing infection. Symptoms of invasive colitis, such as severe abdominal pain or bloody diarrhoea, are contra-indications to the use of anti-motility agents due to the risk of intestinal perforation. Vaccination against cholera is effective, however is not a common cause of travellers' diarrhoea.

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A 19-year-old man attends the Emergency department with a fever for the past one week. He notes that the fever comes on alternate days. He reports also a co-existing headache, but no other particular symptoms. Of note, he returned from Afghanistan two weeks ago. Examination reveals a temperature of 38.1ºC, heart rate 89/min, blood pressure 123/78 mmHg, respiratory rate 17/min and oxygen saturations of 99%. There is no evidence of no neck stiffness, objective photophobia, jaundice or splenomegaly.

What is the most likely diagnosis?

A. Chikungunya
B. Zika virus
C. Plasmodium vivax
D. Plasmodium falciparum
E. Dengue fever

C. Plasmodium vivax

Fever in the returning traveller is a common presentation in the Emergency department. A knowledge of the areas travelled and incubation periods is essential to accurately diagnosing the potential infectious disease.

Chikungunya is a viral disease transmitted to humans via infected mosquitos. Fever and joint pain is the mos common presentation. The incubation period is 2-12 days.

Zika virus is currently not present in Afghanistan.

Dengue fever has a incubation period of 4-10 days.

This leaves plasmodium falciparum, which has a usual incubation of 7-14 days (but up to 3 months), and plasmodium vivax which has a typical incubation of 12-17 days (but reported up to 12 months) following exposure. Factors that point away from p. falciparum are the milder presentation and the much lower prevalence of it in Afghanistan (p. falciparum 5%, p. vivax 95%). Furthermore, fever due to malaria on alternate days, known as benign tertian malaria, is suggestive of either p. vivax or p. ovale. Comparatively, p. falciparum will typically cause more of a continuous fever.

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A 76-year-old female has been under the care of the medical team for the past week. She has been treated for a full sensitive *E. coli* bacteraemia with intravenous co-amoxiclav thought be sourced from the urinary system. On completing the course, the patient remains in for ongoing physiotherapy input. The next day, the patient has a temperature spike and cultures demonstrate regrowth of *E. coli*.

What is the most appropriate next step?

A. Escalate antibiotics to meropenem
B. Image the renal tract
C. Commence treatment for hospital-acquired pneumonia
D. Urology referral
E. Urgent nephrostomy

B. Image the renal tract

Discussion of this case with microbiology would likely result in the suggestion of imaging. In someone who has appropriate antimicrobial treatment, regrowth of the same organism would not be expected so soon. This may indicate there is a source of the bacteria that is not being adequately treated - a renal abscess in this case, which may be identified on ultrasound.

Escalation to meropenem should not be considered necessary if the organism has been fully sensitive to the narrower spectrum antibiotics.

E. coli would be an unlikely source of hospital-acquired pneumonia.

Urology referral would be the next step should a renal abscess be identified. Imaging should first be carried out to determine whether a nephrostomy is required.

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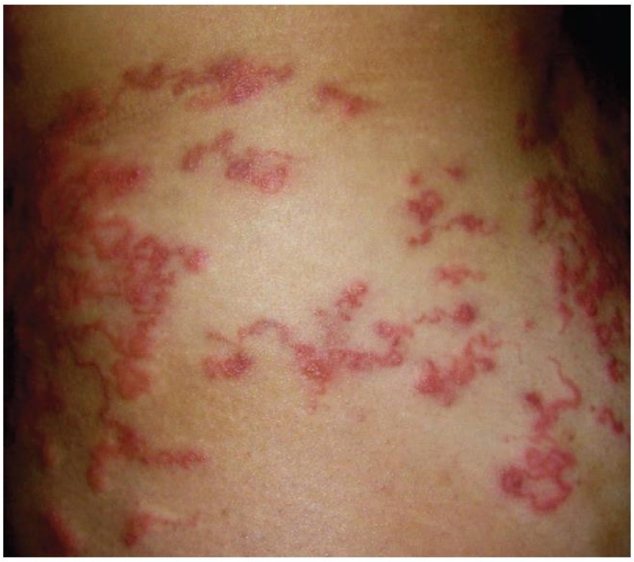
A 26-year-old man presents with an intensely itchy rash which he thinks is spreading rapidly. He has recently returned from a holiday in Brazil, and mentions having seen many stray cats around the hotel resort. Symptoms began approximately 1 week after returning from the holiday. He denies having had any similar rashes previously, has no significant past medical history and is systemically well.

On examination, the rash appears as follows:



Given the most likely diagnosis, which of the following is the most appropriate management?

A. Doxycycline
B. Hydrocortisone
C. Ivermectin
D. Malathion
E. Permethrin

C. Ivermectin

The rash above has the appearance of cutaneous larva migrans, most commonly caused by infection with the cat hookworm *Ancyclostoma braziliense*. It is found in tropical regions throughout the world, including Brazil, from where this patient has recently returned, having mentioned a resort with many stray cats. The rash creeps due to the migration of the larvae. The photo above demonstrates this creeping, serpiginous eruption. Management involves the use of ivermectin, which is the correct answer here, or albendazole.

Doxycycline is incorrect. This is an antibiotic that would be used for the management of Lyme disease. This would instead present with erythema chronicum migrans - annular, bulls-eye lesions, rather than the tortuous lesions seen here.

Hydrocortisone is a topical steroid and is not involved in the management of cutaneous larva migrans. Ivermectin or albendazole should be given.

Malathion is a second-line option for the management of scabies. Whilst also presenting with intense pruritus, scabies lesions would instead appear as small, discrete burrows, particularly in the web spaces between fingers, as well as the palms and wrists. The serpiginous lesions seen here are more indicative of cutaneous larva migrans.

Permethrin is generally the first-line treatment used for scabies. Again, the lesions seen above are more associated with cutaneous larva migrans, not scabies.

Question 153 of 277

A 44-year-old businessman was admitted to the general medical take with fever, jaundice and headaches. He had returned from a business trip to Hong Kong one month ago. He is a keen river sailor and has been unable to sail for the last two weeks.

On examination, he is tachycardic with a temperature of 39.5°C. He appears confused and is combative during the examination. His chest has scattered crackles bibasally with some right upper quadrant abdominal pain

Investigations

Chest X-Ray	Hazy lung infiltrates bibasally

Haemoglobin	135 g/L (130-180)
White cell count	17.8 Ã— 109/L (4.0-11.0)
Neutrophil count	15.7 Ã— 109/L (1.5-7.0)
Platelet count	413 Ã— 109/L (150-400)

Serum C-reactive protein	345 mg/L (<10)
Serum urea	13.3 mmol/L (2.5-7.0)
Serum total bilirubin	110 mol/L (1-22)
Serum alanine aminotransferase	117 U/L (5-35)
Serum alkaline phosphatase	110 U/L (45-105)
Serum gamma glutamyl transferase	61 U/L (<50)

What is the most likely diagnosis?

A. Dengue Fever
B. HIV seroconversion
C. Acute hepatitis A
D. Malaria
E. Leptospirosis

E. Leptospirosis

Leptospirosis is the most likely diagnosis. The river sailing puts this gentleman at risk of contact with contaminated water from animal urine. Incubation is between 4-14 days. Typical symptoms include high fever, headache, muscle aches, vomiting and may have meningitis and jaundice.

Hong Kong is not a malaria endemic region nor is it a common region for Dengue Fever. HIV seroconversion would not explain jaundice or deranged liver function. Acute hepatitis A infection is prevalent in Hong Kong and incubation period is between 2 - 6 weeks but infection would not explain the neurological symptoms.

Question 154 of 277

A 23 year-old artist presents after he woke up with headache, neck stiffness, and photophobia. He is normally fit and well, but for the last three weeks has complained of feeling tired and irritable.

On examination, the left side of the palate does not elevate, and the tongue is deviated to the left upon protrusion. The remainder of the neurological examination is unremarkable.

Plain computed tomography of the head is unremarkable.

Lumbar puncture is performed with results of CSF analysis as follows:

Appearance	Turbid
White blood cells	28 cells/mm^3 (95% lymphocytes)
Red blood cells	<1 cells/mm^3
Gram stain	No organisms seen
Protein	1.32 g/L
Glucose	1.4 mmol/L
Serum glucose	7.5 mmol/L

What is the most likely diagnosis?

A. Meningococcal meningitis
B. Tuberculous meningitis
C. Subarachnoid haemorrhage
D. Skull base tumour
E. Malignant meningitis

B. Tuberculous meningitis

The prodromal phase of malaise and possible personality change, followed by meningism, with the onset of basal cranial nerve palsies, and coupled with the CSF findings of lymphocytic pleocytosis, raised protein, and low glucose, are all highly suggestive of tuberculous meningitis.

There is no particular suggestion of a primary malignancy, but malignant meningitis (leptomeningeal carcinomatosis) is a possibility and it would be reasonable to send a large volume of CSF for cytology. Like tuberculous meningitis, this condition typically presents with headache, meningism, and cranial nerve palsies. However, it is eye movements which are most often affected.

There is often also invasion of spinal meninges which involves nerve roots and mimics radiculopathy. The CSF is hardly ever normal, although there are no specific features other than the direct demonstration of malignant cells.

Meningococcal meningitis is an acute disease, so the three week prodrome described here would be unusual. The presence of cranial nerve palsies would be extremely unusual. The CSF would be expected to show a neutrophilic pleocytosis, with a low glucose (less that 50% of serum glucose), but not as low as in tuberculous meningitis.

Subarachnoid haemorrhage should be considered in any case of acute headache. The normal CT scan is reassuring but does not exclude the diagnosis, but in any case the CSF is not suggestive.

A tumour of the skull base might explain the lower cranial nerve palsies, but one would have expected some more suggestive symptoms and an abnormal CT head. This diagnosis would not explain the grossly abnormal CSF findings.

Question 155 of 277

A 38-year-old man is referred in by his GP with increasing frequency of diarrhoea over the last 4 days. He is a known ulcerative colitis patient who is well managed on azathioprine 200mg OD and mesalazine 2.4mg BD. He last had a flare 2 years ago and has been well in the interim. He is passing up to 10 watery motions a day and is suffering from faecal urgency and nocturnal episodes. He describes cramping left iliac fossa pain. There is no blood or mucus in the stools. On examination, he is febrile at 38.2 degrees celsius. His blood pressure is 120/75 mmHg and his heart rate is 100/min. On examination, he is underweight with a BMI of 18.5 and dehydrated. His abdomen is soft but he is tender in the left iliac fossa. He refuses a PR examination. Respiratory and cardiovascular examinations are normal.

His blood tests show:

Hb	110 g/l
Platelets	189 * 10 /l9
WBC	3.8 * 10 /l9
Neutrophils	0.89 * 10 /l9
INR	1.1 (0.9-1.2)

Na +	136 mmol/l
K +	4.9 mmol/l
Urea	8.0 mmol/l
Creatinine	100 µmol/l
Mag nesium	0.79 mmol/L (0.7-1.0)
Calcium	2.4 mmol/L (2.1-2.58)
CRP	78 mg/l

Bilirubin	5 µmol/l
ALP	78 u/l
ALT	28 u/l
Albumin	33 g/l

He is started on IV hydrocortisone 100mg QDS and IV fluids. Stool specimens are sent and are reported as negative for *C. difficile* toxin. He has a flexible sigmoidoscopy the next day which shows widespread left sided colitis. The biopsy results show the presence of inclusion bodies in the colonic mucosa.

What is the appropriate treatment to start for this gentleman?

A. Infliximab
B. Ganciclovir
C. Metronidazole
D. Fluconazole
E. Tazocin

B. Ganciclovir

This patient has CMV colitis. It can be a consequence of immunosuppressive agents such as azathioprine. It can present with fever and diarrhoea +/- blood. It can also present as an exacerbation of IBD patients in those on immunosuppressants. Inclusion bodies are characteristic at biopsy. It will respond well to an antiviral agent and ganciclovir is the treatment necessary here. Tazocin, metronidazole and fluconazole will not treat appropriately and infliximab would only be indicated if a colitis flare was not due to an infective cause.

Question 156 of 277

A 47-year-old man is referred by the emergency department with increasing shortness of breath. He is a known HIV patient managed by the local GUM consultants. He is not on any antiretroviral treatment. He presents with a two-week history of worsening shortness of breath. He is now breathless on minimal exercise. He has a non-productive cough and has felt lethargic for the last week. On examination he is afebrile, his blood pressure is 120/89 mmHg and he is slightly tachycardic at 110bpm. His respiratory rate is 18 at rest with saturations on 98% on air. When he mobilises to the toilet he becoming very tachypnoeic and his saturations drop to 90%. His blood tests are as follows:

Hb	110 g/l
Platelets	201 * 10 /l9
WBC	9.6 * 10 /l9
Neutrophils	4.5 * 10 /l9

Na +	138 mmol/l
K +	4.1 mmol/l
Urea	7.8 mmol/l
Creatinine	20 µmol/l
CRP	70 mg/l

Bilirubin	5 µmol/l
ALP	89 u/l
ALT	43 u/l
Albumin	34 g/l

An ABG is done which shows

pH	7.35
pO2	7.7
pCO2	4.6
HCO3-	21
BE	-3
Lactate	2.2

His chest x-ray shows fine bilateral reticular nodular shadowing.

What is the most appropriate treatment to start in this gentleman?

A. Tazocin
B. Co-trimoxazole
C. Aciclovir
D. Co-trimoxazole and corticosteroids
E. Highly active anti-retroviral treatment

D. Co-trimoxazole and corticosteroids

This gentleman is immunocompromised and has PCP. Patients with moderate to severe PCP should be treated with Co-trimoxazole and corticosteroids. Patients with mild to moderate PCP need only co-trimoxazole.

Mild:

- Breathlessness on mild exercise, which may be associated with a cough and sweats
- Arterial blood gases and oxygen saturation at rest, on air: PaO2 >11 kPa; SaO2 >96%
- Chest X-ray: normal or minor perihilar infiltrates

Moderate:

- Breathlessness on minimal exercise, fever (with or without sweats)
- Arterial blood gases and oxygen saturation at rest, on air: PaO2 8-11 kPa; SaO2 91-96% Chest X-ray: diffuse interstitial shadowing

Severe:

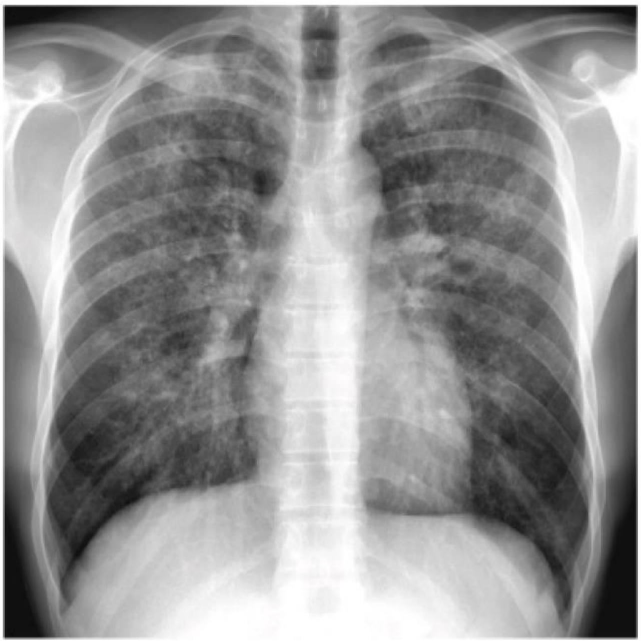
- Breathlessness at rest, persistent fever and cough
- Arterial blood gases and oxygen saturation at rest, on air: PaO2 <8 kPa; SaO2 <91%
- Chest X-Ray: extensive interstitial shadowing, with or without alveolar shadowing

Question 157 of 277

A 38-year-old man is admitted to the Emergency Department with shortness-of-breath and a nonproductive cough. He has recently emigrated from Russia and has just started anti-retroviral therapy for HIV.

Examination of his chest is unremarkable. His temperature is 37.7ºC and oxygen saturations are 95% on room air.

A chest x-ray is performed:



What is the most likely diagnosis?

A. Cytomegalovirus
B. Mediastinal lymphoma
C. Tuberculosis
D. Mycoplasma pneumonia
E. Pneumocystis jiroveci pneumonia

E. Pneumocystis jiroveci pneumonia

The key to answering this question is to look at the history rather than the x-ray. A combination of HIV + chest symptoms + unremarkable auscultatory findings in an exam are highly suggestive of Pneumocystis jiroveci pneumonia (more commonly known as PCP). X-ray changes in Pneumocystis jiroveci pneumonia are generally too subtle for non-radiologists to detect. The x-ray should however help you to exclude some of the other diagnoses.

The chest x-ray shows hazy, perihilar mid and upper zone opacification with some interstitial prominence. A few discrete cysts (termed pneumatocoeles) measuring up to 1cm are present. The presence of pneumatocoeles in a HIV patient suggests Pneumocystis jiroveci pneumonia.

Question 158 of 277

A 53-year-old lady comes into the Emergency Department with a cough productive of green sputum and palpitations. She feels very unwell, feverish and lethargic. On examination, she has bronchial breathing at her right base with respiratory rate 25/min, sats 95% on room air. Her heart sounds are normal with an irregularly irregular heartbeat. Her heart rate was 120/min and blood pressure 90/40 mmHg. An ECG shows atrial fibrillation with a fast ventricular rate. She has no history of atrial fibrillation.

What is the first treatment that should be given for her atrial fibrillation?

A. Bisoprolol
B. Digoxin
C. Intravenous fluids
D. Oral antibiotics
E. Flecainide

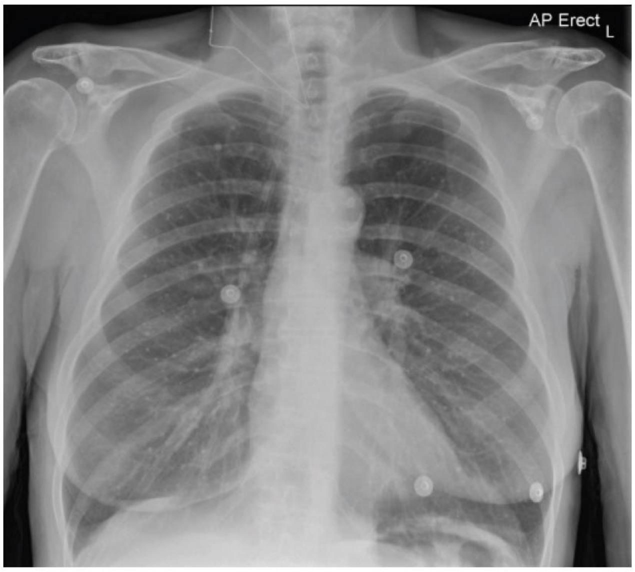
C. Intravenous fluids

This patient is clearly septic from pneumonia. This has tipped the patient into atrial fibrillation (AF) given there is no previous history of atrial fibrillation. It is important to treat the sepsis as the cause of the AF. To do this it is imperative to give IV fluids and IV antibiotics. If the patients AF does not settle following the resolution of the sepsis then other options would be considered.

Question 159 of 277

A 46-year-old woman presents with a 4 week history of cough. This is non-productive and not associated with dyspnoea or chest pain. The symptoms were preceded by a 'head cold'. Her past medical history includes asthma as a child and smoking. Respiratory examination reveals a clear chest with no added sounds. Oxygen saturations are 98% on room air. Temperature is 36.7ºC and heart rate 72/min.

A chest x-ray is requested as she is a smoker:



What is the most likely explanation for the changes on the x-ray?

A. Healed varicella pneumonia
B. Idiopathic pulmonary fibrosis
C. Cryptogenic organizing pneumonia
D. Tuberculosis
E. Sarcoidosis

A. Healed varicella pneumonia

The chest x-ray shows miliary opacities secondary to healed varicella pneumonia. Multiple tiny calcific miliary opacities noted throughout both lungs. These are of uniform size and dense suggesting calcification. There is no focal lung parenchymal mass or cavitating lesion seen.The appearances are characteristic for healed varicella pneumonia.

Question 160 of 277

A 26-year-old pregnant women is seen on the acute medical unit. She is 24 weeks pregnant with her first child and has been admitted by her GP after feeling generally unwell with 'flu like symptoms and developing jaundiced sclera. Her stay in Bangladesh lasted 2 weeks during which time she visited family. Prior to travelling she was vaccinated against hepatitis A.

On examination her sclera are visibly jaundiced. Blood pressure is 108/60 mmHg, temperature 38.1ºC and pulse 96/min. She is slightly tender in the right upper quadrant of the abdomen. Bloods show the following:

Bilirubin	72µmol/l
ALP	252 u/l
ALT	342 u/l
γGT	286 u/l
Albumin	38 g/l
CRP	154 mg/l

What is the most likely diagnosis?

A. Dengue fever
B. Hepatitis E
C. Hepatitis A
D. Intrahepatic cholestasis of pregnancy
E. Acute fatty liver

B. Hepatitis E

Hepatitis E is more likely to cause severe disease in pregnant women. Hepatitis A is unlikely given her previous immunisation against this. There are no other features to support the remaining three distractors.

Question 161 of 277

A 24-year-old recent immigrant from Albania presents to the emergency department with fever, headache and malaise. Over the past 24 hours he has also developed bilateral pain and swelling at the angle of the jaw, which is made worse by talking or chewing. On examination his pulse is 90/min, temperature 38.4ºC and bilateral palpable, tender parotid glands are noted.

Given the likely diagnosis, which one of the following complications is he most likely to develop?

A. Orchitis
B. Pancreatitis
C. Encephalitis
D. Myocarditis
E. Pneumonia

A. Orchitis

Orchitis is the most common complication of mumps in post-pubertal males.

There is a link between mumps and pancreatitis (the 'M' in GET SMASHED) but this is much less common than orchitis.

Question 162 of 277

A 41-year-old man with poorly controlled type 1 diabetes mellitus presents with a nodular lesion on the right-side of his face around the angle of the jaw. One month ago he had a tooth extraction at the dentist. The nodule is around 2 cm in diameter, raised and purple-red in colour. On examination a sinus tract is seen in the middle of the nodule which is draining a blood-stained fluid.

Microscopy of the discharge shows microscopic yellow granules.

What is the most likely causative organism?

A. Actinomyces israelii
B. Tunga penetrans
C. Klebsiella
D. Pseudomonas aeruginosa
E. Escherichia coli

A. Actinomyces israelii

The correct answer is ***Actinomyces israelii***. The patient's presentation of a nodular lesion with a sinus tract draining blood-stained fluid and the presence of microscopic yellow granules in the discharge are characteristic features of actinomycosis, which is most commonly caused by *Actinomyces israelii*. Actinomycosis is an indolent, slowly progressive infection that often occurs following dental procedures or oral trauma. The presence of microscopic yellow granules, also known as 'sulfur granules,' further supports this diagnosis.

Tunga penetrans, also known as the chigoe flea, causes tungiasis, which typically presents with pruritic papules or nodules on the feet. This clinical presentation does not match the patient's facial lesion and history of tooth extraction.

Klebsiella species are gram-negative bacteria that can cause a variety of infections, including pneumonia, urinary tract infections, and soft tissue infections. However, these infections do not typically present with nodular lesions and sinus tracts containing sulfur granules.

Pseudomonas aeruginosa is another gram-negative bacterium that can cause various infections such as skin and soft tissue infections, particularly in immunocompromised individuals. Although *Pseudomonas aeruginosa* can cause skin lesions, it does not produce sulfur granules nor form sinus tracts like those seen in actinomycosis.

Finally, ***Escherichia coli*** is a gram-negative bacterium commonly associated with gastrointestinal and urinary tract infections. It can occasionally cause skin and soft tissue infections but would not present with the characteristic nodular lesion containing sulfur granules seen in this patient.

Question 163 of 277

A 55-year-old man attends after puncturing the skin of his left hand with a small garden fork whilst weeding. On removing the temporary bandage there is no active bleeding but a puncture wound is noted. He has tried to clean the wound but there remains some soil in situ. He is unsure of his tetanus vaccination history but thinks he 'has never not had a vaccine he's been offered'. You can find no record on his tetanus vaccine history on his medical notes.

What is the most appropriate course of action?

A. Tetanus immunoglobulin
B. Tetanus vaccine booster
C. Tetanus vaccine booster + tetanus immunoglobulin
D. Oral co-amoxiclav
E. Tetanus vaccine booster + oral co-amoxiclav

C. Tetanus vaccine booster + tetanus immunoglobulin

This is a high-risk wound (soil contamination which may contain tetanus spores) in a patient without a clear history of tetanus vaccination. He therefore requires both a tetanus vaccine booster and tetanus immunoglobulin.

Question 164 of 277

A 52-year-old male presents with a cough, fatigue, fever, and shortness of breath. He had been feeling unwell for around 6 months with recurrent colds that he couldn't seem to shake off. Over this period he had lost around one and a half stone in weight. He attributed this to a poor appetite. His only past medical history was asthma. He had been a widower for 7 years and was a keen wildlife photographer which had allowed him to travel the world. His last trip had been to Africa 2 years ago.

On examination he had a temperature of 38.2ºC, heart rate is 102/min and regular, blood pressure 110/70 mmHg and saturations 92% air. He had fine bibasal crackles on chest auscultation. Heart sounds were normal. Arterial blood gas showed a type 1 respiratory failure. Chest x-ray showed bilateral pulmonary infiltrates. He was treated with intravenous antibiotics. After two days of admission, his condition worsened and his type 1 respiratory failure worsened and he was given continuous positive airway pressure (CPAP). The beta glucan test came back negative. Urinary antigens were negative. The patient remarked that he had noted that his vision in his right eye was blurred and he had noted flashing lights and spots in the same eye.

What is the most likely diagnosis?

A. Pneumocystis pneumonia
B. CMV pneumonia
C. Fungal pneumonia
D. Legionella pneumonia
E. Asthma exacerbation

B. CMV pneumonia

This gentleman has CMV pneumonia and then developed CMV retinitis on the background of an undiagnosed HIV infection.

CMV infection is typically unnoticed in healthy people but can be life-threatening for those who are immunocompromised, which would include transplant patients, cancer patients, dialysis patients, HIV-infected individuals and those on immunosuppressants.

CMV pneumonia often presents with a cough, fever, malaise, poor appetite, myalgia, shortness of breath and night sweats. CMV can be detected using serology. The virus can also be cultured from urine, throat swabs, bronchial lavages and tissue samples to detect active infection. PCR testing can also be done and this can help monitor viral load.

Ganciclovir or valganciclovir are treatments for CMV infection. Foscarnet or cidofovir are alternatives when patients are resistant to ganciclovir. Foscarnet can cause nephrotoxicity and electrolyte disturbance.

CMV retinitis often presents with blurred vision, floaters, flashing lights, eye pain, photophobia and can lead to blindness. CMV retinitis most commonly develops when the CD4 count is less than

50cells/mL. However, it can occur in 20-30% when then CD4 count is less than 100 cells/mL.

Question 165 of 277

A 32-year-old woman who has received a renal transplant from an unrelated donor some 4 weeks earlier, comes to the clinic complaining of fevers, arthralgia, abdominal pain and diarrhoea. She had initially made a good recovery after her transplant. Testing for human herpes virus 5 is positive and there has been a rise in CRP and serum creatinine, (values are shown below).

Hb	10.2 g/l	Na +	138 mmol/l
Platelets	203 * 10 /l 9	K +	5.2 mmol/l
WBC	10.9 * 10 /l 9	Urea	9.2 mmol/l
Neuts	8.7 * 10 /l 9	Creatinine	211 µmol/l
Lymphs	2.0 * 10 /l 9	CRP	88 mg/l
Eosin	0.1 * 10 /l9		

Which of the following is the most appropriate intervention?

A. IV acyclovir
B. IV ganciclovir
C. Oral lamivudine
D. Oral valganciclovir
E. Oral valaciclovir

B. IV ganciclovir

Human herpes virus 5 (HHV 5), is cytomegalovirus (CMV). In patients who are post-transplant, CMV infections can occur from reactivation of latent infection, infection via the transplanted organ, or because of a new primary infection. Treatment is with IV ganciclovir, and where there is ganciclovir resistance, foscarnet or cidofovir are alternative options. Without treatment, further deterioration in renal function is the likely outcome.

IV acyclovir is the usual treatment option for herpes simplex or herpes zoster infection and oral valaciclovir is an alternative option to oral acyclovir for the long term treatment of herpes simplex. Lamivudine is used in the treatment of HIV/AIDs and hepatitis B infection. Valganciclovir is an alternative oral option to IV ganciclovir with potential use for CMV prophylaxis.

Question 66 of 277

A 98-year-old lady presents with a fever and confusion and on initial review is started on broad spectrum antibiotics. Despite five days of therapy, her temperature is 38.9°C and her clinical condition seems to have deteriorated. She has no relevant past medical history. The diagnosis of urinary tract infection (UTI) is confirmed when urine cultures confirm an extended spectrum Blactamase (ESBL) producing *Escherichia coli* (E. coli).

Following the failure to respond to the first-line agent, which antibiotic is most likely to be effective?

A. Trimethoprim
B. Tetracycline
C. Amoxicillin
D. Cefalexin
E. Ertapenem

E. Ertapenem

Extended spectrum B-lactamase (ESBL) producing organisms are typically resistant to penicillins and cephalosporins and as such the carbapenem class of antibiotics are typically first-line although nitrofurantoin or fosfomycin are also frequently effective. ESBL producers are most commonly *Escherichia coli* (E. coli) and *Klebsiella* species.

Question 167 of 277

You see a 26-year-old Caucasian man with poorly controlled HIV (recent CD4 count of 113 cells/mm) with a negative Mantoux test. He was routinely tested after his father (whom he lives with) was diagnosed with active pulmonary disease, but himself shows no features of active disease. On examination, his chest is clear with no overt lymphadenopathy. Blood pressure is 132/91 mmHg, heart rate 85 beats per minute, respiratory rate 14 breaths per minute, oxygen saturation 96% on air and temperature 36.8°C.

What is the next step?

A. 5 days oral ciprofloxacin
B. QuantiFERON-TB (interferon gamma assay)
C. Induced sputum for AAFB
D. Monitor and repeat Mantoux in 2 weeks
E. Rifampicin and isoniazide for 6 months with pyrazinamide and ethambutol for 2 months

B. QuantiFERON-TB (interferon gamma assay)

In people with HIV and a CD4 count of less than 200 cells/mm an interferon-gamma test should be done alongside the Mantoux as it will be unreliable. If either test is positive a clinical assessment should be performed to exclude active TB and if no active disease found consideration made to treating a latent infection.

Question 168 of 277

A 72-year-old male diabetic is admitted to the hospital via the Emergency Department with severe cellulitis of his right arm. He is hypertensive and also suffers from coronary artery disease.

On examination he is febrile, with a temperature of 38.5°C. His pulse is 112 bpm and his blood pressure is 150/95 mmHg.

His right arm is grossly swollen and tender, with marked erythema and discharge of small amounts of pus.

Lab reports reveal:

Hb	90 g/l
MCV	71 fl
WBC	23 * 10 /l9
Plt	500 * 10 /l9
Urea	9.2 mmol/l
Creatinine	145 µmol/l

Urine examination reveals proteinuria 1+ and glycosuria 2+

Preliminary blood cultures post admission reveal the growth of MRSA.

Which of the following would be the most appropriate antibiotic for this patient?

A. IV Vancomycin
B. IV Flucloxacillin
C. IV Linezolid
D. IV Rifampicin
E. IV Gentamicin

A. IV Vancomycin

IV Vancomycin is the treatment of choice for MRSA septicaemia. The renal function is slightly deranged here so the dosing will need to be adjusted accordingly.

Question 169 of 277

A 28-year-old patient has presented with upper limb weakness. He is extremely anxious and tells you that this morning he noticed that he had a dry mouth and found it difficult to swallow. His friends also commented that his voice sounded different to normal. Several hours later he began to notice weakness in both of his arms. He has not noticed any weakness in his lower limbs. He has no relevant past medical history or family history of note.

He smokes 10 cigarettes a day, drinks approximately 30 units of alcohol per week and occasionally injects heroin.

His Glasgow Coma Score is 15/15. Neurological examination reveals power 3/5 in the upper limbs, 5/5 in the lower limbs. Biceps and supinator reflexes are absent. Knee and ankle reflexes are normal. His pupils are dilated and sluggish in reaction to light. He is unable to abduct either eye. You notice needle track marks on the patient's forearm and an erythematous wound in the patient's right antecubital fossa.

What is treatment would you give to the patient immediately?

A. IV Beta Interferon
B. IV immunoglobulin
C. Plasma exchange
D. Steroids
E. Trivalent antitoxin

E. Trivalent antitoxin

This patient has presented with typical features of botulism, which is likely to have been contracted via the patient's wound in this case. He has suffered from a descending weakness, initially affecting his bulbar and pharyngeal muscles, then his upper limbs. If untreated this would progress to involve his respiratory muscles and lower limbs. In contrast, Guillain Barre Syndrome (GBS) presents with an ascending weakness usually involving the lower limbs, then upper limbs then cranial nerves. Both conditions show autonomic features and progress rapidly.

One particularly distressing feature of botulism is that it does not affect GCS.

The immediate treatment of choice would be IV Trivalent antitoxin immediately and general supportive measures.

Question 170 of 277

A 32-year-old man presents to the emergency department with a three-week history of progressive shortness of breath, dry cough, and fever. He also mentions unintentional weight loss and generalized fatigue in the past two months.

On further questioning, he admits to having had multiple sexual partners in the past year and not using protection consistently.

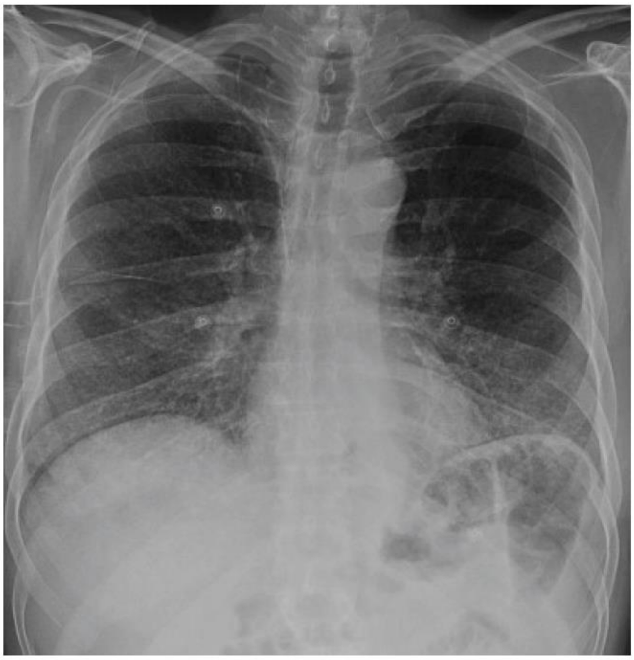
Observations are as below:

- Temperature 38.9°C (102°F)
- Respiratory rate of 28 breaths/min
- Oxygen saturation of 85% on room air

Arterial blood gases:

Bicarbonate	15 mmol/L	(22 - 29)
PaO2	8.8 kPa	(10.5 - 13.5)
PaCO2	3.8 kPa	(4.5- 6)

Chest examination is unremarkable. The chest x-ray is given below.



What is the most appropriate management in this case?

A. Co-trimoxazole
B. Co-trimoxazole and steroids
C. IV furosemide
D. IV pentamidine
E. Supportive treatment

A. Co-trimoxazole and steroids

Co-trimoxazole and steroids is the correct answer.

The chest x-ray shows increased bibasilar ground glass opacities, combined with the patient's clinical presentation and history of unprotected intercourse, strongly hint at a possible HIV infection complicated by *Pneumocystis jirovecii* pneumonia (also known as PCP). Co-trimoxazole is the first-line treatment for *Pneumocystis jirovecii* pneumonia. The addition of steroids is recommended in hypoxic patients (evidenced here by a PaO2 of 8.8 kPa on air). Steroids have been demonstrated to decrease the risk of respiratory failure by 50% and reduce mortality.

Co-trimoxazole on its own would be inadequate, as the pronounced hypoxia in this patient poses a significant risk of mortality, thus warranting the simultaneous use of steroids.

IV furosemide is not suitable. The imaging changes here are not in keeping with pulmonary oedema, due to peri-hilar distribution of opacification, and lack of other features such as costophrenic angle blunting or fluid in the fissures, and as such diuretic therapy would not be indicated.

IV pentamidine is not the best choice. It can serve as a second-line treatment for *Pneumocystis jirovecii pneumonia* if there are contraindications or adverse reactions to co-trimoxazole but there are no known reasons not to use co-trimoxazole in this case. This patient's marked hypoxia also calls for the administration of steroids.

Supportive treatment on its own isn't adequate. Although vital measures such as oxygen supplementation are necessary for symptom management, they fail to tackle the primary issue - the fungal infection.

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A 25-year-old woman presents to the clinic with a one-month history of persistent, watery diarrhoea that is sometimes difficult to flush but never bloody. She also reports associated bloating and flatulence. Since returning from a trip to South America, she has experienced increasing weakness and an inability to maintain her usual level of physical activity. Over this period, she has lost 10 kg in weight but denies joint pains.

On examination, she is afebrile. Neurological examination shows no abnormalities. Cardiac auscultation reveals normal first (S1) and second (S2) heart sounds without additional sounds or murmurs. There are no skin rashes present.

What is the most likely diagnosis?

A. Amoebic dysentery
B. Coeliac disease
C. Giardiasis
D. Traveller's diarrhea 2%
E. Whipple's disease

C. Giardiasis

Ongoing diarrhoea, lethargy, bloating, flatulence, steatorrhoea, weight loss +/- recent travel → ?giardiasis

Giardiasis is the correct answer.

Giardiasis should be considered in cases of persistent non-bloody diarrhoea, mainly when it presents as watery and sometimes steatorrhea. Accompanying symptoms may include lethargy, bloating, flatulence, and weight loss. G. lamblia transmission occurs via the fecal-oral route, often through contaminated water or food. The parasite can produce cysts resistant to chlorine and other disinfectants, complicating its eradication from water supplies. Upon ingestion, the cysts excyst within the small intestine and release trophozoites that adhere to the intestinal wall and proliferate.

Amoebic dysentery is incorrect. E.histolytica, the cause of amoebic dysentery, also follows a faecal-oral transmission route, usually via contaminated food or water. Post-ingestion, this parasite may invade the intestinal lining, causing abdominal discomfort and diarrhoea with potentially bloody stools. Additionally, it can disseminate to other organs, including the liver, lungs, and brain, leading to severe infections which could be life-threatening. However, given the presence of steatorrhea in this scenario, E.histolytica is less likely.

Coeliac disease is a long-term autoimmune disorder primarily affecting the small intestine, where individuals develop intolerance to gluten, which is present in foods such as wheat, rye and barley. Abnormal bowel response to the ingested gluten leads to villous atrophy and subsequent destruction of surface absorption observed in the duodenum and jejunum. Although the symptoms are similar to giardiasis, the relation between symptoms and travelling makes giardiasis more likely. Testing for coeliac disease may be considered part of investigations if the treatment for giardiasis is unsuccessful.

Traveller's diarrhoea is incorrect. *Enterotoxigenic Escherichia coli* (ETEC) is the most common cause of traveller's diarrhoea and is typically associated with watery diarrhoea that may occasionally contain blood rather than steatorrhea. The presence of steatorrhea makes it unlikely.

Whipple's disease caused by *T.whipplei,*—is a rare condition predominantly affecting middle-aged men and can manifest with gastrointestinal symptoms, including steatorrhoea. Nevertheless, the absence of additional signs like joint pain, central nervous system involvement or headaches makes this diagnosis unlikely in this context.

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A 23-year-old man presents to the emergency department with shortness of breath, fever and a cough. He has had a non-productive cough for two weeks. Before this, he had felt unwell with muscle aches and malaise. He went to his GP and was given amoxicillin but this has made no difference and now he is feeling more breathless and unwell. He has not been outside of the UK in the last year. He has no other medical problems and takes no regular medications. His cough is non-productive and comes in paroxysms which make him concerned.

On examination, there are a few bilateral crepitations, but the examination is otherwise normal. His observations show fever and mild hypoxia. Blood tests show mild anaemia and raised inflammatory markers. A chest X-ray shows bilateral consolidation, but he improves with IV coamoxiclav and clarithromycin over a few days.

What is the most likely causative organism?

A. Klebsiella pneumoniae
B. Legionella pneumophila
C. Mycoplasma pneumoniae
D. Chlamydia psittaci
E. Coxiella burnetii

C. Mycoplasma pneumoniae

The correct answer is Mycoplasma pneumoniae. There are several hints in this history and investigations to suggest Mycoplasma pneumoniae as the likely cause. First of all, he is a young and fit patient who describes a non-productive cough preceded by flu-like symptoms and not responding to penicillins. Secondly, he has anaemia which may be due to haemolysis, and finally there is bilateral consolidation on the chest X-ray. Klebsiella pneumoniae is more typical in alcoholics, Legionella pneumophila is associated with a history of foreign travel, Chlamydia psittaci is associated with contact and exposure to birds and finally, Coxiella burnetii is rare and associated with farm animal exposure.

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A 35-year-old gentleman presents to the emergency department with worsening shortness of breath on exertion. His symptoms have been progressing for two weeks, and he now finds it difficult to take a full breath. He has no chest pain and has not been coughing but he has been feeling increasingly fatigued. He has a past medical history of HIV. He has been struggling to take his tablets recently. He is unsure of the names of any tablets, but he knows that he is supposed to take one tablet each day at the same time and to take two tablets twice a day on Mondays, Wednesdays, and Fridays. He has no other medical problems.

On examination, auscultation shows normal breath sounds. Observations:

Saturations	93%
Respiratory rate	22/min
Blood pressure	136/71mmHg
Heart rate	91/min
Temperature	37.6°C

On mobilisation, his saturations reduce to 88%. Chest X-ray is clear.

What investigation is most likely to be diagnostic?

A. CT pulmonary angiogram
B. High resolution CT chest
C. ECG
D. Echocardiogram
E. Broncho-alveolar lavage

E. Broncho-alveolar lavage

The correct answer is broncho-alveolar lavage which is most likely to be able to demonstrate PCP characteristic cysts with silver staining. There are several factors highly suggestive of PCP. This gentleman is poorly compliant with his HIV medication and the other medication regime he is describing is PCP prophylaxis, suggesting a low CD4 count. He also is tachypnoeic with low saturations on mobilising and a clear chest X-ray. All of these factors suggest PCP. In the case of a productive cough, sputum could be obtained without the need for a broncho-alveolar lavage. Imaging can be suggestive of PCP but is not as sensitive or diagnostic as broncho-alveolar lavage. ECG and echocardiogram have no role in PCP diagnosis.

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A 42-year-old woman presents to the emergency department with a dry cough. Over the last five days, she has been suffering from a worsening dry cough, shortness of breath that is worse on exertion, and she has felt hot. She has a past medical history of HIV which was recently diagnosed, but no other medical problems. She has been started two months ago on antiretroviral treatment and PCP prophylaxis, to which she reports good compliance.

Fundoscopy shows white branches alongside blood vessels. She denies any eye pain or change in her vision.

Observations:

Saturations	93%
Respiratory rate	20/min
Blood pressure	131/70mmHg
Heart rate	89/min
Temperature	38.1°C

Blood tests:

Hb	122 g/l
Platelets	401 * 10 /l9
WBC	10.2 * 10 /l9
Na^+	138 mmol/l
K^+	4.2 mmol/l
Urea	5.1 mmol/l
Creatinine	71 µmol/l
HIV viral load	12,198 copies/ml
CD4 count	70 cells/mm^3

A chest X-ray shows bilateral interstitial infiltrates. A broncho-alveolar lavage is organised but shows no organisms on microscopy, including with silver staining. PCR studies are pending.

What is the most appropriate treatment?

A. Prednisolone
B. Low-molecular weight heparin
C. Tazocin
D. IV co-trimoxazole
E. Ganciclovir

E. Ganciclovir

The correct answer is ganciclovir as this patient is likely to have CMV pneumonitis. Prednisolone would be more appropriate during PCP treatment, heparin would be appropriate if a pulmonary embolus was expected, tazocin is a broad-spectrum antibiotic used in many bacterial infection whilst co-trimoxazole is first line treatment for PCP. There are are several factors strongly suggestive of CMV as the cause of infection; fever with desaturation in poorly controlled HIV and evidence of retinitis as well. 'Chest X-ray of bilateral interstitial infiltrates is highly suggestive too. The diagnosis should be confirmed by a biopsy or broncho-alveolar lavage which can use PCR to amplify CMV. The negative silver staining makes PCP less likely as well.

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A 71-year-old gentleman presented to the emergency department with a headache, myalgia and fever. These symptoms all started one day ago. He has a past medical history of hypertension, ischaemic heart disease, type 2 diabetes, congestive cardiac failure and bilateral hip replacement. His medications include amlodipine, ramipril, aspirin, atorvastatin, gliclazide, calcium carbonate with colecalciferol (Adcal-D3) and paracetamol. He denies any allergies. He is concerned that he about influenza as he visited a friend in hospital last week who had been diagnosed with influenza A.

On examination, he appears sweaty and feels hot to touch. His chest is clear on auscultation. He is placed in a side room for observation and viral throat swabs are requested.

Observations:

Oxygen saturation	96% on room air
Respiratory rate	18/min
Blood pressure	129/93mmHg
Heart rate	77/min
Temperature	38.1°C

What is the most appropriate treatment?

A. Supportive management only
B. Salbutamol
C. Zanamivir
D. Amantadine
E. Oseltamivir

E. Oseltamivir

The correct answer is oseltamivir. This is a patient presenting with symptoms of influenza following recent exposure. The first aspect to consider is if he needs any antiviral drugs. Since he presented within 48 hours of the onset of symptoms and is in an at-risk group because of his diabetes he would warrant antiviral treatment and therefore supportive management is not the most appropriate treatment. Salbutamol is not indicated for flu alone. Zanamivir is second line treatment for influenza, whilst amantadine is no longer recommended for use in the treatment of influenza.

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An 82-year-old gentleman inpatient is reviewed on the ward round. He originally presented with diarrhoea on a background of ulcerative colitis flare and is managed with oral and rectal mesalazine. His bowel has settled but he developed a rash around his left antecubital fossa, and he has developed a temperature of 38.2ºC. On examination, his chest sounds clear and his abdomen is soft, but the left antecubital fossa has a tender and warm erythematous rash. Stool results show no growth, abdominal and chest X-ray are clear but a MRSA swab from the nose is positive.

What is the most appropriate treatment?

A. Mupirocin and chlorhexidine
B. Benzylpenicillin
C. Tazocin
D. Vancomycin
E. Flucloxacillin

D. Vancomycin

The correct answer is vancomycin. This is a patient with a new fever and the most obvious cause is cellulitis. Given that the location of the cellulitis is in a location common for cannulation the most likely explanation is iatrogenic cellulitis. This makes MRSA a much higher likelihood and therefore the most appropriate treatment is vancomycin. Mupirocin and chlorhexidine are used to eradicate MRSA when present as commensal organisms. In non-iatrogenic cellulitis benzylpenicillin and flucloxacillin are commonly used.

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A 32-year-old gentleman presents to the emergency department with severe nausea, vomiting and diarrhoea. He was recently at a reunion where 18 out of 25 guests have developed similar symptoms shortly afterwards. On examination he appears clinically dehydrated but his vital parameters are all normal. He has no past medical history and otherwise his examination is normal.

You suspect norovirus; what is the most appropriate investigation?

A. Serum serology
B. Faecal or vomitus toxicology
C. Faecal or vomitus viral PCR
D. Serum toxins
E. Serum viral PCR

C. Faecal or vomitus viral PCR

The correct answer is faecal or vomitus viral PCR which can confirm norovirus infection. Tests can detect antibodies by the ELISA method but these lack sensitivity and specificity and are therefore seldom used in the clinical setting. Norovirus does not make a toxin and therefore there is no such thing as norovirus toxin testing. The virus is not normally present in the bloodstream and therefore serum is likely to be negative for PCR testing. Norovirus is a viral cause of gastroenteritis commonly associated with large outbreaks in winter.

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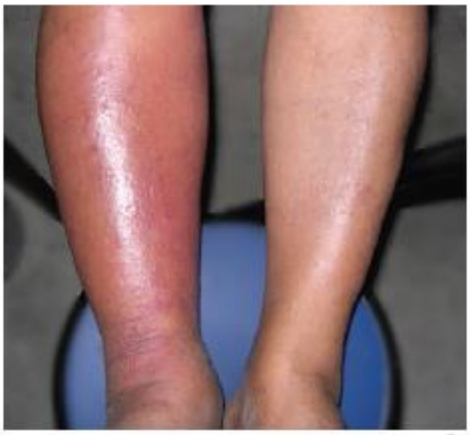
A 69-year-old woman with diabetes and rheumatoid arthritis presents to the clinic complaining of malaise. She takes metformin, sulfasalazine and hydroxychloroquine daily and methotrexate once a week. She volunteers at a care home near her home. On examination, she appears frail, and her fingers and toes are deformed. She is alert, afebrile and comfortable at rest. Her pulse rate is 70 beats per minute, BP is 120/80 mmHg, and respiratory rate is 16 breaths per minute. On inspection of her legs, you notice the following:



What is the most appropriate next step in the management of this patient?

A. Admit and treat with intravenous antibiotics
B. Order for an x-ray of the limb
C. Order for blood cultures and review with reports after one week
D. Prescribe oral antibiotics and arrange a follow-up visit for the next week
E. Prescribe oral prednisolone and arrange a follow-up visit

A. Admit and treat with intravenous antibiotics

The most appropriate next step is to **admit and treat with intravenous antibiotics**. The image provided shows a swollen and erythematous right lower limb. Differential diagnoses for a swollen limb include cellulitis, deep vein thrombosis (DVT), lymphoedema, and fluid overload conditions such as chronic heart failure. Cellulitis should be suspected when there is a history of fever and malaise, with superficial bullae potentially observed over the affected area. DVT typically presents in patients who have risk factors (such as recent immobilisation, surgery, or pregnancy) with unilateral limb oedema, erythema, and tenderness. Lymphoedema often has an insidious onset and manifests as a slowly progressive swelling of a limb; it is commonly observed in patients postlymph node dissection proximal to the affected area. Fluid overload states usually present with bilateral pitting pedal oedema without associated warmth or tenderness.

In this case, we are presented with a frail, immunocompromised woman exhibiting symptoms of right leg cellulitis. These characteristics'frailty and an immunocompromised state'are indications for in-patient management according to NICE guidelines on admission criteria for cellulitis. It is important to note that methotrexate is typically withheld for patients who are being hospitalized with severe infections requiring intravenous antibiotic therapy.

Ordering an x-ray of the limb is not correct. While cellulitis is diagnosed clinically, an x-ray may be warranted only if there is strong clinical suspicion of osteomyelitis in the underlying bone; otherwise, it does not play a role in the management of cellulitis itself. Ultrasound (USG), computed tomography (CT), and magnetic resonance imaging (MRI) can help exclude deeper tissue infections such as myositis or detect abscess formation.

Ordering blood cultures and reviewing after one week is inappropriate for this scenario. This patient has an acute infection necessitating immediate antibiotic treatment. Blood cultures are indicated when sepsis is suspected but should not delay empirical antibiotic therapy aimed at preventing infection progression.

Prescribing oral antibiotics and arranging follow-up is incorrect for this patient's presentation. Given her frailty and immunocompromised status, she requires hospital admission for intravenous antibiotic therapy rather than outpatient management with oral antibiotics, which are reserved for Eron class I and II cases of cellulitis.

Prescribing oral prednisolone would be ill-advised here as it could exacerbate the infection by suppressing the immune response instead of treating it. Prednisolone may be indicated in patients experiencing acute rheumatoid arthritis flares but is not applicable to this clinical situation.

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A 14 month-old Syrian girl is brought to the health centre by her mother. They are living in a Jordanian refugee camp after fleeing violence in their country. She has a widespread maculopapular rash that first affected the face before spreading to the torso and limbs. On examination, she has a cough, rhinorrhoea and conjunctivitis bilaterally. White spotting is seen on her buccal mucosa. The mother reports that her other children have had similar symptoms.

Which is the most appropriate therapy?

A. Azithromycin
B. Vitamin A
C. Vitamin B1 (thiamine)
D. Aciclovir
E. Reassurance

B. Vitamin A

The child has a classical case of measles, showing signs of the three C's (Cough, Coryza & Conjunctivits). Although usually self limiting, measles can have severe consequences. All children in developing countries should receive two dose of vitamin A. This replenishes the low levels of vitamin A caused be measles which can lead on to diarrhoea, corneal perforation and secondary bacterial pneumonia. Vitamin A has been shown to reduce deaths from measles by 50%.

Passive immunity from maternal antibodies protects infants under the age of 1 from measles.

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A 23-year-old white Caucasian left-handed male presents with a 24-hour history of slurred speech. He is a 'frequent flyer' in the Emergency Department with repeated admissions and known is a known user of intravenous drugs and alcohol. His past medical history includes depression and previously treated in adolescence for functional neurological symptoms. He denies having had a drink for the past 2 weeks but used intravenous cocaine yesterday.

On examination, he is obviously unkempt and a lack of prominent veins. There is an old abscess scar over his right femoral crease. His speech is markedly slurred but he remains orientated to time and place. You also note a significant loss of forehead creasing and facial weakness on both sides of his face with normal facial sensation. In addition, his power his 4-/5 in shoulder abduction, shoulder adduction, hip flexion and hip extension, with 5/5 power on all other movements. Reflexes were present in biceps, supinator, patella and ankle, with absent triceps reflex. Both plantar reflexes were downgoing. Sensation to cotton wool, proprioception and pinprick was normal, with the patient reporting no pain.

What is the most likely diagnosis?

A. Heavy metal intoxication
B. Guillain Barre syndrome
C. Botulism
D. Chronic inflammatory demyelinating polyneuropathy (CIDP)
E. Myasthenia gravis

C. Botulism

The patient has presented with bilateral cranial neuropathies and symmetrical proximal weakness, in the context of intravenous drug use. The descending pattern of weakness is atypical for GuillainBarre syndrome. The time course of within 8 weeks also rules out CIDP. Myasthenia gravis is a possibility but there is no suggestion of fatigability. The distinction between heavy metal intoxication and botulism is difficult: intravenous drug users, particularly those sourcing from Eastern Europe, who have been known to present with manganese poisoning due to dirty constituents. Clinical features of course depend on the precise metal involved but acute poisoning typically includes encephalopathy, gastrointestinal upset and myalgia in addition to peripheral neuropathy. Significant sensory neuropathy causing neuropathic pain is commonly prominent. In the case of this patient, with bilateral cranial neuropathies and symmetrical proximal weakness, in the context of intravenous drug use, makes botulism most likely. Treatment is with antitoxin, removal of the original source of infection in addition to close monitoring of respiratory function.

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A 45-year-old male is brought into the emergency department by his partner. He is known to be HIV +ve and his partner reports good compliance with his medications since his diagnosis 4 years ago. A collateral history reveals persistent confusion over the past 3 weeks. His past medical history includes outpatient treatment 2 years ago for lymphogranuloma venereum and type 2 diabetes mellitus. On examination, the patient is not orientated in time or place. He scores 0/10 on the abbreviated mental test. Both heart sounds are present, include a mild early diastolic murmur. Neurological examination is difficult due to poor patient compliance but you note absent reflexes in both lower limbs, with an upgoing plantar on the left and withdrawn plantar on the right. You also note that he has erythematous soles on both feet. Blood tests and blood glucose are awaited.

What is the most likely diagnosis?

A. Subacute combined degeneration of the cord
B. Motor neurone disease
C. Neurosyphilis
D. Hypoglycaemia and diabetic peripheral neuropathy
E. HIV dementia and peripheral neuropathy

C. Neurosyphilis

A number of features suggest neurosyphilis from the history. Firstly, the likely aortic regurgitation on cardiovascular examination should make you suspicious of aortitis, of which syphilitic infection is a classic cause. He has had a range of sexually transmitted diseases. A painless red rash is also a classic sign of syphilis, particularly in palms of the hands or the soles of the feet. He also has a combined upgoing plantar with absent ankle jerks, which narrows the diagnosis to the classic five MRCP favourite of motor neurone disease, tabes dorsalis, subacute combined degeneration of the cord, Friedrich's ataxia and dual pathology of central and peripheral causes. There is little to suggest a genetic disorder presenting in his mid-40s or a vitamin B deficiency to account for SCDC. Neurosyphilis would be the most likely diagnosis.

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A 22-year-old lady, with lupus and antiphospholipid syndrome, presented to the Rheumatology clinic with sharp chest pain across her right side.

This pain had started 3 days prior to presentation. She had some shortness of breath. She reported that she has a vesicular rash that had started 1 day ago over that area.

She weighs 65KG Her medications include:

- Methotrexate 20mg once weekly
- Prednisolone 30mg once a day
- Aspirin 75mg once a day
- Folic Acid 5mg once a day

She was admitted to the rheumatology ward and underwent a CT pulmonary angiography which showed no pulmonary emboli, but widespread bilateral changes of subsolid nodules and groundglass opacification.

Her shortness of breath started to get worse and her observations were taken. Observations:

Temperature	39.1ºC
Blood pressure	107/55mmHg
Heart rate	122beats/min
Oxygen saturation	88% on air

What is the best treatment for her likely diagnosis?

A. Treat with dalteparin subcutaneously
B. Treat with intravenous amoxicillin and oral clarithromycin
C. Treat with intravenous amoxicillin
D. Treat with aciclovir intravenous
E. Treat with aciclovir orally

D. Treat with aciclovir intravenous

The most likely diagnosis for this immunocompromised lady with sharp pain and a vesicular rash is varicella zoster chickenpox. She is systemically unwell with it and has widespread pulmonary changes that are consistent with varicella pneumonitis.

The treatment of choice in an immunocompromised host with varicella zoster is intravenous aciclovir.

Answers 1,2 and 3 would not treat the varicella zoster infection.

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A 32-year-old lady of Ugandan origin presents to the HIV clinic.

She has been known to the HIV service since coming to the UK 10 years ago. She currently takes tenofovir, emtricitabine, and dolutegravir. She is extremely compliant with her current regime and is currently well.

She is 14 weeks pregnant and this is her first pregnancy. Despite some vomiting earlier in the pregnancy, she is excited and feeling well.

Her blood tests show:

CD4 count	0.84x10^9/L
CD8 count	1.01x10^9/L
Viral load	<20RNA/ml

She has come to discuss the management of her HIV with you.

What is the most appropriate management for this lady?

A. Stop this patients current regime and change her onto zidovudine, lamivudine and nevirapine
B. Continue the current medications, but at a lower dose so that the medications do notcross the placental barrier and effect the foetus
C. Continue current regime and advise patient she will need to deliver by caesarean sectionto prevent transmission during birth
D. Continue current management and advise patient there is currently no reason why sheshould not have a vaginal delivery
E. Stop all HIV medications and advise this patient she will have to deliver by caesareansection

D. Continue current management and advise patient there is currently no reason why sheshould not have a vaginal delivery

It is recommended that women conceiving on an effective antiretroviral (ART) regimen should continue this even if it contains efavirenz or does not contain zidovudine.

Exceptions are: (1) Protease inhibitor (PI) monotherapy should be intensified to include (depending on tolerability, resistance and prior antiretroviral history) one or more agents that cross the placenta. (2) The combination of stavudine and didanosine should not be prescribed in pregnancy

Although there is most evidence and experience in pregnancy with zidovudine plus lamivudine, tenofovir plus emtricitabine or abacavir plus lamivudine are acceptable nucleoside backbones. In the absence of specific contraindications, it is recommended that the third agent in ART should be efavirenz or nevirapine (if the CD4 cell count is less than 250 cells/L) or a boosted PI.

No routine dose alterations are recommended for ARVs during pregnancy if used at adult licensed doses.

With a viral load <50 at 36 weeks a vaginal delivery is recommended in the absence of any obstetric complications. Since her viral load has been undetectable for the last 6 years it is reasonable to assume this is likely to continue throughout her pregnancy. She will continue to need monitoring of her viral load throughout her pregnancy, particularly at 36 weeks.

In 2015 there were an estimated 1.5 million people living in Uganda with HIV.

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An 80-year-old gentleman presented with a 4 month history of feeling generally unwell, being more breathless and tired than usual, feeling feverish and having a dry cough. He had already been treated by the General Practitioner with 2 courses of antibiotics in the community with only temporary improvement. His past medical history includes previous myocardial infarction, permanent pacemaker (PPM) with box-change 8 months ago, hypertension, diabetes, anaemia and chronic kidney disease (stage 2). He is a lifelong smoker.

On examination his heart sounds were normal with no murmurs, he had scattered crackles and his abdomen was soft and non-tender. There was mild leg oedema and a faint purpuric rash on his shins. His vital signs revealed heart rate = 80 beats per minute, blood pressure = 130/70 mmHg, T=37.8oC, SaO2 = 96% on air and respiratory rate = 20 breaths per minute. His chest X-ray did not show and consolidation and his urine was clear.

The following blood tests have been obtained:

Hb	10.5 g/dl
MCV	95 fl
Platelets	160 * 10 /l9
WBC	13.4 * 10 /l9

Na +	132 mmol/l
K +	4.9 mmol/l
Urea	12 mmol/l
Creatinine	150 µmol/l
CRP	100 mg/l

Blood cultures grow coagulase negative staphylococci and you notice that during his previous admission in the hospital he also had positive blood cultures for coagulase-negative staphylococci.

What is the best next investigation?

A. Repeat blood cultures
B. Computed tomography thorax / abdomen / pelvis
C. Biopsy of the rash
D. Vasculitic screen
E. Urgent transoesophageal echocardiogram

E. Urgent transoesophageal echocardiogram

This question aims to raise awareness of the significance of blood cultures positive for coagulasenegative staphylococci.

Coagulase-negative staphylococci are often considered skin contaminant or apathogenic organisms. However, in the presence of prosthetic devices such as PPM, prosthetic heart valves, central venous lines and orthopaedic prostheses they are the commonest cause of infection causing device-related infection. This is because of their ability to establish biofilms on artificial layers. *Staphylococcus epidermidis* device-related infections: pathogenesis and clinical management, JPP 2008, 60:1551-1571

This patient presented with symptoms and sings suggestive of a chronic infection and had blood cultures positive for coagulase-negative staphylococci. The presence of the PPM and the purpuric rash indicate that the source of sepsis is possibly related to the medical device. The box-change also increases the risk of infection.

Repeating the blood cultures is a good idea but it does not help identify the source of sepsis. CT thorax / abdomen / pelvis is an investigation that can identify most sources of sepsis especially if there is a clinical suspicion of lung or intra-abdominal abscess. However, in this case, it will not be particularly useful to verify the source of sepsis. Vasculitis is less likely in that scenario and so biopsy of the rash or vasculitic screen should not be the next investigation.

Transoesophageal echocardiogram is the best investigation to rule out pacemaker-wire related infections. It requires a lot of expertise and it might not be readily available but even so it is the next best investigation in this scenario.

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A 19-year-old from Cameroon has moved to the UK to study nursing. She arrived three months ago. She has been having diarrhoea for a month and noticed a fleeting erythematous rash on her torso. Her GP orders a set of bloods, which reveal a dramatic eosinophilia. She has not lost weight and, if anything, seems concerned that she is becoming overweight since moving to the UK. She has been well previously with no allergies or medication. There is no significant family history but she tells you that her brother had an 'eye worm' last year.

A stool is sent for ova cysts and parasites and microscopy and culture. Multiple *Strongyloides stercoralis* larvae can be seen on charcoal culture. She is commenced on a seven-day course of Ivermectin.

Four days later she is brought into the Emergency Department, with a GCS of 6.

What is the diagnosis?

A. Disseminated Strongyloides infection
B. Co-infection with Onchocerciasis
C. Co-infection with Loa
D. Ivermectin related hypoglycaemia
E. Insulin abuse

C. Co-infection with Loa

The give away in this history is the family history of 'eye worm'. This suggests that she lives in an area of high Loa Loa endemicity.

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A 24-year-old Caucasian girl returned from a two week scuba diving holiday in Honduras four days ago. She had not taken malaria prophylaxis. She had been drinking alcohol moderately during her trip. She had unprotected sex during her holiday with her diving instructor. Yesterday she had felt hot and cold with pain all over her body and a headache. Today the pain is worst behind her eyes. It is a constant pain and there is no photophobia or neck stiffness. She has an erythematous rash over her trunk and back with patches of white skin surrounded by erythema. There is no lymphadenopathy

She is observed in hospital for 48 hours and tests for HIV (including PCR and antibody), malaria and blood cultures were negative. Further tests remain unreported.

She recovers and is feeling much better. Prior to discharge, she asks if she is safe to go back to Honduras next year.

What should you advise?

A. She should not go back to any Dengue endemic area as there is a risk of Dengue haemorrhagic fever (with a 20% mortality) with a second exposure to Dengue virus
B. She can go back to Honduras, although there is a small risk of Dengue haemorrhagic feverwith a second exposure, the risk is small enough not to be overly worried. She should takeprecautions not to be bitten by mosquitos during the day
C. She has had Dengue fever so will be immune to Dengue fever on subsequent exposure
D. She has had flu and therefore she can go back to Honduras next year
E. Staying in the UK from now on would be the most sensible given that she is at high risk ofmalaria, HIV and Dengue fever from her behavioral tendencies

B. She can go back to Honduras, although there is a small risk of Dengue haemorrhagic feverwith a second exposure, the risk is small enough not to be overly worried. She should takeprecautions not to be bitten by mosquitos during the day

There are four serovars of Dengue (1-4) and therefore infection with one serovar will not confer immunity to the other serovars. There is a theoretical risk that consequent infection with serovar 2 following infection with serovar 1 will increase your risk of progression to Dengue Haemorrhagic Fever. However, the risk is far smaller than other risks posed by travelling (e.g road traffic accident, acquiring HIV from unprotected sex etc.) Therefore universal travel advice (including avoidance of day biting mosquitos) is appropriate.

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You are called to see a 50-year-old Indian gentleman who has presented to the Emergency Department (ED) from Heathrow airport. He gives a 3 week history of a brown productive cough associated with fevers and drenching nights sweats. He denies any weight loss or haemoptysis, however his wife is concerned that his sputum has been very brown/red in colour since his illness started. On further questioning you find that the patient is a resident of a rural community in north east India and is visiting family who live in London. He is a non-smoker and had been in very good health apart from a bout of diarrhoea several weeks ago.

On examination of the patient he appears relatively well, his heart rate is 95 beats per minute and respiratory rate is 20 breaths per minute. You note that he has a productive cough, producing dark brown sputum which you send for routine microscopy sensitivity and culture (MC&S) and acid fast bacilli (AFB) and tuberculosis PCR. On auscultation of his chest you hear course crepitations at the right base with some bronchial breathing. The remainder of his physical examination is unremarkable.

You perform a number of investigations described below

Hb	110 g/l	Na^{+}	138 mmol/l
Platelets	500 * 10 /l 9	K^{+}	4.5 mmol/l

WBC	16.0 * 10 /l 9	Urea	6.0 mmol/l
Neuts	10.0 * 10 /l 9	Creatinine	99 µmol/l
Lymphs	2.0 * 10 /l 9	CRP	55 mg/l
Eosin	2.0 * 10 /l9		
Sputum MC&S	Negative		
Sputum AFB	Negative		
Chest X-Ray	Right lower zone consolidation		

What is the likely diagnosis?

A. Tuberculosis
B. Streptococcal pneumonia
C. Paragonimiasis
D. Malignancy \
E. Pulmonary hemorrhage

C. Paragonimiasis

The diagnosis is paragonimiasis or lung fluke infection.

Background

Paragonimiasis is a common tropical infection, the presentation of which can often be misinterpreted as tuberculosis. Patients classically present with a productive cough of brown / red sputum (which can often be mistaken for haemoptysis). Patients may also exhibit constitutional symptoms, however, these are often less severe than in tuberculosis or malignancy. Furthermore, paragonimiasis is often associated with an eosinophilia.

Although more commonly associated with East Asia, paragonimiasis is also prevalent in North East India, Africa and parts of the Americas.

Treatment of paragonimiasis is with praziquantel.

Question 188 of 277

You are called to the Emergency Department to review a 60-year-old Indian female who returned from a six month trip to the north east of India five days ago. She presented with a three week history of a productive cough, producing large quantities of brown sputum. She has also been experiencing fevers daily and complains of being extremely sweaty over night. On further questioning there is no history of weight loss or TB contacts. The patient reports that as she has felt relatively well with these symptoms she had not presented earlier. It was, in fact a concerned relative who thought the patient had coughed up blood who had brought her in to hospital today.

Examination reveals a comfortable patient with evidence of a right lower zone pneumonia. The is confirmed on chest radiograph. You send a number of investigations including sputum for routine culture and acid fast bacilli. The results of which are shown below:

Hb	120 g/l	Na +	139 mmol/l
Platelets	350 * 10 /l 9	K +	3.9 mmol/l
WBC	13.0 * 10 /l 9	Urea	8.0 mmol/l
Neuts	8.0 * 10 /l 9	Creatinine	110 µmol/l
Lymphs	1.5 * 10 /l 9	CRP	35 mg/l
Eosin	1.8 * 10 /l9		
Sputum MC&S	Negative		
Sputum TB PCR	Negative		

What is the most appropriate treatment for this patient?

A. Rifater and ethambutol
B. Amoxicillin
C. Praziquantel
D. Augmentin and clarithromycin
E. Fluconazole

C. Praziquantel

In this case the diagnosis is paragonimiasis, or lung fluke infection. Paragonimiasis is a common tropic al disease, which often presents in a similar fashion to tuberculosis. The patient will have a productive cough of classically brown or red sputum (which may be misinterpreted as haemoptysis). They often will also present with fever and night sweats as well as rashes or urticaria. Constitutional symptoms will often not be as severe as in tuberculosis and the patient may also have an eosinophilia.

Treatment of paragonimiasis is with praziquantel

Question 189 of 277

As the medical registrar on-call you are called to see a 25-year-old male in the Emergency Department (ED). The young male presented to the ED with a fever and productive cough of green sputum. When you arrive he looks unwell and although alert is a difficult historian.

Concerned, you immediately assess him from head to toe:

Airway	Airway patient, able to speak in sentences
Breathing	Sats 90% on room air, Resp Rate 25 / min, course creps bilaterally on auscultation
Circulation	Heart rate 110 beats per minute, blood pressure 90/55 mm/Hg
Disability	Glasgow coma sclae 15/15, capillary blood glucose 5.5
Exposure	Multiple boils noted distributed widely across the patients body

You inquire about the history of the boils identified on examination. The patient is unsure how long he has had them for, however, mentions that two of his family members recently were prescribed antibiotics by their family GP for similar lesions.

You start the patient on high flow oxygen, gain IV access taking blood cultures and give a fluid bolus. An urgent portable chest X-ray is requested which appears to show bilateral consolidation with multiple cavitating lesions.

What is the most likely diagnosis

A. Streptococcal infection
B. PVL-producing staphylococcal infection
C. Tuberculosis
D. Fungal chest infection
E. Klebsiella infection

B. PVL-producing staphylococcal infection

Panton-Valentine Leukocidin is a pore-forming toxin which is produced by staphylococcus aureus. It has an affinity for white blood cells and the endothelium. Clinically PVL will often present with a necrotising pneumonia, characterised by severe bilateral pneumonia with cavitations on X-ray. Often patients presenting will have a history of boils or necrotic skin lesions. As the staphylococcus is easily transmitted there may be a recent family history of similar infections / boils.

Although all of the answers in this stem could be associated with cavitating pneumonia, the severity of the clinical presentation and history of boils in the family points towards a PVLpneumonia.

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A 36-year-old male from India presents with worsening headache and vomiting. He is known to have HIV but has been lost to follow up over the last 5 years. His CD4 count from his last appointment was 400.

On examination he has no localising signs however fundoscopy revealed papiloedema. Investigations:

haemoglobin	115 g/L (115-165)
white cell count	9.4 Ã— 109/L (4.0-11.0)
platelet count	220 Ã— 109/L (150-400)
erythrocyte sedimentation rate	50 mm/1st h (<30)
serum urea	7.0 mmol/L (2.5-7.0)
serum creatinine	105 mol/L (60-110)
serum alanine aminotransferase	17 U/L (5-35)
serum aspartate aminotransferase	26 U/L (1-31)
Cryptococcal antigen test	Negative
CD4 Count	65 cells/mm3
CT Head	5cm ring enhancing lesion in right temporal lesion with surrounding oedema

What is most appropriate management?

A. Amphotericin, Fluconazole and prednisolone
B. Rifampicin, Isoniazid, Pyrazinamide, Ethambutol and prednisolone
C. Trimethorpim-Sulphamethoxazole, Rifampicin, Isoniazid, Pyrazinamide, Ethambutol andprednisolone
D. Start prednisolone but defer treatment until lumbar puncture performed
E. Pyrimethamine, Sulfadiazine, Rifampicin, Isoniazid, Pyrazinamide, Ethambutol, andPrednisolone

E. Pyrimethamine, Sulfadiazine, Rifampicin, Isoniazid, Pyrazinamide, Ethambutol, andPrednisolone

The question represents a real diagnostic conundrum. You should automatically think of compliance issues given that the patients has defaulted from treatment and is therefore unlikely to have been controlling his HIV. The CD4 count of 400 is therefore not going to be representative of his current immune system.

This leads us to the differential diagnosis of a intra-cerebral ring enhancing lesion in a severely immunocompromised host. This includes tuberculosis, lymphoma, toxoplasmosis and cryptococcus amongst others.

Amphotericin and fluconazole is used in the initial 2 week phase of treatment for cryptococcal meningitis. However we are told that the cryptococcal antigen test is negative. This test has a relatively high sensitivity and specificity is therefore reassuring.

We are also told that the patient has papilloedema and a 5cm lesion with oedema, as a result attempting a lumbar puncture at this early stage is fraught with risk. Although vital to diagnosis it would be prudent to reduce the intra-cerebral swelling first by which time the patient may have seriously deteriorated, therefore empirical treatment becomes a priority.

Diagnosis of CNS tuberculosis is notoriously difficult and without access to CSF it is likely that empirical treatment will be required in the interim. When CSF is available culture, staining and PCR can be used to help detect the organism. Toxoplasmosis is treated with pyrimethamine and sulfadiazine as a first line. Again, empirical treatment may be required before access to CSF is available. As a result the correct answer is treatment of TB and Toxoplasmosis with steroids to reduce oedema.

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A 36-year-old Somali male presents on the medical take with a severe generalised headache associated with nausea and vomiting. He has been living in the UK for the last 10 years. He was diagnosed with HIV 5 years ago and has been well maintained on therapy with an undetectable viral load and a CD4 count of 500 cells/mm3.

He has a long history of headaches since the age of 10 which are normally controlled with simple analgesia. Over the last 2 days he has been suffering with a particularly bad attack which culminated in him becoming aggressive and vomiting profusely. He was noted to have a tonicclonic seizure whilst in accident and emergency which resolved with diazepam.

Clinical examination reveals a pulse of 78 beats per minute, a blood pressure of 130/90, oxygen saturations of 98% on air and a temperature of 36.8 degrees. There was no focal neurological defects.

A CT scan subsequently showed cystic and calcified lesions within the brain and mild hydrocephalus.

What is the most likely diagnosis?

A. Tuberculous meningitis
B. Cryptococcal meningitis
C. Lymphoma
D. Neurocysticercosis
E. Human african trypanosomiasis

D. Neurocysticercosis

Cysticercosis is caused by the larval stage of the tapeworm Taenia solium. Clinical syndromes that can arise include neurocysticercosis (NCC) and extraneural cysticercosis. In endemic areas NCC is an important cause of adult-onset seizures.

Cysticercosis is transmitted by ingestion of T. solium eggs shed in the stool of a human tapeworm carrier. Following ingestion, embryos (oncospheres) hatch in the small intestine, invade the bowel wall and disseminate hematogenously to the brain, muscle, liver and other tissues. Cysts located in the brain result in neurocysticercosis and can lead to hydrocephalus if they obstruct the outflow of CSF from the ventricles. The condition can be chronic and develop over a protracted period of time.

This question relies primarily on knowledge of the relevant condition and your ability to think beyond the fact that the patient has HIV. While the other options are all plausible you are given clues throughout the question on which you can discount each one until you are left with the correct answer.

The first hint is that his HIV was diagnosed early and is very well controlled. This makes an opportunistic infection incredibly unlikely therefore discounting crypotcocosis and TB meningitis. There are two forms of human african trypanosomiasis both transmitted by the tsetse fly, T.gambiense a chronic condition contracted in west africa and T.rhodiesiense that is acutely progressive and contracted in east africa. Given that he has been in the UK for 5 years we can discount T.rhodesiense and the finding of calcified lesions on the CT would be most unusual for T.gambiense.

This leaves us with lymphoma and neurocistercicosis, lymphoma will generally present with progressive focal neurology over a period of time and is unlikely to show calcified lesions in the brain.

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A 69-year-old man from Pakistan presents to his general practitioner with a three month history of cough associated with episodes of haemoptysis and has had some episodes of shortness of breath. His only past medical history of note is asthma for which he takes salbutamol inhaler as required and 2 puffs of beclomethasone inhaler twice daily. He has a 25 pack-year history and he is teetotal. The patient's GP performs blood tests and refers the patient to the rapid access chest clinic.

Blood test results come back as all within normal reference ranges, however, chest x-ray shows a solid lesion at the apex of the right lung, that is associated with a rim of air.

Of the following options, which is the most likely diagnosis?

A. Tuberculosis
B. Mesothelioma
C. Aspergilloma
D. Squamous cell carcinoma
E. Small cell lung carcinoma

C. Aspergilloma

The most likely diagnosis in this case is an aspergilloma - a fungal ball located in a previous cavity in the lung, as seen by the solid mass coupled with the rim of air and the haemoptysis, which can sometimes be severe.

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A 34 year-old man from South Korea presents with a two week history of abdominal pain in the right upper quadrant, fever and weight loss. The pain does not radiate anywhere and is 6/10 on the pain scale. Apart from the above he has no other symptoms. His only family history is his father who has hepatitis B and HIV. Examination reveals a 3cm tender palpable liver edge, pyrexia of 38.9ºC, pallor and right upper quadrant pain. There is no jaundice.

Blood tests reveal:

Bilirubin	29 µmol/l
ALP	200 u/l
ALT	90 u/l
γGT	100 u/l
Albumin	38 g/l

An ultrasound scan is requested and shows a 3x3cm abscess in the right lower lobe of the liver. The liver abscess is subsequently aspirated.

Apart from aspiration of the large liver abscess, what is the most appropriate treatment?

A. Blood cultures
B. Karyotyping
C. Metronidazole treatment
D. Liver biopsy
E. Mebendazole treatment

C. Metronidazole treatment

The most likely diagnosis in this case is an amoebic abscess, which are normally found in endemic tropical countries. Aspiration of large liver abscesses and a course of metronidazole is the preferred treatment for this condition.

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A 19 year-old University student presented to her General Practitioner with a three month history of intermittent diarrhoea and anorexia. She had lost approximately 10 kg of weight over a similar period. The patient had no previous abdominal symptoms and no significant past medical history. The symptoms had started several weeks after her return from a gap year expedition to Mali. On close questioning the patient did recall a short-lived intensely itchy erythematous rash on one of her feet while she was in Africa. This had spontaneously resolved and not subsequently recurred.

Initial investigations are listed below:

Hb	12.8 g/dl
Platelets	198 * 10 /l9
WBC	10.8 * 10 /l9
Neutrophils	5.2 * 10 /l9
Lymphocytes	2.1 * 10 /l9
Monocytes	0.8 * 10 /l9
Eosinophils	2.7 * 10 /l9
Basophils	0.0 * 10 /l9

Na +	136 mmol/l
K +	4.2 mmol/l
Urea	3.9 mmol/l
Creatinine	78 µmol/l

Stool microscopy: *Strongyloides stercoralis* larvae

Strongyloides stercoralis filariform larvae IgG: positive Under supervision from Infectious Disease department the patient was treated with Ivermectin.

What is the best method to assess for Strongyloides stercoralis eradication six months after completion of treatment course?

A. Repeat blood eosinophil count
B. Assessment of patient symptoms
C. Repeat Strongyloides stercoralis serology
D. Repeat stool microscopy on two occasions one week apart
E. Repeat stool microscopy with stool concentration

C. Repeat Strongyloides stercoralis serology

Repeat serological testing is the best method of confirming *Strongyloides stercoralis* eradication following treatment. A post-treatment to pre-treatment titre ratio of < 0.6 is taken to represent a good indicator of treatment success.

Blood eosinophil count is also often found to decrease following treatment although this method clearly lacks specificity given the numerous other causes of eosinophilia.

Strongyloides stercoralis infection is often asymptomatic therefore resolution of symptoms does not provide good evidence of successful treatment. While stool microscopy is the gold standard diagnostic test for strongyloidiasis it has a sensitivity of only 50 % making it an unreliable tool to confirm eradication. Stool concentration techniques are used in specialist laboratories to improve sensitivity of stool microscopy for strongyloidiasis.

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A 22-year-old man presented to his General Practitioner with an itchy rash across his arms and legs. Symptoms had developed within the last few days and were causing the patient significant discomfort and preventing him from sleeping. The patient was a university student currently enjoying his summer vacation. He reported returning from a backpacking trip around Eastern Europe one week previously when he had stayed mostly in youth hostels. Since his return he had been staying in his parent's home. Interestingly, the patient had discovered on Facebook that a similar rash had afflicted a friend he had made at the end of his holiday.

He also worked part-time as a barman and denied coming into contact with any unusual chemicals at work. He had not recently introduced any new skin care products.

The patient had suffered from mild atopic eczema as an infant but had no recent skin complaints or other past medical history. He took no regular medications and had no known allergies. The patient smoked 10 cigarettes per day and drank roughly 40 units of alcohol each week.

On examination, the patient's arms and legs were covered with 2-5 mm maculopapular lesions with a central haemorrhagic punctum. In several places, several lesions were noted to be lying close together in a curve formation. There were minor marks of excoriation associated with the lesions. The patient's groin, abdomen, chest and back were free from rash. There was no axillary, cervical or inguinal lymphadenopathy.

What organism is the likely cause for the patient's rash?

A. Sarcoptes scabiei
B. Leishmania donovani
C. Ancylostoma braziliense
D. Mycobacterium leprae
E. Cimex hemipterus

E. Cimex hemipterus

The patient has symptoms and signs of exposure to bed bug infestation. In temperate regions this is caused by the insect *Cimex hemipteru* (or *Cimex lectularius* in tropical regions). A rash develops up to 11 days after exposure due to an allergic reaction. Bed bug infestation has increased in frequency over the past 10 years with the patient likely exposed during a recent stay at youth hostel.

The other organisms are all potential causes of skin disease in the returned traveller.

- *Sarcoptes scabiei* (scabies)
- *Leishmania donovani* (cutaneous leishmaniasis)
- *Ancylostoma braziliense* (cutaneous larva migrans)
- *Mycobacterium leprae*(leprosy)

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A 45-year-old female presents to your clinic with bilateral lower limb lesions. She said that it initially started as a small nodule on the right shin which then broke down into an exudative ulcer. Over the past year she has developed widespread 'cauliflower-like' lesions over her whole body. On examination you note a painless ulcer with scab over the anterior right shin, and generalised papillomas over the face, trunk, genitalia and buttocks.

Investigations demonstrate:

TPHA (Treponema pallidum Haem Agglutination test)	Positive

What is the most likely diagnosis?

A. Pinta
B. Syphilis
C. Yaws
D. Pyoderma gangrenosum
E. Kaposi's sarcoma

C. Yaws

Treponemal specific antibody tests (e.g. TPHA) are very specific to Treponema but not necessarily syphillis, as the species Treponema can cause a variety of diseases including syphilis, yaws and pinta

Care must be taken when interpreting syphilis investigations. Cardiolipin tests (e.g. VDRL and RPR) are non-specific and can be positive in many diseases such as TB, malaria and HIV. Treponemal specific antibody tests (e.g. TPHA) are very specific to Treponema, however the species Treponema can cause a variety of diseases including syphilis, yaws and pinta.

Yaws is a chronic infection that affects mainly the skin, bone and cartilage. The disease occurs mainly in low socio-economic communities in tropical areas of Africa, Asia and Latin America. The causative organism is a bacterium called Treponema pertenue, a subspecies of Treponema pallidum that causes venereal syphilis. However, yaws is a non-venereal infection.

In primary yaws, a single skin lesion develops at the point of entry of the bacterium after 2-4 weeks. This nodule can break down into an exudative ulcer. Without treatment, secondary yaws can occur, resulting in multiple lesions appear all over the body, more commonly over the face, trunk, genitalia and buttocks. Later on in the disease course, widespread bone, joint and soft tissue destruction can occur.

Question 197 of 277

A 25-year-old HIV-positive male goes to his GP with complaints of headache and left-sided weakness of recent onset. His temperature is 38ºC, blood pressure is 115/70 mmHg, respirations are 14/min and pulse is 73/min.

Neurological examination reveals decreased power, hyperreflexia in the left upper and lower limb with associated upgoing plantars. CT head shows multiple ring-enhancing lesions.

What is the most appropriate next step in management?

A. Trimethoprim-sulfamethoxazole
B. Sulfadiazine and pyrimethamine
C. Brain irradiation
D. Brain biopsy
E. Start albendazole

B. Sulfadiazine and pyrimethamine

HIV, neuro symptoms, multiple brain lesions with ring enhancement - toxoplasmosis

The most common cause of central nervous system mass lesion in AIDS patients is toxoplasmosis. This patient's findings are consistent with cerebral toxoplasmosis. Trimethoprim-sulfamethoxazole is used for prophylaxis of toxoplasmosis, while sulfadiazine and pyrimethamine are used for treatment purposes.

Choice 3: Brain irradiation is used in the management of primary CNS lymphoma. Patients with primary CNS lymphoma are usually afebrile; lesions are weakly enhancing and are single (can be multiple). This is suspected if the patient does not respond to antibiotic therapy.

Choice 4: Brain biopsy is reserved for patient's whose lesions do not respond to treatment with sulfadiazine and pyrimethamine.

Choice 5: Albendazole is used for neurocysticercosis. This condition is not usually seen in AIDS patients.

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A 44-year-old man is reviewed in the renal clinic. He has a diagnosis of end-stage renal failure due to IgA nephropathy. The team is working him up for a renal transplant.

As part of his transplant workup, he has an interferon-gamma release assay performed:

Interferon-gamma release assay	Positive

He subsequently has further testing:

Nucleic acid amplification tests	Negative for *Mycobacterium tuberculosis*.

Sputum acid-fast bacilli smear	Negative for acid-fast bacilli

Chest X-ray	Lung fields appear clear. Normal cardiomediastinal contour.

What is the most appropriate management?

A. No treatment required
B. Pyridoxine for 6 months
C. Rifampicin and isoniazid (with pyridoxine) for 3 months
D. Rifampicin, isoniazid (with pyridoxine), pyrazinamide, and ethambutol for 2 months, thenrifampicin and isoniazid for 10 months
E. Rifampicin, isoniazid (with pyridoxine), pyrazinamide, and ethambutol for 2 months, thenrifampicin and isoniazid for 2 months

C. Rifampicin and isoniazid (with pyridoxine) for 3 months

Positive IGRA, but asymptomatic and normal CXR → treat as for latent tuberculosis

Rifampicin and isoniazid (with pyridoxine) for 3 months is correct. This patient has latent *Mycobacterium tuberculosis* infection: he has a positive interferon-gamma release assay but negative sputum for acid-fast bacilli and nucleic acid amplification tests as well as a normal chest x-ray. Latent *Mycobacterium tuberculosis* infection can either be treated with 3 months of rifampicin and isoniazid or with 6 months of isoniazid.

No treatment required is incorrect. This patient has a positive interferon-gamma release assay. This suggests either active or latent *Mycobacterium tuberculosis* infection. Given the negative nucleic acid amplification test, sputum smear for acid-fast bacilli, and normal chest x-ray, it is likely this patient has latent *Mycobacterium tuberculosis* infection. This requires treatment with either 3 months of rifampicin and isoniazid or 6 months of isoniazid alone.

Pyridoxine for 6 months is incorrect. Pyridoxine is a form of vitamin B6 and is used for the prevention of isoniazid-induced neuropathy. It is not in itself used as part of the treatment of *Mycobacterium tuberculosis* infection.

Rifampicin, isoniazid (with pyridoxine), pyrazinamide, and ethambutol for 2 months, then rifampicin and isoniazid for 10 months is incorrect. This is the treatment regimen for active *Mycobacterium tuberculosis* infection with CNS involvement. It is also usually accompanied by a course of steroids. The patient in this case, however, has latent *Mycobacterium tuberculosis* infection, evidenced by the positive interferon-gamma release assay but negative sputum for acidfast bacilli and nucleic acid amplification tests as well as a normal chest x-ray.

Rifampicin, isoniazid (with pyridoxine), pyrazinamide, and ethambutol for 2 months, then rifampicin and isoniazid for 4 months is incorrect. This is the treatment regimen for active *Mycobacterium tuberculosis* infection without CNS involvement. The patient in this question has latent *Mycobacterium tuberculosis* infection, so this answer is incorrect.

Question 199 of 277

A 48 year-old civil servant was referred to the Acute Medical Unit by his general practitioner with progressive cognitive impairment and severe lethargy over the last 3 weeks. He had also become intermittently agitated and his sleep was disturbed during the night. He had returned from visiting his partners relatives in Zimbabwe 1 month ago. He suffered from type II diabetes which was well controlled through dietary modification. He smoked 10 cigarettes a day and drank 14 units of alcohol per week.

On examination, his temperature was 37.2ºC, pulse was 60 beats per minute and blood pressure was 134/88. His Glasgow Coma Score was 14/15 due to confused speech. His trachea was central and right-sided posterior cervical lymphadenopathy was present. His jugular venous pressure was not elevated and chest was clear on auscultation. Heart sounds were normal.

Investigations:

Haemoglobin	143 g/L (130-180)
White cell count	7.7 x 109/L (4.0-11.0)
Neutrophil count	3.0 x 109/L (2.0-7.5)
Lymphocyte count	1.8 x 109/L (1.3-3.5)
Eosinophil count	0.4 X 109/L (0.1-0.4)
Platelets	310 x 109/L (150-400)

Sodium	134 mmol/L (135-145)
Potassium	4.8 mmol/L (3.5-5.0)
Urea	9.0 mmol/L (2.5-7.5)
Creatinine	100 mol/L (25-95)
Fasting plasma glucose	4.4 mmol/L (3.0-6.0)

CT head	No abnormality detected

What is the most appropriate next investigation?

A. MRI brain
B. Lymph node biopsy
C. Lumbar puncture
D. Stool microscopy
E. Blood film

C. Lumbar puncture

This patient has African trypanosomiasis, sometimes known as sleeping sickness. The acute presentation and country of origin suggest the East African variant, caused by *Trypanosoma bruicei rhodesiense*. Although the parasites may be detected in a peripheral blood film or lymph node biopsy, determining whether they have penetrated the central nervous system (CNS) will most influence choice of drug therapy. Pentamidine is the treatment of choice in early infection, while choices for CNS infection include suramin, melarsoprol, eflornithine and nifurtimox. These medications are not widely available and can result in extremely severe side effects, so are only prescribed by tropical medicine specialists.

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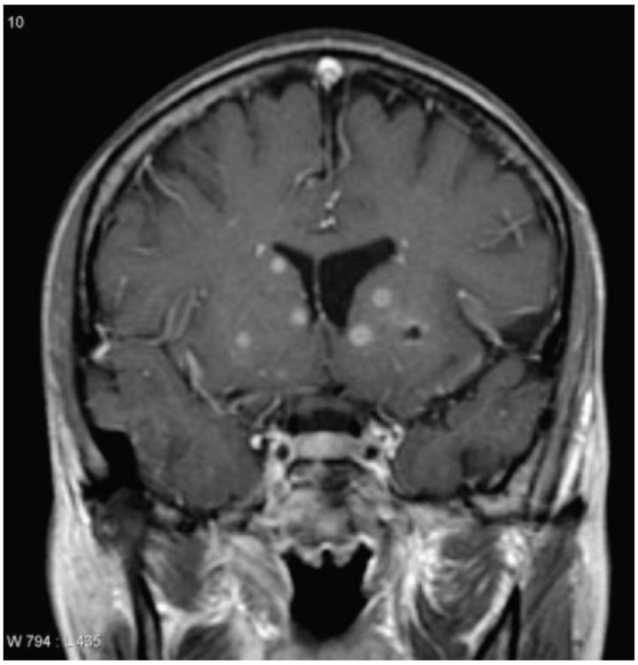
A 33-year-old man who has recently emigrated from Zimbabwe is admitted after being found confused at home. His partner reports that he has been acting strangely for a number of weeks and complaining of altered taste and dizziness. She reports that she is HIV positive but her partner refuses to be tested as he does not believe in 'medical lies'.

On admission he is apyrexial with a blood pressure of 114/82mmHg and pulse 78/min. Rapid HIV testing confirms that he has the infection. His CD4 count is 11 cells/µl. An MRI (T1 C+) shows the following:



Other than starting highly active antiretroviral treatment, what is the most appropriate treatment?

A. Amphotericin B
B. Surgical resection
C. Steroids + methotrexate + whole brain irradiation
D. Rifampicin + isoniazid + pyrazinamide + ethambutol + steroids
E. Sulfadiazine + pyrimethamine

E. Sulfadiazine + pyrimethamine

HIV, neuro symptoms, multiple brain lesions with ring enhancement - toxoplasmosis

The correct answer in this case is **sulfadiazine + pyrimethamine**. The patient's presentation with confusion, altered taste, dizziness, and a low CD4 count of 11 cells/µl suggests that he has an opportunistic infection due to his HIV infection. The MRI image provided shows multiple ringenhancing lesions, which are consistent with cerebral toxoplasmosis. Cerebral toxoplasmosis is caused by the protozoan parasite *Toxoplasma gondii* and is a common opportunistic infection in patients with advanced HIV disease. The recommended treatment for cerebral toxoplasmosis is a combination of sulfadiazine and pyrimethamine.

Amphotericin B is an antifungal medication used primarily to treat invasive fungal infections such as cryptococcal meningitis or disseminated histoplasmosis. Although these infections can occur in patients with HIV, the MRI findings and clinical presentation are more suggestive of cerebral toxoplasmosis.

Surgical resection might be considered for space-occupying lesions such as brain abscesses or tumours. However, given the patient's multiple ring-enhancing lesions on MRI and low CD4 count, cerebral toxoplasmosis is much more likely than a primary brain tumour or abscesses.

Steroids + methotrexate + whole brain irradiation would be a treatment approach for primary central nervous system (CNS) lymphoma, which can also present with neurological symptoms in patients with HIV. However, CNS lymphoma typically presents as a solitary lesion rather than multiple ring-enhancing lesions seen in this case.

Rifampicin + isoniazid + pyrazinamide + ethambutol + steroids represent the standard regimen for treating tuberculosis (TB). While TB can involve the CNS in patients with HIV, the MRI findings are not typical for CNS TB. Moreover, the patient's symptoms of altered taste and dizziness do not fit well with a diagnosis of CNS TB.

Question 201 of 277

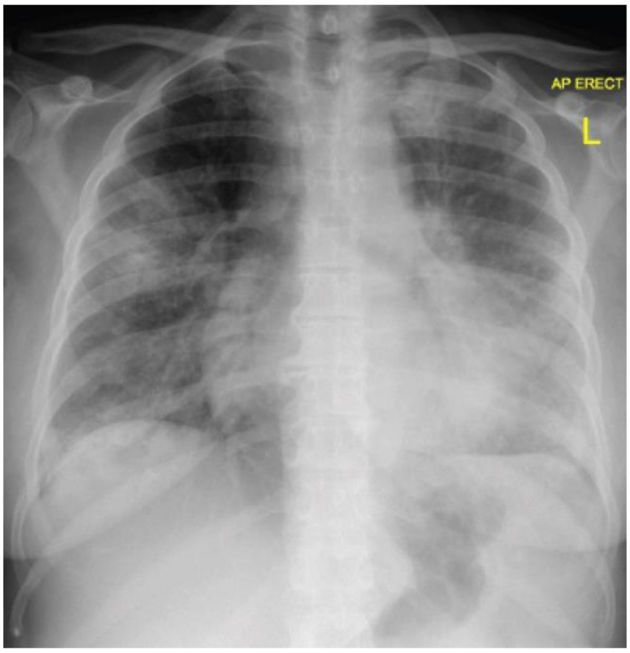
A 54-year-old woman presents to the Acute Medical Unit with a three day history of fever, myalgia and a non-productive cough. She initially thought her symptoms were due to the 'flu but over the past 24 hours has been feeling progressively more poorly. She now also has nausea, abdominal pain, headaches and diarrhoea.

On examination her pulse is 84/min, temperature 39.4ºC and blood pressure 106/76 mmHg. Bilateral scattered crackles are noted on auscultation.

Bloods show the following:

Hb	12.4 g/l	Na +	133 mmol/l
Platelets	363 * 10 /l 9	K +	5.0 mmol/l
WBC	12.1 * 10 /l 9	Urea	7.8 mmol/l
Neuts	10.8 * 10 /l 9	Creatinine	88 µmol/l
Lymphs	1.1 * 10 /l 9	CRP	145 mg/l
Eosin	0.2 * 10 /l9		

The chest x-ray is shown below:



Which investigation is most likely to be diagnostic?

A. Sputum culture
B. Cold agglutinins
C. Bronchoalveolar lavage
D. Blood serology
E. Urinary antigen

E. Urinary antigen

Legionella pneumophilia is best diagnosed by the **urinary antigen** test

The chest x-ray shows the asymmetrical, patchy bilateral consolidation typical of *Legionella*. This combined with high fever, hyponatraemia, abdominal pain and lymphopaenia make the diagnosis the most likely.

Question of 202 of 277

A 17-year-old man who has recently come to the UK from Rwanda is admitted to hospital. His friend describes him complaining of a headache and fever for the past four days. Further history reveals that he is very lethargic with a dry cough and generalised myalgia. The patient also describes passing some dark urine this morning. He has no past medical history of note. On examination his pulse is 110/min, temperature 38.1ºC, oxygen saturations 98% on room air and blood pressure 110/68 mmHg, His sclera are jaundiced and there is enlargement of the liver and spleen. Bloods show the following:

Na +	142 mmol/l
K +	4.8 mmol/l
Urea	12.3 mmol/l
Creatinine	144 µmol/l

What is the most likely diagnosis?

A. Typhoid
B. Dengue fever
C. African trypanosomiasis
D. Malaria
E. Leishmaniasis

D. Malaria

This patient clearly has an infective process so much of the data (headache, fever, myalgia, vital signs) can be discarded as being common to all the answers.

We're now left with more concrete findings: jaundice, hepatosplenomegaly and a raised creatinine. Now we take one of these findings, hepatosplenomegaly and consider the differential diagnosis:

Causes of hepatosplenomegaly

- chronic liver disease with portal hypertension
- infections: glandular fever, malaria, hepatitis
- lymphoproliferative disorders
- myeloproliferative disorders e.g. chronic myeloid leukaemia
- amyloidosis

This suggests that malaria is the most likely diagnosis. Jaundice and a raised creatinine are consistent with this.

Question 203 of 277

A 23-year-old woman presents to the emergency department. She was brought in by ambulance following left-sided weakness and difficulty in finding words. She also complains of a headache and nausea for the last three days. She has no past medical history and does not smoke nor does she drink alcohol. She recently emigrated from Tanzania to the UK.

On examination, she has marked weakness in upper and lower left limbs and is unable to walk without assistance. Her responses to questions are slow and limited.

Blood tests:

Hb	129 g/l
Platelets	316 * 10 /l9
WBC	8.9 * 10 /l9
Na +	141 mmol/l
K +	4.7 mmol/l
Urea	5.1 mmol/l
Creatinine	71 µmol/l
HIV-1 serology	positive
HIV-2 serology	negative
HIV viral load	pending
CD4 count	pending
Toxoplasmosis serology	pending
Cryptococcal antigen	pending

A CT scan of her head demonstrates multiple ring-enhancing lesions and mass effect. Dexamethasone is started immediately.

What is the most appropriate next step?

A. Antiretroviral treatment
B. Amphotericin
C. Methotrexate
D. MRI brain and whole spine
E. Pyrimethamine and sulfadiazine

E. Pyrimethamine and sulfadiazine

HIV, neuro symptoms, multiple brain lesions with ring enhancement - toxoplasmosis

The correct answer is pyrimethamine and sulfadiazine as the patient likely has cerebral toxoplasmosis as an AIDS-defining illness. Methotrexate is the treatment for a CNS lymphoma, alongside whole brain radiotherapy, but CNS lymphoma is unlikely since there are multiple lesions. Antiretroviral treatment will need to be started but should be done in specialist settings and not in the acute one. Further imaging would not be needed as there are sufficient evidence to start treatment at this point and would cause an unnecessary delay.

Question 204 of 277

A 36 year old male Intravenous drug user presents to genito-urinary clinic. He was diagnosed with HIV 3 years ago after presenting with tuberculosis (TB). He was treated for TB for 6 months. He is now on third line anti-retroviral therapy for previous virological failure and co-trimoxazole. His most recent CD4 count was 104, and his viral load was 3,000 copies/ml.

He complains of weakness on his left side, and deterioration in vision, getting worse for four weeks. He has a moderately severe headache. On examination his visual acuity is 3/60 in his left eye and 6/6 in his right eye. He has weakness, in his left arm and leg, brisk reflexes and mildly increased tone.

He is immediately admitted.

A CT scan shows several ring enhancing lesions in his right cerebral hemispheres and one on the left cerebral hemisphere.

On ophthalmological review he has a large area of retinal necrosis in his left eye.

What is the diagnosis?

A. CNS lymphoma
B. Cerebral toxoplasmosis
C. Cerebral TB
D. Cerebral abscesses
E. CNS Kaposi sarcoma

B. Cerebral toxoplasmosis

HIV, neuro symptoms, multiple brain lesions with ring enhancement - toxoplasmosis

The most important three differential diagnosis for a ring-enhancing lesion in an HIV patient is CNS lymphoma, TB and Toxoplasmosis.

In this case the retinal necrosis in characteristic of ophthalmic toxomplasmosis. Multiple lesion also favour toxoplasmosis. His poor compliance with HIV treatment (as indicated by the low CD4 count and the high viral load) suggest his compliance with co-trimoxazole is also poor.

Question 205 of 277

A 68-year-old man presents to hospital with progressive shortness of breath over the last four days and a low-grade fever. He has a past medical history of mild asthma for which he occasionally needs to use his salbutamol inhaler and has previously had bilateral knee replacements for osteoarthritis. He has smoked on average 5 cigarettes per day for the past 40 years and drinks a couple of glasses of wine per week. His travel history includes a holiday to Cyprus, from which he arrived back in the UK 5 days ago.

Examination revealed some right mid zone crackles and reduced breath sounds over this area. Observations revealed a temperature of 38.8ºC, heart rate of 110 bpm, blood pressure of 105/66 mmHg, respiratory rate of 22 breaths per minute and oxygen saturations of 91% on room air.

Blood tests revealed:

Hb	145 g/l
Platelets	290 * 10 /l9
WBC	9.4 * 10 /l9
Na +	132 mmol/l
K +	3.7 mmol/l
Urea	4.1 mmol/l
Creatinine	67 µmol/l

Urinary sodium concentration was measured and found to be 36mmol/L (normal range 40-220 mmol/d).

Which of the following investigations is most useful in the diagnosis of this condition?

A. Urine antigen test
B. Serum antibody test
C. Sputum PCR
D. CT pulmonary angiography
E. D-dimer

A. Urine antigen test

Legionella pneumophilia is best diagnosed by the **urinary antigen** test

This scenario is most likely due to the organism *Legionella pneumophila* (causing Legionnaire's disease) - an infection which is often due to air conditioning units and heating units. This patient may have got the infection when on holiday and his sodium is low, often a result of syndrome of inappropriate ADH secretion. The most accurate investigation for Legionella infection is via the urine antigen test, which is quick and specific.

Question 206 of 277

A 37-year-old gentleman presents to the Emergency Department with acute onset shortness of breath and right sided pleuritic chest pain. He is known to be HIV positive and his latest CD4 count is $190/mm^3$. The last clinic letter suggests he may be non-compliant with his anti-retroviral medication due to persistent diarrhoea. On examination, he has decreased breath sounds on the right with fine scattered crepitations in both lung fields. Oxygen saturations are 93% on breathing air. A chest x-Ray reveals a right sided pneumothorax measuring 1 cm at the hilum.

What pulmonary infection would you suspect?

A. Mycobacterium tuberculosis
B. Urine antigen test
C. Aspergillus fumigatus
D. Histoplasma capsulatum
E. Haemophilus influenzae

A. Urine antigen test

NICE guidance of pleural disease (pneumothorax):

- A spontaneous pneumothorax in someone known to be HIV-positive should warrant investigation for a diagnosis of *Pneumocystis jirovecii* pneumonia.

Invasive fungal infection (including *Pneumocystis jirovecii* pneumonia) is likely in this gentleman given the CD4 count $<200/mm^3$.

Question 207 of 277

An 81-year-old nursing home resident presents with a 48-hour history of pyrexia, myalgia and coryza. On examination, her temperature is 37.9ºC and she is visibly coryzal. Her chest is clear to auscultation. Her chest X-ray is clear. Her relatives inform you that the nursing home has recently been closed to visitors due to an Influenza outbreak.

Which of the following drugs should you also consider adding?

A. Empirical antibiotics for pneumonia
B. Zanamivir
C. Oseltamivir
D. Ribavirin
E. Salbutamol nebulisers

C. Oseltamivir

Oseltamivir (Tamiflu) is the first-line treatment recommended for patients with suspected or confirmed Influenza A. Ribavirin has not be shown to be efficacious in the management of influenza. Zanamivir is useful in patients with a poor swallow or in those with suspected or confirmed exposure to oseltamivir-resistant influenza.

Her chest X-ray is clear and therefore there is no role for the addition of antibiotic therapy at this stage.

Salbutamol nebulisers can be useful in patients with wheeze, however, appropriate isolation precautions should be secured prior to the administration of nebulisers in patients with Influenza infection.

Question 208 of 277

A 44 year-old lady presented to the medical outpatient clinic with leg swelling which had progressively increased over the last 2 years. Her past medical history consisted only of a hospital admission for malaria as a child. She had grown up in urban Nigeria and moved to the United Kingdom 6 years ago to work as a teacher, but had been forced to stop working due to mobility problems. She was a non-smoker and did not drink alcohol.

On examination, her temperature was 36.5ºC, heart rate 80 beats per minute, blood pressure 133/89 mmHg, respiratory rate 16 breaths per minute and oxygen saturations 98% on room air. Non-pitting leg swelling was apparent bilaterally extending proximally to the hips, with thickening of the overlying skin. Her chest was clear on auscultation and heart sounds were normal. The JVP was not elevated.

Investigations:

Haemoglobin	138 g/L
White cell count	7.0 * 10 /l9
Neutrophil count	4.8 * 10 /l9
Lymphocyte count	2.0 * 10 /l9
Eosinophil count	0.1* 10 /l9
Platelets	246 * 10 /l9

Sodium	142 mmol/L
Potassium	4.3 mmol/L
Urea	6.0 mmol/L
Creatinine	84 mol/L
Alkaline phosphatase	40 IU/L
Alanine aminotransferase	32 IU/L
Gamma-glutyl transferase	23 IU/L
Bilirubin	16 mol/L
Albumin	41 g/L
Fasting plasma glucose	5.3 mmol/L

What is the most likely causative organism?

A. Wuchereria bancrofti
B. Brugia malayi
C. Brugia timori
D. Onchocerca volvulus
E. Mansonella streptocerca

A. Wuchereria bancrofti

This patient has presented with elephantiasis caused by chronic lymphatic inflammation and blockage in the lower limbs. The condition is caused by infiltration of the lymphatic system by filarial nematodes, which are transmitted by mosquitoes. *Wuchereria bancrofti*, *Brugia malayi* and *Brugia timori* are all potential causative organisms, but *Wuchereria bacrofti* is by far the most common, accounting for 90% of cases. *Onchocerca volvulus* causes skin disease and river blindness, while *Mansonella streptocerca* causes skin disease alone.

Question 209 of 277

A 28-year-old male presents to the emergency department complaining of shortness of breath. For the last week he has felt generally unwell with a headache, malaise and lethargy, but felt breathless this afternoon hence his presentation. On systems review, he also admits to having some generalised abdominal pain throughout the day yesterday associated with diarrhoea. He is otherwise fit and well, takes no regular medications and does not smoke. He admits to drinking one or two beers every evening and occasionally more at the weekends. He is a primary school teacher and lives with his wife and two small children, none of whom have been unwell recently. On examination, he has a temperature of 38.9ºC, a heart rate of 105 beats/minute and a blood pressure of 105/70 mmHg. His respiratory rate is 26 breaths/minute, his oxygen saturations 92% breathing room air and on auscultation to his chest there are a few crepitations bibasally. He has some routine blood tests performed.

Hb	109 g/L
MCV	105 fL
Platelets	390 * 10 /L9
WBC	16.5 * 10 /L9
CRP	240 mg/L
Bilirubin	50 µmol/L
ALT	40 u/L
ALP	135 u/L

What is the most likely diagnosis?

A. Alcoholic liver disease
B. Gilbert's syndrome
C. Mycoplasma pneumonia
D. Hypothyroidism
E. Acute cholecystitis

C. Mycoplasma pneumonia

Mycoplasma pneumonia is one of the atypical pneumonias and classically occurs in clusters every few years. It normally produces a fairly mild picture with fairly normal clinical findings although the chest x-ray may have marked changes. It is associated with cold agglutinins in around 50% of cases which can result in a cold autoimmune haemolytic anaemia - as in this case, explaining the slight bilirubin rise and the raised MCV which represents a reticulocytosis. Other associations with mycoplasma infection include erythema multiforme, encephalitis, arthralgia and diarrhoea. Alcoholic liver disease is possible given this patients alcohol intake, bilirubin and MCV, but would be unlikely given his young age and no other clinical findings to support this. Gilbert's syndrome is hyperbilirubinaemia in the context of transient illness, viral or otherwise, and could explain many of the findings in this case but would not explain the raised MCV.

Question 210 of 277

A 39-year-old HIV-positive man complains of a two-week history of worsening headache, facial weakness and visual hallucinations. He also reports new onset eye pain. An MRI head reveals multiple ring-shaped contrast-enhancing lesions. He is regularly followed up in the HIV clinic and his latest CD4 count was 140 cells/mm³.

Which treatment should be started immediately?

A. Valganciclovir
B. Trimethoprim + sulfamethoxazole
C. Dexamethasone
D. Pyrimethamine + sulfadiazine
E. Ethambutol + rifampicin

D. Pyrimethamine + sulfadiazine

The symptoms and MRI findings are highly suggestive of toxoplasmosis, a disease caused by the protozoan *Toxoplasma gondii.*

A 6-month treatment regimen of sulfadiazine and pyrimethamine should be commenced immediately. Patients should be given lifelong secondary prophylaxis.

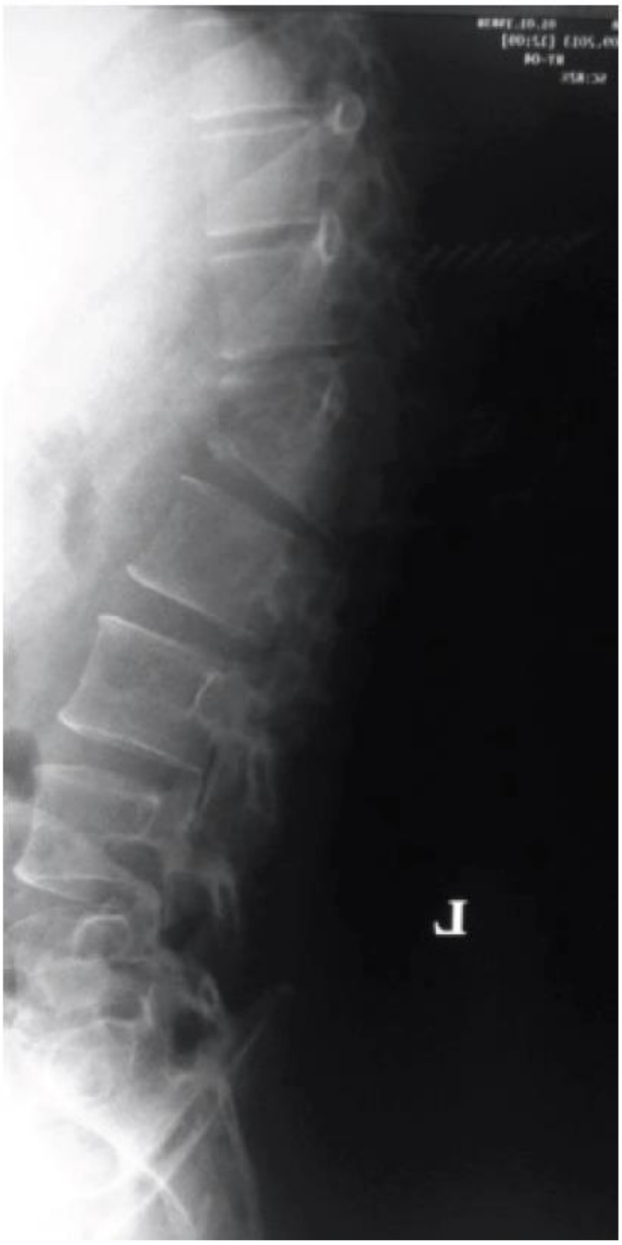
Question 211 of 277

A 34-year-old man presents with back pain and leg weakness. This has been getting worse for the past two months and he now complains that he is 'bent over'. Over the past three months he describes feeling generally unwell, with a poor appetite and night sweats.

His past medical history includes hypothyroidism. He emigrated from Bangladesh around 6 months ago.

On examination he has a kyphosis. He is most tender over the lower part of the thoracic spine. Neurological examination of his lower limbs demonstrates reduced power in both legs.

A lumbar spinal film is shown below:



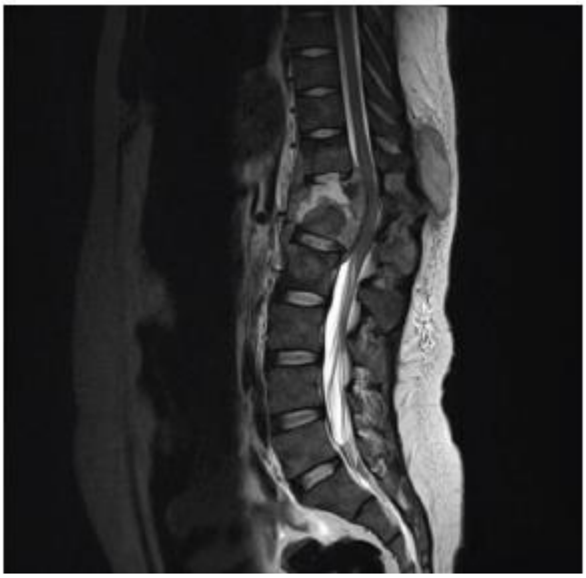
What is the most likely diagnosis?

A. Osteoporosis secondary to hypogonadism
B. Pott's disease
C. Actinomycosis
D. Sarcoma
E. Hodgkin's lymphoma

B. Pott's disease

The x-ray shows destruction of T12 and L1, with a resultant gibbus deformity (a short-segment structural thoracolumbar kyphosis resulting in sharp angulation). The destructive process is centred on the T12/L1 disc space.

A MRI from the same patient is shown below:

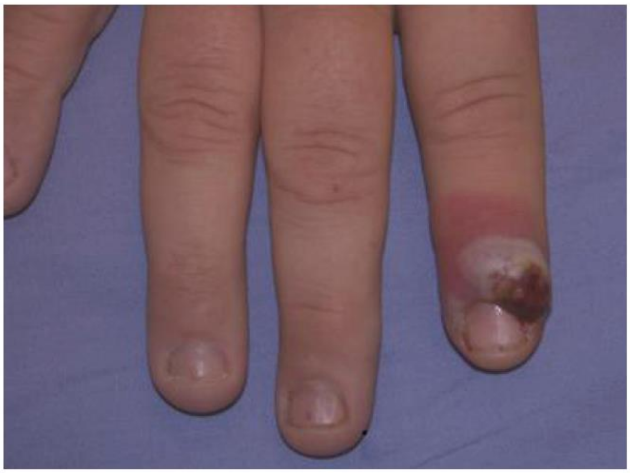


T12/L1 spondylodiscitis - with avid contrast enhancement involving the T11-L2 vertebral bodies, T12/L1 disc space and the adjacent paravertebral collections.

Hypogonadism is less likely given the systemic symptoms.

Question 212 of 277

A farmer presents with a tender lesion on his finger:



What is the most likely diagnosis?

A. Paronychia
B. Orf
C. Tetanus
D. Anthrax
E. Hand, foot and mouth disease

B. Orf

The correct answer is **Orf**. Orf, also known as contagious ecthyma, is a zoonotic disease caused by a parapoxvirus that primarily affects sheep and goats. Humans can contract the disease through direct contact with infected animals or via fomites. The typical presentation includes a painful, red nodule on the hand or finger, often with central ulceration. This aligns with the farmer's occupation and the clinical picture presented.

Paronychia is an infection of the skin around the nail, typically caused by staphylococci or streptococci bacteria. It presents with redness, swelling and tenderness at the side of the nail bed.

Although this could be plausible given the patient's profession, it does not account for all his symptoms nor does it match the image provided.

Tetanus, caused by *Clostridium tetani*, is characterised by muscle stiffness and spasms due to neurotoxins produced by this bacterium following wound contamination. While farmers are at an increased risk due to potential exposure to soil and animal faeces where *Clostridium tetani* spores reside, tetanus would not present as a single tender lesion on a finger.

Anthrax is another zoonotic disease caused by *Bacillus anthracis*. Cutaneous anthrax usually presents as a painless black eschar after an incubation period of 1-7 days post-exposure. However, in this case, there is no mention of exposure to livestock products such as hides or wool which would increase suspicion of anthrax.

Finally, **Hand, foot and mouth disease (HFMD)**, commonly caused by coxsackievirus A16 or enterovirus 71, primarily affects children under 10 years old. It presents with fever and vesicular lesions in the mouth and on hands/feet/buttocks. The age group affected and characteristic distribution of lesions make HFMD less likely in this scenario.

Question 213 of 277

A 26-year-old woman presents to the Emergency department some 2 weeks after returning from her honeymoon in the Seychelles. Since returning she has suffered from intermittent abdominal bloating and diarrhoea and feels she has lost a little weight. Physical examination reveals a blood pressure of 118/82 mmHg, and her pulse is 70 and regular. There is mild abdominal distension and her body mass index is 22 kg/m^2

Investigations:

Hb	110 g/l	Na +	138 mmol/l
Platelets	210 * 10 /l 9	K +	4.0 mmol/l
WBC	11.2 * 10 /l 9	Urea	6.9 mmol/l
Neuts	8.1 * 10 /l 9	Creatinine	89 µmol/l
Lymphs	1.5 * 10 /l 9	CRP	82 mg/l
Albumin	34 g/l		

Which of the following is the most appropriate intervention?

A. Amoxicillin
B. Ciprofloxacin
C. Gluten free diet
D. Lactose free diet
E. Metronidazole

E. Metronidazole

The most likely diagnosis, given the trip to a tropical region of Africa, intermittent bloating and diarrhoea, and an elevated CRP, is Giardiasis. Empirical treatment is often given, with either a course of metronidazole or a single dose of 2g tinidazole. Food intolerances may persist, particularly with respect to lactose, for some time after the initial infection.

Amoxicillin and ciprofloxacin would usually be used for listeriosis and campylobacter respectively, and neither infection is likely. Neither a wheat free diet nor a lactose-free diet is indicated given the likelihood of ongoing giardiasis, although many patients may suffer symptoms of lactose intolerance post-giardiasis.

Question 214 of 277

A 67-year-old woman is referred to the Emergency department by her GP because of an acute rise in serum creatinine. She has a history of Type 2 diabetes and takes lisinopril, amlodipine and indapamide as well as metformin and simvastatin, and has recently been prescribed trimethoprim for a UTI. Her blood pressure is 148/84 mmHg, with no postural drop on standing. There are no signs of fluid overload.

Na +	140 mmol/l
K +	4.9 mmol/l
Urea	5.8 mmol/l
Creatinine	162 µmol/l
Creatinine (one month earlier)	112 µmol/l

Which of the following is the most likely cause of her creatinine elevation?

A. Amlodipine
B. Lisinopril
C. Post-renal failure
D. Pre-renal failure
E. Trimethoprim

E. Trimethoprim

Trimethoprim is associated with a reversible elevation in serum creatinine because of a reduction in tubular excretion. Creatinine returns to normal once therapy ceases, and there is no effect on the GFR.

There is no suggestion of postural drop on blood pressure testing to support either pre-renal failure or amlodipine being the likely cause of creatinine elevation, and it's likely that the lisinopril has been prescribed for a prolonged period, rather than it being the cause of an acute rise in creatinine due to renal artery stenosis.

There are no symptoms of urinary obstruction to suggest post-renal failure.

Question 215 of 277

A 36-year-old gentleman was admitted to the Medical Admission Unit. His principle complaint was watery diarrhoea which has been present for the last few weeks. He had not noticed the presence of blood or mucus in the stool. He also complained of transient pain on swallowing, and weight loss of 2 stones over the last year. He denied the presence of respiratory symptoms and abdominal pain. He had no past medical history of note from his GP records. He consumes 20 cans of standard strength lager per week, smokes 20 cigarettes per day and denies recreational drug use. He had no fixed abode.

On examination, he was unkempt and dishevelled, with a BMI of 17.1 kg/m^2. Cardiovascular and respiratory examinations were unremarkable except for an oxygen saturation of 94% on room air. His heart rate was 92/min, blood pressure 112/62 mmHg and temperature 36.7ºC. Abdominal examination was also unremarkable though examination of the oral cavity revealed the presence of multiple aphthous ulcers. Examination of the neck revealed multiple small palpable cervical lymph nodes. Fundoscopy revealed the presence of white patches but otherwise, nil else and central and peripheral nervous system examination was otherwise normal. Skin examination revealed multiple pearly pink umbilicated nodules.

Initial investigations revealed the following:

Hb	122 g/l
Platelets	189 * 10 /l9
WBC	3.6 * 10 /l9

Chest x-ray: normal appearances of the heart and chest ECG: 92bpm normal sinus rhythm, no other abnormalities seen Urinalysis: normal

Stool MCS: normal interim results, pending further analysis

Which is the single investigation most likely to lead to the underlying diagnosis?

A. CT head, chest, abdomen and pelvis
B. Upper gastrointestinal endoscopy
C. Bone marrow biopsy
D. Colonoscopy
E. HIV serology

E. HIV serology

This gentleman has several conditions strongly suggestive of Acquired Immunodeficiency Syndrome (AIDS). He has features of cytomegalovirus (CMV) retinitis and oesophagitis caused by either CMV or other infections such as herpes simplex virus (HSV) and candidiasis. His loose stools are most likely representative of Cryptosporidium infection which would not be found on a conventional initial stool microscopy, culture and sensitivity test. He also has a skin infection in keeping with Molluscum contagiosum. Testing for HIV serology is, therefore, the best option.

Question 216 of 277

A 38-year-old male presented to the Emergency Department complaining of feeling unwell. He complained of a headache that had been present for the last two days which he described a continuous ache worse in the morning, as well as a sore throat. He later developed diarrhoea, opening his bowels three times per day passing watery loose stool. Since then he has deteriorated, complaining of nausea and vomiting on the day of admission. He also noted a new rash on his chest which developed over the last few hours and complained of pains in his muscles. He had returned two weeks ago from a six-month backpacking expedition around Africa and Far East Asia; he was prescribed doxycycline as antimalarial prophylaxis but had discontinued them two weeks into his trip after suffering from severe sunburns. He had not taken recommended vaccinations prior to his trip as he was engaged with work commitments. His past medical history comprised of asthma and depression for which he was prescribed sertraline 100mg OD and Clenil modulite 2 puffs BD.

Examination revealed the presence of an unwell gentleman. His temperature was 40.1ºC, heart rate 122 bpm, respiratory rate 22/min, oxygen saturations 95% on air and he had a blood pressure of 94/68 mmHg. He had a plethoric facial appearance, and multiple petechiae were noted on his chest and face. Examination of his cardiovascular system revealed the presence of normal heart sounds with a JVP of 2cm and a bounding radial pulse and vasodilated peripheries. Examination of his respiratory system revealed tachypnoea but with vesicular breath sounds. Examination of the gastrointestinal and neurological systems was unremarkable, with no neck stiffness, negative Kernig's and Brudzinski's signs. His GCS was 15. Multiple insect bites were noted on his legs and arms.

He was promptly transferred to the resuscitation area where two large bore cannulae were inserted and aggressive intravenous fluid resuscitation was commenced. A urinary catheter was inserted. The doctor inserting the cannulae noted that the sites of venepuncture continued to ooze despite the application of continual mechanical pressure.

Initial investigations revealed the following results:

Hb	102 g/l
Platelets	8 * 10 /l9
WBC	3.2 * 10 /l9
Neutrophils	2.2 * 10 /l9
Lymphocytes	0.8 * 10 /l9
Monocytes	0.1 * 10 /l9
Eosinophils	0.1 * 10 /l9
ESR	48 mm/hr

PTT	17 (NR 12-14s)
APTT	53 (NR 30-46s)
Fibrinogen	0.6 (NR 2-4g/l)
D-Dimer	428 (NR <230ng/ml)

Na +	130 mmol/l
K +	6.2 mmol/l
Urea	18.1 mmol/l
Creatinine	262 µmol/l
CRP	96 mg/l

Bilirubin	28 µmol/l
ALP	162 u/l
ALT	122 u/l
Total protein	52 g/l
Albumin	22 g/l

Blood MCS x3: awaiting result

Urine MCS: awaiting result

Thick film: negative

Thin film: awaiting result Portable chest x-ray: normal appearance of heart and lung fields

ECG: sinus tachycardia 122bpm

Urinalysis: trace protein, ketones ++, nil else

What is the most likely diagnosis?

A. Viral haemorrhagic fever
B. Malaria
C. Typhus
D. Thrombotic thrombocytopaenic purpura
E. Typhoid fever

A. Viral haemorrhagic fever

The Viral Haemorrhagic Fevers describe infection by a group of RNA viruses which include Yellow fever, Lassa fever and Ebola virus. Given the current outbreak in West Africa, this group of infections must be considered as a differential in returning travellers presenting with a fever. They are characterised by initially non-specific symptoms such as fever, headache, vomiting, sore throat, diarrhoea and myalgic muscle pains, progressing to shock, renal failure and the presence of disseminated intravascular coagulation (DIC). Investigations characteristically reveal an anaemia with thrombocytopaenia, low lymphocyte count, deranged liver function and the presence of DIC. This condition requires intensive supportive care and early diagnosis is imperative. There are of course many differentials, malaria being a key one here, but the absence of parasites on thick film make this a less likely diagnosis.

Question 217 of 277

A 17-year-old man is referred to the infectious disease department with a sore throat, fever and lymphadenopathy. He has been generally unwell for the previous 3 days but presented as he noticed some yellowing of his eyes. His routine blood test show

Bilirubin	55 µmol/l

ALP	54 u/l
ALT	402 u/l
AST	188 u/l
γGT	17 u/l
Albumin	43 g/l

He has no travel or sexual history and has never used intravenous drugs.

What is the most likely causative organism?

A. Viridans group Streptococci
B. Hepatitis B virus
C. Echinococcus granulosus
D. Hepatitis C virus
E. Epstein Barr virus (EBV)

E. Epstein Barr virus (EBV)

Epstein-Barr virus (EBV) is a herpes virus which commonly leads to infectious mononucleosis. This presents, often in teenagers and young adults with fevers, painful throat, lymphadenopathy and flu-like symptoms. Although it can last longer the symptoms are usually self-limiting within 2 weeks. Acute infection often leads to splenomegaly or hepatosplenomegaly and although a transient hepatitis is common it can lead to clinical jaundice.

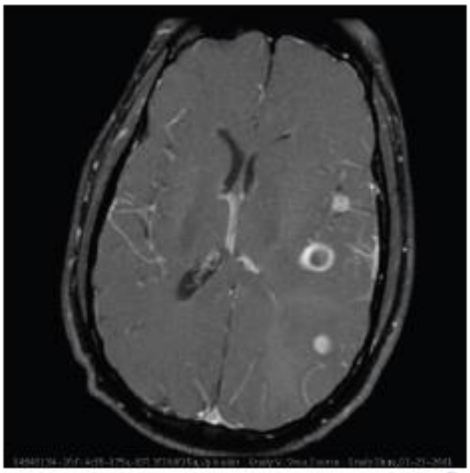
Question 218 of 277

A 50-year-old man with HIV presents to the emergency department with fits for 10 minutes. His vital signs are temperature 39 ºC, blood pressure 110/75 mmHg, and pulse 87 /minute. Examination reveals a Glasgow coma scale (GCS) score of 11 (E3V4M4). There is no focal neurological deficit.

Laboratory tests are requested as shown below:

Hb	135 g/L	(135-180)
Platelets	300 * 10 /L^9	(150 - 400)
WBC	4.5 * 10 /L^9	(4.0 - 11.0)
CD4 cell count	120 cells/mm^3	(500 - 1500)

An MRI brain is requested, as shown below:



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What is the most appropriate treatment for this patient?

A. Intravenous amphotericin-B
B. Methotrexate with whole brain irradiation
C. Oral pyrimethamine-sulfadiazine
D. Oral trimethoprim-sulfamethoxazole (TMP-SMX)
E. Surgical excision

D. Oral trimethoprim-sulfamethoxazole (TMP-SMX)

The most appropriate treatment option is **oral pyrimethamine-sulfadiazine** because the scenario describes a patient with HIV-related cerebral toxoplasmosis. MRI reveals intra-cerebral ringenhancing lesions with peri-lesional oedema. History of fits in an HIV-positive patient along with the MRI findings in this scenario confirms the diagnosis of cerebral toxoplasmosis. A 3-month treatment with pyrimethamine-sulfadiazine is the most appropriate management option.

Cerebral toxoplasmosis is not treated with **intravenous amphotericin B** . Amphotericin B treats cryptococcal meningoencephalitis, which is another important neuro-complication of HIV. The MRI scan in cryptococcal meningitis shows cryptococcomas, meningeal enhancement, abscesses, and gelatinous pseudocysts.

Methotrexate with whole brain irradiation is the most appropriate treatment for primary CNS lymphoma which is sometimes indistinguishable from cerebral toxoplasmosis in HIV patients. In this scenario, an important differentiating feature of primary CNS lymphoma and cerebral toxoplasmosis is the presence of a ring-enhancing lesion on MRI. Primary CNS lymphoma is characterised by a single mass lesion on an MRI scan that enhances homogeneously. When there is diagnostic uncertainty, a 3-month treatment with pyrimethamine-sulfadiazine is given.

Prophylaxis with **oral trimethoprim-sulfamethoxazole (TMP-SMX)** is given against cerebral toxoplasmosis in HIV patients when the CD4 count is below 100 cells /mm³. The patient in this scenario requires definitive treatment and not chemoprophylaxis with TMP-SMX because he has already developed cerebral toxoplasmosis, and has a CD4 count of above 100 /mm³.

Low-grade primary CNS lymphoma in HIV can be treated by **surgical excision** . Surgical excision has no role in treating cerebral toxoplasmosis, which is the case here. Primary CNS lymphoma is the most common HIV-associated brain tumour. The treatment of choice for primary CNS lymphoma is high-dose methotrexate with radiotherapy. Surgical excision can be considered for low-grade tumours.

Question 219 of 277

A 23-year-old man presents for the second time to HIV clinic. He was found to have positive HIV serology on opportunistic screening in a local GUM clinic and was referred to HIV services. He had originally presented with dysuria and had been diagnosed with non-specific urethritis and treated with doxycycline. Following his diagnosis, he has separated with his partner, as she feared contracting HIV from him. He is anxious with managing his disease and is keen to start treatment. He has no other symptoms, feeling systemically well and no significant past medical history.

During his previous consultation, his diagnosis was discussed with him, and the role of antiretroviral treatment was fully explained.

Blood tests:

Hb	134 g/l
Platelets	378 * 10 /l9
WBC	7.2 * 10 /l9
Na +	141 mmol/l
K +	4.1 mmol/l
Urea	4.6 mmol/l
Creatinine	64 µmol/l
HIV viral load	223 copies/ml
CD4 count	914 cells/mm3

What is the most appropriate management plan offer?

A. Start antiretroviral therapy and PCP prophylaxis

B. Start antiretroviral therapy
C. Start treatment when CD4 count becomes <350
D. Start treatment when CD4 count becomes <550
E. Start treatment once HIV related symptoms develop

B. Start antiretroviral therapy

This is a patient with newly diagnosed HIV and should be offered HIV treatment regardless of CD4 count or viral load. PCP prophylaxis is only necessary when the CD4 count becomes much lower.

Question 220 of 277

A young 27 year-old man who has been travelling on a jungle expedition across south east Asia presents to the local doctors with a 3 day history of fever, headache and a widespread maculopapular rash. He has been kayaking through rivers, trekking through long grass and jungle, and he reports being bitten by mosquitos, flies and mites. On close inspection he has a black necrotic eschar on his leg. A malaria rapid diagnostic test (RDT) is negative.

What is the most appropriate management?

A. Artemether / lumefantrine
B. Benzylpenicilin
C. Corticosteroids
D. Doxycycline
E. Supportive care

D. Doxycycline

The history describes a classical case of scrub typhus - and in particular the black eschar is the key diagnostic clue. Scrub typhus is caused by the tropical rickettsial bacteria *Orientia tsutsugamushi* and is transmitted by the bite of an infective trombiculoid 'chigger' mite. Its distribution is limited to South East Asia. Complications of disease include jaundice, meningoencephalitis, myocarditis, pneumonia and renal failure. However the disease can be easily treated with doxycyline.

Similar disease presentations are seen in other continents. *Rickettsia ricketsii* causes the potentially fatal Rocky Mountain spotted fever (RMSF), but its distribution is limited to North and Central America. African tick typhus is a mild and self limiting infection with no reported deaths and caused by *R. africae*. Other tropical rickettsial disease include endemic murine typhus and epidemic louse borne typhus which are more serious forms of disease.

The other answers are trying to catch you out as fever, headache and rash are non specific symptoms. Artemether / lumefantrine is the correct treatment for uncomplicated *Plasmodium falciparum*; Benzylpenicillin is the treatment for Leptospirosis; Corticosteroid would be used to treat Katayama fever (acute schistosomiasis); and supportive care would be appropriate in the setting of uncomplicated dengue fever (typically an urban disease).

Question 221 of 277

An 18-year-old male presents to the general medical take.

He presents with large grey lesions around his perineum and in his mouth which started 2 days ago. He reports that he has a more general rash on his trunk and the palms and soles of his feet. He generally feels unwell with malaise and fever. He has never had anything like this before but mentions that he did see a small ulcer on his scrotum a month ago that didn't hurt and healed on its own.

He reports that he is generally fit and well and has just moved to the area to start his university studies.

On examination, he has a symmetrical, widespread, macular papular rash on his trunk, limbs and the palms and soles of his feet. This is different from the large greyish lesions that you see in his mouth and perineum. He has general lymphadenopathy that is minimally tender on palpation. His chest is clear on auscultation, there are no added heart sounds and his abdomen is soft and nontender.

Investigations show:

Haemoglobin	13g/dl
WCC	11 x 10^9/l
Platelets	352 x 10^9/l
CRP	89mg/L
Sodium	145 mmol/l
Potassium	3.8 mmol/l
Urea	4 mmol/l
Creatinine	82 µmol/l

What is the most likely diagnosis?

A. Epstein Barr virus
B. HIV
C. Cytomegalovirus
D. Herpes simplex virus
E. Treponema pallidum

E. Treponema pallidum

This young man presents with a symmetrical rash on his trunk, palms, and soles that is pathognomonic for secondary syphilis. He also has a further rash of grey lesions in the mouth and perineum associated with syphilis. Although he was not concerned about it, he has also mentioned that he had a painless genital ulcer one month ago that may have represented his primary syphilis infection. He has general lymphadenopathy and epitrochlear nodes are particularly suggestive of the diagnosis.

He is a young man that has recently started university and is likely to be enjoying this period of parties and new acquaintances. It would be worth exploring his sexual history further as syphilis is more common in men who have sex with men.

Patients are often co-infected with other sexually transmitted diseases and this patient should be further investigated for these including HIV.

Answers 1,2 and 3 may all be associated with lymphadenopathy, but one would not expect to get genital ulceration or grey mucosal lesions. HIV can be associated with a rash on the limbs, palms and soles.

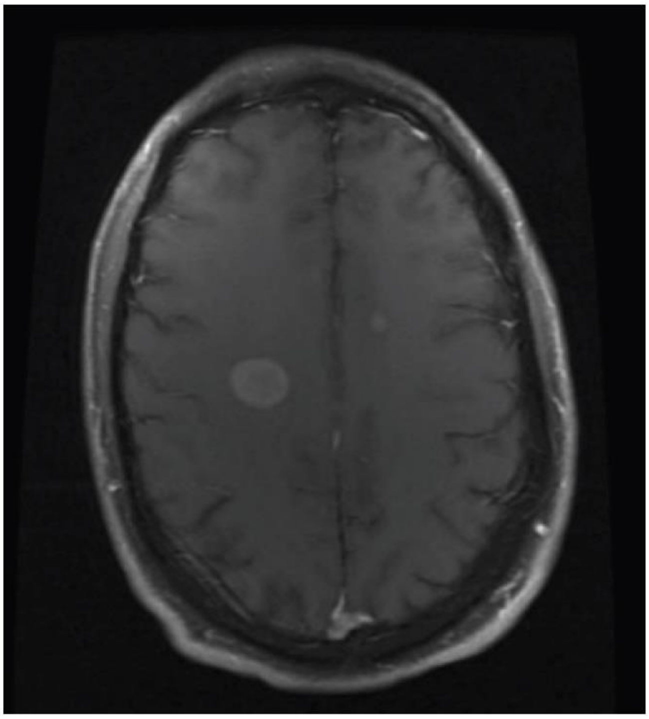
Answer 4 is associated with genital ulceration, but they are usually multiple, extremely painful and do not cause the systemic symptoms described in this question.

Question 222 of 277

A 45-year-old man presents to the emergency department with a 10-day history of progressively worsening headaches. Despite regular analgesia, the pain is not improving. He also complains of intermittent episodes of dizziness associated with nausea and vomiting. His past medical history includes human immunodeficiency virus for which he reports compliance with highly active antiretroviral therapy (HAART).

On examination, there is equal power and sensation in all 4 limbs. His cranial nerve and cerebellar examinations are unremarkable. Fundoscopic examination reveals bilateral papilloedema.

He is referred for neuroimaging, as shown below:



What is the most likely diagnosis?

A. CNS lymphoma
B. Cryptococcal meningitis
C. CMV encephalitis
D. Progressive multifocal leukoencephalopathy
E. Toxoplasmosis

A. CNS lymphoma

This patient's neuroimaging demonstrates 2 homogenous enhancing lesions; 1 larger lesion in the right hemisphere with 1 smaller lesion in the left hemisphere. Along with the history of worsening headaches associated with red-flag features (e.g. dizziness, vomiting), the presentation is in keeping with CNS lymphoma. CNS lymphoma accounts for up to 30% of cerebral lesions in patients with HIV and is associated with infection with the Ebstein-Barr virus. Like many cerebral lesions, presenting symptoms vary depending on the structures involved and include headache, nausea and vomiting, focal neurological signs and signs of raised intracranial pressure (e.g. papilloedema), as seen here.

Cryptococcus neoformans causes an opportunistic fungal infection that causes headache, fevers, confusion and signs of raised intracranial pressure. It is the most common fungal infection in patients with HIV. Imaging studies show leptomeningeal enhancement, cryptococcomas, hydrocephalus or dilated perivascular spaces. It does not account for lesions seen in this patient's MRI.

Cytomegalovirus (CMV) is a viral infection that commonly causes illness in patients with HIV, particularly in those with a CD4 count < 50 cells/mL. The most common form of CMV is CMV retinitis. However, CMV encephalitis is also common. On imaging, cerebral oedema may be seen. Meningoencephalitis and ventriculitis are also features of infection with CMV. These are not seen here, making CMV encephalitis unlikely.

Infection with JC virus is responsible for a condition known as progressive multifocal leukoencephalopathy, a demyelinating disease of the CNS. Symptoms include behavioural changes and alterations in speech, vision and movement. On imaging, single or multiple lesions may be seen, with demyelinating white matter lesions seen on MRI studies.

Toxoplasmosis is an opportunistic infection caused by the parasite *Toxoplasma gondii*. It is the most common cause of neurological brain abscesses in patients with HIV with symptoms including headache, fever and neurological changes including seizures, hemiplegia, cerebellar signs, or an acute confusional state. Domestic cats are a common source of transmission. Unlike CNS lymphoma, toxoplasmosis presents as ring-enhancing lesions on neuroimaging. These are not seen on this patient's scan, making it an unlikely diagnosis.

Question 223 of 277

A 45-year-old man presents to the Emergency Department. He told the admitting doctor that he had fallen onto his left hip last night while he had been drinking at home. He had extensive bruising around his hip and some bony tenderness which resulted in a hip x-ray being taken.

His x-ray shows no fracture nor bony abnormality, but several 2x4mm specs of calcification are visible in his psoas and thigh muscles.

Later on, his wife asks to speak to you in confidence. She states that over the last year her husband's personality has gradually changed and he is sometimes forgetful. He lost his job 3 months ago as an engineer. He had worked for 8 years in Peru on an engineering project in his thirties. He drinks 8 units of alcohol per day and seems to have had a low mood for several years.

Yesterday he had fallen, lost consciousness, and had a jerking of his arms and legs lasting 1 minute. He had hit his hip on a coffee table and had wet himself. Afterwards, he felt tired and lethargic but had refused to go to hospital.

What is the most likely diagnosis?

A. HIV dementia
B. Alcohol withdrawal seizure
C. Neurocysticercosis
D. Emboli secondary to calcified aortic value
E. Primary idiopathic epilepsy

C. Neurocysticercosis

This is neurocysticercosis. It is the most common cause of epilepsy worldwide. Eggs of the Taenia Solium tapeworm are ingested, oncospheres hatch and migrate to peripheral sites (classically muscles and brain) and become cysticerci. They may remain asymptomatic or cause inflammatory pathology when the cysticerci die. They eventually become calcified.

Diagnosis is made with MRI or CT imaging. Calcified cysts in skeletal muscle may be found incidentally on x-ray. Serology may be helpful.

Management is with praziquantel or albendazole and prednisolone.

Question 224 of 277

A 37-year-old man is referred to the medical outpatient clinic. He was born in Brazil but has been in the UK since university and now works in marketing.

He has been complaining of increasing breathlessness, worse on exertion, and is now breathless on climbing a flight of stairs. He has had several faints in the last few months, associated with feeling a 'funny' sensation in his chest, following which he blacks out. On one occasion he sustained a cut on his eye brow. There are no triggers to these episodes and there is a rapid recovery. He has never had any episodes of incontinence or tongue biting.

His past medical history is unremarkable. He was treated with several courses of antibiotics as a child in Brazil for an eye infection that persisted for several months. He has been seeing his GP for the past year for increasingly troublesome constipation. His medications are:

Senna 15mg at night

Movicol two sachets tds

On examination he has a displaced apex beat, normal heart sounds, and mild coarse crepitations at both lung bases.

What is the likely diagnosis?

A. Chagas disease
B. Endomyocardial fibrosis
C. Churg Strauss Syndrome
D. Hypertrophic Obstructive Cardiomyopathy
E. Vasovagal syncope

A. Chagas disease

This is Chagas disease caused by *Trypanosoma cruzi*. Keys is the question are presence of Romana's sign (periorbital swelling following inoculation of Trypanosome) as a child, and heart failure and constipation in later life.

Question 225 of 277

As the doctor on call you are called to review a 28-year-old male on the haematology ward. He recently underwent an allogenic stem cell transplant, which so far had been reasonably uncomplicated. Earlier in the week the patient had complained of coryzal symptoms and a dry cough. A nasopharyngeal aspirate (NPA) had been sent along with other routine tests 24 hours ago.

When you arrive to review the patient on the ward you note that he appears extremely sweaty. You take his temperature using a tympanic thermometer which reads 39.5ºC. His heart rate is 95 beats per minute and his respiratory rate is 16 breaths per minute. Blood pressure is 122/76 mmHg. Physical examination is unremarkable and chest X-ray reveals clear lung fields.

You review the patients most recent results:

Hb	95 g/l	Na +	134 mmol/l
Platelets	50 * 10 /l 9	K +	3.6 mmol/l
WBC	0.1 * 10 /l 9	Urea	3.0 mmol/l
Neuts	0.1 * 10 /l 9	Creatinine	65 µmol/l
Lymphs	0.0 * 10 /l 9	CRP	12 mg/l
Eosin	0.0 * 10 /l9		
NPA	Influenza A		

What is the most appropriate treatment to commence?

A. Amoxicillin
B. Oseltamivir
C. No treatment required
D. Flucloxacillin
E. Zanamivir

E. Zanamivir

In this case they key points to realise are that the patient is immunosuppressed (neutropaenic post transplant) with symptoms and PCR confirmed Influenza A infection.

Whilst in most cases, oseltamivir (a neuraminidase inhibitor) is first line treatment for influenza, the BNF advises that for severely immunocompromised individuals zanamivir should be first line treatment. This can help reduce the length of symptoms and prevent complications from developing during the course of the infection.

Question 226 of 277

A 44-year-old man presents with a 1 day history of fever. He returned to the UK 12 days ago after visiting his family in northern Uganda. He has been resident in the UK for the last 20 years and returns home on average once a year. He does not routinely take malaria prophylaxis.

On examination his temperature is 39.4ºC, pulse is 86 beats per minute, his blood pressure is 115/78 mmHg and his oxygen saturations are 98% on air. The remainder of the physical examination is normal. A thick and thin blood film are sent to the lab which confirms *Plasmodium falciparum* malaria. The report reads 2.1% parasitaemia with the presence of schizonts.

What is the most appropriate initial management strategy?

A. Intravenous artesunate
B. Oral quinine sulfate
C. Oral chloroquine
D. Oral atovaquone-proguanil (Malarone)
E. Intravenous quinine

A. Intravenous artesunate

This question examines your ability to differentiate severe malaria from uncomplicated malaria and then apply the correct therapy.

We are told the patient has been living in the UK for the last 20 years. As a result any natural immunity built up in earlier life would have dramatically reduced putting him at high risk of developing severe malaria with a relatively low level parasitaemia.

The most important information given is that schizonts are present in the blood film. Schizonts are the final step before red blood cells rupture and release more parasites (merozoites) into the blood.

Given his lack of immunity and an impending rise in parasitaemia it would be pertinent to consider this a case of severe malaria and treat accordingly with intravenous therapy early on. This excludes answers B, C and D. Furthermore chloroquine is only used routinely in the treatment of non falciparum malaria.

WHO guidelines recommend the use of intravenous artesunate for at least the first 24 hours until the patient can tolerate oral medication.

Question 227 of 277

A 30-year-old male refugee presents with fever, rigours and right flank pain. With the help of a translator you discover he has been unwell for 7 days with these symptoms that are getting progressively worse. He says he is otherwise fit and well and does not take any regular medications. He moved to the UK 2 months ago having lived his entire life in Sudan.

On examination he has a temperature of 38.2 degrees, a heart rate of 98 beats per minute, a blood pressure of 110/70 and a respiratory rate of 20. He is lying calmly in the bed but is tender over the renal angle on the right side.

Investigations:

serum sodium	138 mmol/L (137-144)
serum potassium	5.5 mmol/L (3.5-4.9)
serum creatinine	240 mol/L (60-110)
haemoglobin	98 g/L (130-180)
white cell count	15.4 Ã— 109/L (4.0-11.0)
eosinophil count	0.89 Ã— 109/L (0.04-0.40)

platelet count	378 Ã— 109/L (150-400)
urine microscopy	red cells 2+, white cells 3+, protein 2+
ultrasound scan of abdomen	right sided hydronephrosis and hydroureter, fibrotic and calcified bladder

What is the most likely underlying diagnosis?

A. Schistosomiasis japonicum
B. Chronic vesico-ureteric reflux and secondary pyelonephritis
C. Squamous cell carcinoma of the bladder
D. Schistosomiasis haematobium
E. Schistosomiasis mansoni

D. Schistosomiasis haematobium

The clinical picture here is of an acute pyelonephritis with fever, rigours and tenderness over the renal angle. However blood analysis and imaging suggest there is another entity at play that has predisposed the patient to this.

S. mansoni and *S japonicum*, *S. mekongi* and *S. intercalatum* produce eggs that can invade the

bowel wall causing an intense inflammatory reaction that gives rise to loose bloody stools. Eggs

can also migrate to liver through the portal venous system where they can elicit a granulomatous

fibrosing reaction. This can eventually lead to the blockade of venous blood and flow. Portal

venous hypertension results which can give rise to varicies and upper gastrointestinal bleeding.

S. haematobium on the other hand leads to granulomatous inflammation, ulceration of the vesicle

and ureteral walls. Subsequent fibrosis can cause bladder neck obstruction, hydroureter and

hydronephrosis. These changes can cause a chronic renal impairment and predispose to secondary

bacterial infection as well as squamous cell carcinoma.

In addition, all schistosome species can result in immune complex deposition in the kidneys leading to a proteinuria and nephrotic syndrome.

Question 228 of 277

A 42-year-old male from Bolivia presents with a 8 month history of progressive fatigue, dyspnoea and intermittent chest pains. He is a chronic smoker and during a recent severe episode of breathlessness had consulted his GP who prescribed amoxicillin and prednisolone to combine with his regular inhaler therapy. Shortly after this he experienced a febrile episode which had lasted 7 days before resolving. His breathlessness also worsened during this period.

On examination he has a temperature of 37.2 degrees, a heart rate of 98 beats per minute, a blood pressure of 110/70 mmHg and a respiratory rate of 24/min. His jugular venous pressure was raised, there was lower limb oedema to the mid shin and a pan systolic murmur heard best in inspiration in the left parasternal region. An ECG showed a prolonged PR interval.

What is the most likely underlying diagnosis?

A. Ischaemic heart disease
B. Chronic pulmonary emboli
C. Chronic Chagas cardiomyopathy
D. Sarcoidosis
E. HIV

C. Chronic Chagas cardiomyopathy

Teasing out the infective aetiology in this question is the key to finding the answer. Chagas disease can present as an acute illness or as a chronic disease years after infection and is caused by the parasite *Trypanosoma cruzi*.

Acute disease is usually asymptomatic but may manifest as a febrile episode in association with a swelling over the site of inoculation (if in the skin this is called a chagoma if through the conjunctiva of the eye this is known as Romanas sign). In severe cases an acute myocarditis can evolve with a secondary pericardial effusion.

Chronic disease begins to develop weeks or months after the initial inoculation and in some cases does not present until years after the infection (as in this case). Chronic disease can effect the heart causing a biventricular cardiomyopthy with predominant right sided features as well as varying degrees of heart block and conductive abnormalities due to fibrous inflammation around amastigotes embedded in the heart muscle. Chest pain can also be common and does not imply an ischaemic component. Chronic disease can also effect the bowel causing dilation.

The first clue in the case is that the patient is from an area where disease is endemic. Secondarily there is an absence of ischaemic related risk factors other than his smoking history and he is of a relatively young age. Perhaps the biggest clue of an infective cause is the febrile episode following steroids therapy. This likely represents a relapse of acute infection which is common with immunosupression. At this point you may be able to see trypomastigotes in the blood but they soon disappear on resolution of the fever.

Chronic PE is also a possibility given the predominance of right sided signs but does not explain the first degree heart block or fever following steroid therapy.

Question 229 of 277

A 50-year-old man presents to the Emergency Department after becoming increasingly unwell over the previous two weeks. His initial symptoms were fevers increasing shortness of breath. In the last 24 hours he had experienced several episodes of haemoptysis and bloody diarrhoea precipitating his attendance to hospital.

Past medical history included a diagnosis of systemic lupus erythematous five years previously. This disease had been poorly controlled in recent months requiring the patient to take prednisolone 40 mg daily and hydroxychloroquine 400 mg daily. The patient was originally from Mali and had emigrated to the UK twenty years previously. He had returned to visit family in Africa earlier in the year and had not taken malaria prophylaxis whilst abroad.

Initial assessment revealed the patient to be very unwell. Respiratory rate was 32 with O2 saturations of 94 % on high flow oxygen. Blood pressure was 80/50 mmHg with no improvement in hypotension following aggressive fluid resuscitation. The patient was intubated and ventilated and transferred to the Intensive Care Unit for inotropic support.

Results of immediate investigations are listed below.

Chest x-ray: widespread diffuse pulmonary infiltrates; left sided pleural effusion; no free air under the diaphragm

Abdominal x-ray: multiple loops of distended small bowel; no evidence of free air

Hb	11.2 g/dl
Mean cell volume	83 fL
Platelets	150 * 10 /l9
WBC	18.7 * 10 /l9
Neutrophils	14.8 * 10 /l9

Lymphocytes	2.0 * 10 /l9
Monocytes	1.0 * 10 /l9
Eosinophils	0.9 * 10 /l9
Blood film	no abnormality detected
Internationalised normalised ratio	1.2

Na +	124 mmol/l
K +	4.9 mmol/l
Urea	15 mmol/l
Creatinine	190 µmol/l

C-reactive protein	284 mg/dL

Blood cultures: Gram negative cocci (positive after 24 hours)

Pleural fluid microscopy: filariform larvae

What is the most likely underlying diagnosis?

A. Caecal carcinoma
B. Strongyloides hyperinfection syndrome
C. Churg-Strauss vasculitis
D. Plasmodium falciparum infection
E. Disseminated haematogenous tuberculosis

B. Strongyloides hyperinfection syndrome

Strongyloides hyperinfection syndrome occurs in immunosuppressed individuals with coexistent Strongyloides stercoralis infection. Uncontrolled proliferation of larvae occurs with dissemination to end organs.

As in this individual, systemic sepsis often occurs due to involvement of the gut wall allowing translocation of gut bacteria. Other features include pulmonary infiltrates, paralytic ileus, gastrointestinal bleeding and syndrome of inappropriate ADH secretion.

Filariform larvae can be found in body fluids by microscopy and it is this that confirms the diagnosis in this case. Due to immunosuppression, eosinophilia is often absent. Given the patient's country of origin, strongyloidosis is most likely to have been chronic, but could have been acquired on his recent trip to Africa.

Plasmodium falciparum infection can cause a similar presentation, although the normal blood film in this case makes this unlikely. Severe tuberculosis infection tends to predominantly involve the lungs with a varying degree of involvement of other organ systems.

Churg-Strauss vasculitis can cause severe abdominal symptoms including infarction and perforation. However, the normal eosinophil count, short history and immunosuppresion do not support this diagnosis.

Colonic carcinoma can lead to translocation of gut bacteria and gram-negative sepsis. However, this diagnosis would not explain all of the findings in this case and is not supported by the normal mean cell volume.

Question 230 of 277

A 23-year-old woman is admitted to the hospital with acute-onset fever, neck stiffness, headache and photophobia. She has no significant past medical history. A CT scan of the head is normal. The patient subsequently undergoes a lumbar puncture which confirms a diagnosis of bacterial meningitis.

She lives with a group of flatmates, including her partner, with whom she has had close contact up until 2 days ago. The patient visited her parents' house and had close contact with them 9 days ago; she visited her grandparents' house 16 days ago and had close contact with them.

What is the most appropriate next step?

A. No prophylaxis required for contacts
B. Prophylaxis required for all flatmates
C. Prophylaxis required for all flatmates and parents
D. Prophylaxis required for flatmates, parents and grandparents
E. Prophylaxis required for partner only

B. Prophylaxis required for all flatmates

People who have been exposed to a patient with confirmed bacterial meningitis should be given prophylactic antibiotics if they have close contact within the seven days before onset

Guidance around meningitis prophylaxis is quite clear. Prophylaxis should be offered to household members and those in close contact within the past 7 days. As such, it should be **given to all flatmates** as they have been in close contact up until 2 days prior. Oral ciprofloxacin or rifampicin may be used. Ciprofloxacin is now the drug of choice.

Advising **no prophylaxis** is therefore incorrect.

Similarly, advising **both flatmates and parents** to have prophylaxis is incorrect. Whilst the flatmates should be offered prophylaxis, the parents last had contact over 7 days ago.

Prophylaxis for **flatmates, parents and grandparents** is unwarranted. The parents and grandparents last had contact with the patient more than 7 days ago and so it is not needed for them.

Only advising the partner to have prophylactic antibiotics is incorrect. The entire household has had close contact with her and so all flatmates, not just her partner, should be offered them.

Question 231 of 277

A 35-year-old ex intravenous drug user has been diagnosed with has HBe-Ag positive Hepatitis B. Her investigation results are shown below:

HBV DNA	2100 IU/ml
ALT	60 IU/L

ALT last checked 3 months ago and found to be ALT 60 IU/L.

What is the recommended first line treatment for this patient?

A. Tenofovir disoproxil
B. Entecavir
C. Peginterferon alfa-2a
D. Telbivudine
E. Sofosbuvir

C. Peginterferon alfa-2a

The correct answer here is Peginterferon alfa-2a.

In patients who do not undergo HBeAg seroconversion or who relapse, tenofovir disoproxil is second line. Entecavir is an alternative second line when patients are unable to tolerate tenofovir disoproxil. Telbivudine is no longer recommended for the treatment of chronic Hepatitis B. Sofosbuvir is used for treating hepatitis C.

Question 232 of 277

A 32-year-old lady presented after returning from Nigeria 3 weeks ago to her GP complaining of feeling feverish and pain in her joints making walking difficult. She denied any rash, or headaches however she has been very drowsy and lethargic in the past week. She spent a week in Nigeria and visited her relatives both in urban and rural areas. She has been taking her malaria prophylaxis tablets. She denies being sexually active for the past year. Her GP sent off three malaria screens which were negative.

Two months later she represents to the emergency department following a seizure and is found to be confused and disorientated. She is irritable, and you notice her hand is trembling. Her sister says she has lost her appetite and weight and has been behaving strangely in the past month. She spends most of her time in bed during the day complaining of a headache and has become withdrawn. On examination, there is hypertonia in her limbs, with hyperreflexia, but the rest of

examination is difficult as she is uncooperative. Investigations:

Na+	140mmol/l
K+	4.3 mmol/l
Urea	5.3 mmol/l
Creatinine	85 µmol/l
Serum bilirubin	15 µmol/l
Serum alkaline phosphatase	85 IU/l
Serum aspartate aminotransferase	19 IU/l
Serum Albumin	25 g/l
C Reactive protein (CRP)	25 mg/l
Erythrocyte Sedimentation Rate (ESR)	75 mm/hr
Haemoglobin	96 g/l
White cell count	10.2 x 10^9/L
Neutrophils	8.9 x 10^9/L
Eosinophilia	0.1 x10^9/L
Lymphocytes	1.8 x 10^9/L
Platelets	140 x 10^9/L
INR	1.0

What is the likely causative organism?

A. Plasmodium vivax
B. Borrelia burgdorferi
C. Trypanosoma gambiense
D. Trypanosoma rhodesiense
E. Trypanosoma cruzi

C. Trypanosoma gambiense

This lady of African background presents initially with fever after travelling from Nigeria and goes on to develop chronic symptoms of daytime somnolence, continuous headaches and behavioural changes. These symptoms suggest African Sleeping Sickness or African Trypanosomiasis. *Trypanosoma gambiense* is more common than *Trypanosoma rhodesiense* in patients from West Africa, such as Nigeria.

Early stages of the disease tends to present about 3 weeks after being bitten by an infected tsetse fly. Symptoms include fevers, arthralgia, lethargy, drowsiness. Late stages include behavioural and mood changes, headache and the pathognomonic daytime somnolence with nocturnal insomnia.

Borrelia burgdorferi is transmitted by ticks (Ixodes spp.) and causes Lyme disease. They occur in the USA, northern and central European countries, and temperate forested areas of Asia.

Plasmodium vivax is one species of Plasmodium, the parasite that causes malaria. It is spread by infected mosquitoes. It is an important differential of fever in the returning traveller, however as the initial malaria screens were negative, and the central nervous system symptoms in the late stages are not consistent with a malarial infection.

Trypanosoma cruzi is responsible for American Trypanosomiasis (or Chagas' disease). It is transmitted by contamination with infected faeces of assassin bugs (reduviid bugs). It is found in South America and the south USA. Chronic infection can lead to chronic congestive heart failure, megaoesophagus and megacolon.

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A 42-year-old immigrant from Nigeria has been referred to the infectious disease clinic. He was feeling tired and generally unwell over several weeks since last visiting Nigeria, his country of origin. He has also travelled for business to the United States six months ago. He has had fever and rigors as well as headaches. Posterior lymph nodes have been present and have been aspirated one week ago, and trypomastigotes were present.

What is the most likely causative organism?

A. Trypanosoma rhodesiense
B. Trypanosoma gambiense
C. Trypanosoma cruzi
D. Leishmania mexicana
E. Leishmania tropica

B. Trypanosoma gambiense

The correct answer is Trypanosoma gambiense. This is a patient with a clinical history suggestive of African trypanosomiasis with a slow wasting course. The presence of trypomastigotes means that there will be a Trypanosoma infection. The species can be determined by geography. Rhodesiense occurs in east Africa whilst gambiense occurs in west Africa, including Nigeria. Uganda is the only country with both. Cruzi is endemic in South America and causes American trypanosomiasis. Leishmania are other protozoa occurring in Africa, South America and India.

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A 23-year-old gentleman presents to GUM clinic. He has been referred by his GP as he presented with dysuria and a negative urine dipstick test. A sexual health screen, including blood tests for HIV and syphilis serology, are requested.

What form of consent is required?

A. No consent
B. Implied consent
C. Verbal consent
D. Written consent
E. Written consent for HIV testing and verbal consent for sexual health screening

C. Verbal consent

The correct answer is verbal consent. Consent clearly is needed as an investigation is done but since syphilis and HIV are transmittable diseases consent needs to be specific towards each. This means that the patient must be aware that they are being tested for HIV and syphilis rather than just aware that they are having a routine blood test. Written consent is not needed, and may discourage patients from having testing.

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A 30-year-old man returns from a stag do in Amsterdam with fever, headache and abdominal pain. He was away for 2 days and drunk 40-50 units of alcohol. He also had unprotected sex with a local girl and vaguely remembers falling in the canal on the second night. He is febrile with a blood pressure 100/70 mmHg. His bloods tests reveal :

Routine tests in the emergency department are ordered: Hb 95 g/l WBC 20 10 /l , Platelets 150 10 /l Na 135 mmol/l , K 5.0 mmol/l

Urea 15 mmol/l , Creatinine 300 µmol/l Bilirubin 35 µmol/l , ALP 200 u/l , ALT 70 u/l CXR - pulmonary haemorrhage

What is the most likely diagnosis?

A. Acute hepatitis A
B. Acute hepatitis B
C. Goodpastures syndrome
D. Leptospirosis
E. Atypical pneumonia

D. Leptospirosis

This gentleman should be screened for viral hepatitis and have an HIV test, however given the results of his investigations infection with Leptospira from falling in the contaminated water is the most likely diagnosis. The acuity and neutrophilia go against Goodpasture's syndrome and an atypical pneumonia would not classically cause pulmonary haemorrhage.

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A 68-year-old female was admitted to the ward with left arm cellulitis. She was septic on admission and the sepsis 6 bundle was completed in one hour. She is treated with IV flucloxacillin initially and was switched to oral flucloxacillin on day 2. On day 3 of admission you receive a call from the duty microbiologist with the following results:

Anaerobic blood culture bottle	Positive for *Staphylococcus aureus*
Aerobic blood culture bottle	Positive for *Staphylococcus aureus*

How will you manage this patient?

A. IV vancomycin
B. Oral linezolid
C. IV flucloxacillin for two weeks
D. Oral flucloxacillin for two weeks
E. Oral flucloxacillin for one week

C. IV flucloxacillin for two weeks

Staphylococcus aureus bacteraemia (SAB) is a serious condition which may occur secondary to soft tissue, joint, bone, indwelling IV line or cardiac infection. It is treated with a minimum of two weeks IV flucloxacillin

Staphylococcus aureus bacteraemia (SAB) is a serious condition with significant morbidity and mortality. SAB is generally caused by soft tissue, joint, bone, indwelling IV line or cardiac infections. Once the bacteria has entered the bloodstream, they are particularly effective at infecting various organs, potentially causing infective endocarditis, septic arthritis, discitis, and osteomyelitis.

Treatment is with intravenous high dose (2 grams) flucloxacillin for a minimum of two weeks. An MRSA risk assessment should be performed. If the patient is at high risk of MRSA then they should be treated with vancomycin instead.

It is important to look for sources of infection and to consider investigating for sequelae (e.g. transthoracic echo to assess for endocarditis).

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A 34-year-old female attended the emergency department with a rash. Her past medical history was unremarkable apart from being on the oral contraceptive pill. She had been recovering from a widespread, pruritic, vesicular rash over the past 8 days after coming into contact with her nephew with chickenpox. Yesterday she began to feel lethargic and unwell, with a high temperature and a painful left calf. This morning she had noticed an area of erythema on her lower leg which seemed to get larger and more painful throughout the day.

On examination, she looked unwell. There were healed varicella scars and a few crusted scabs present over her lower limbs and torso. There was a diffuse erythematous rash extending from her mid-calf to her ankle. The area was oedematous and very tender. She had a normal range of movement in all joints of the leg, albeit limited by pain.

Her observations were as follows:

- temperature 39.7ºC
- respiratory rate 24/min
- blood pressure 91/55mmHg
- heart rate 112bpm
- oxygen saturations 98% on room air

Her blood results were as follows:

Hb	109 g/L	115 - 160
WBC	20 * 10 /L 9	(4.0 - 11.0)
CRP	208 mg/L	(< 5)

She was started on intravenous fluids and given analgesia. Her IV morphine was titrated up to control her pain and she was started on broad-spectrum antibiotics.

The following morning she remained tachypnoeic and tachycardic. The area of erythema now extended proximally to 2cm below her knee. A small area of dark purple-black discolouration was present at the distal aspect of the rash.

What is the next most appropriate step in management?

A. IV aciclovir
B. Plasmapheresis
C. MRI lower leg
D. Surgical debridement
E. Treatment dose low molecular weight heparin

D. Surgical debridement

Chickenpox is a risk factor for invasive group A streptococcal soft tissue infections including necrotizing fasciitis

This is a case of bacterial superinfection complicating acute varicella infection. The systemic involvement and rapid spread of cellulitis despite broad-spectrum antibiotics should alert you to the possible diagnosis of necrotising fasciitis. Whilst the most common type of necrotising fasciitis is type 1 (caused by polymicrobial infection), most cases of post-varicella necrotising fasciitis are caused by a monomicrobial infection by group A streptococcus (e.g. *Streptococcus pyogenes*).

Bacteria are introduced into the skin via skin trauma (in this case, the varicella skin lesions formed a portal of entry) and track along fascial planes, extending beyond the area of overlying cellulitis.

The definite management of necrotising fasciitis is surgical debridement of all infected tissue. As the infection progresses, necrosis and hypoperfusion of the area limit the delivery of antibiotics to the area. Without debridement, mortality is 100% and any delay increases the risk of death.

Whilst IV aciclovir is important in the treatment of acute varicella infection, the most important next step is to remove the source of the bacterial infection. Chickenpox has contributed to this presentation but is not the cause of her acute deterioration or necrotic skin.

Whilst MRI can confirm the extent of necrotising fasciitis, it may delay surgical debridement. This patient is clearly unwell and needs immediate definitive management.

Treatment dose low molecular weight heparin would be appropriate if the cause was a deep vein thrombosis but this presentation is more in keeping with a rapidly progressive bacterial infection.

Plasmapheresis does not have a role in the management of necrotising fasciitis.

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A 27-year-old male presents with high-grade fever and vomiting for 8 days. He recently went to Africa for a jungle safari with a group of friends and began feeling unwell whilst he was there. During the trip, he spent most of the time camping outdoors. He does not have any history of fits or loss of consciousness, although he has been feeling drowsy and complains of generalised malaise. He also has pain in his wrist and shoulder joints but they are not swollen. He takes alcohol regularly and smokes cannabis socially.

On examination, he has a fever of 39°C and a pulse of 135bpm. His blood pressure is 100/70mmHg. He is icteric but does not have any flapping tremors. There is evidence of an enlarged spleen which is palpable 3 finger breadths below the left costal margin. The liver span is normal.

Lab reports reveal:

Hb	115 g/l
Platelets	100 * 10 /l9
WBC	9.5 * 10 /l9
Reticulocytes	5% (0.2 - 2%)

Na +	140 mmol/l
K +	4.6 mmol/l
Urea	5.1 mmol/l
Creatinine	83 µmol/l

Bilirubin	49 µmol/l
AST	50 u/l
ALT	25 u/l
Glucose	6.0 mmol/l

CT scan brain: Normal CSF examination reveals:

Appearance	Clear
Protein	0.3 g/L (0.2 0.4 g/L)
Glucose	5.3 mmol/l
Lymphocytes	$15/mm^3$

Neutrophils	10

Which of the following is the most appropriate treatment option?

A. Chloroquine
B. Artemether/lumefantrine
C. Primaquine
D. IV acyclovir
E. Co-trimoxazole

B. Artemether/lumefantrine

The high-grade fever, history of travel to an endemic area, splenomegaly, thrombocytopaenia, evidence of haemolysis (raised bilirubin and reticulocytosis) and normal blood leucocyte count all favour a diagnosis of cerebral malaria

The treatment modality most suited would be a combination of artemether and lumefantrine which would provide adequate coverage from plasmodium falciparum.

Diagnosis is through examination of blood films (thick and thin films) for evidence of parasitaemia and the ICT-MP.

The CSF findings are non-specific with the only abnormality being a mild lymphocytosis which is of no bearing on the scenario. It, however, rules out bacterial or tuberculous meningitis.

The mildly raised AST is in keeping with his history of alcohol intake.

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A 38-year-old woman presents to her HIV clinic for her 6-monthly review having had her routine blood tests two weeks prior to her clinic visit. She has diagnosed with HIV seven years ago and has been stable on antiretroviral therapy since then. Her other medical problems include asthma which is well controlled. She reports generally good compliance with her medications but has unfortunately missed two of her treatment doses as she was on a weekend holiday and forgot to take her tablets with her. She feels well in herself.

Blood tests:

Today	Two weeks ago	One year ago	
HIV viral load	110 copies/ml	<50 copies/ml	<50 copies/ml
CD4 count	983 cells/mm^3	912 cells/mm^3	Not tested

How should she be further investigated?

A. Urgent CD4 count
B. Urgent viral resistance testing
C. Urgent HIV-1 and HIV-2 serology
D. Repeat viral load in one month
E. Routine viral load and CD4 count in six months

D. Repeat viral load in one month

The question describes a possible viral blip, a single viral load that is detectable but less than 200 copies/ml. This is a common feature in chronic HIV management and does not imply failure of treatment. However, good compliance should be reinforced and viral load should be repeated within one month so exclude virological failure or rebound. A CD4 count would not be reassuring in this circumstance as if normal there could still be a rebound happening. Serology only aids diagnosis. Viral resistance testing is useful in order to determine the cause of virological failure. Routine monitoring is not appropriate as a viral rebound will need to be excluded.

HIV monitoring

HIV, once stable, should be monitored with a viral load every six months and CD4 counts annually at least once virological suppression, defined as sustained viral load less than <50 copies/ml, is established. If the viral load increases to between 50-200 copies/ml on a single sample this is defined as a viral blip and is unlikely to be significant, but a viral load should be re-tested within one month. If a viral load above >200 copies/ml is sustained this is a virological rebound and can imply failure. This can be due to new viral resistance, and, if suspected, would need resistance testing.

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A 15-year-old male is investigated following a one week history of fever, non-productive cough, sore throat and headaches. Today he noticed a skin rash. His temperature is 38.5ºC, pulse is 90/min, blood pressure is 115/78 mmHg and respirations are 16/min.

On examination his throat is hyperaemic, but there is no cervical lymphadenopathy. Chest auscultation and percussion reveal no abnormalities. You note dusky red, target shaped skin lesions over all four extremities. Chest x-ray reveals interstitial infiltrates in the left lower lobe. Sputum Gram stain reveals polymorphonuclear cells but no organisms.

Which of the following organisms is most likely responsible for this presentation?

A. Streptococcus pneumoniae
B. Haemophilus influenzae
C. Legionella pneumophila
D. Mycoplasma pneumoniae
E. Epstein Barr virus

D. Mycoplasma pneumoniae

Stereotypical history of mycoplasma pneumonia: worsening flu-like symptoms and a dry cough. Erythema multiforme is noted on examination

This young man presents with a history suggestive of atypical pneumonia. Atypical pneumonia is distinguished from typical pneumonia by its more indolent course, non-productive cough and higher incidence of extra-pulmonary symptoms. This patients low grade fever and weeks worth of symptoms are consistent with an indolent course. Furthermore, he has multiple extra-pulmonary symptoms including headache, sore throat and skin rash. The different types of atypical pneumonia can present similarly and are at times challenging to distinguish. In this case, the skin rash (erythema multiforme) is typical of *Mycoplasma*. *Mycoplasma* is an organism that does not have a cell wall, and therefore does not stain with Gram stain; hence the finding of only polymorphonuclear cells on sputum Gram stain analysis.

Choice 1: *Pneumococcus* is the most frequent cause of community-acquired pneumonia requiring hospitalisation. Cough is often productive of rusty-coloured sputum. Compared to atypical pneumonias, pneumococcal pneumonia causes fewer extra-pulmonary manifestations but has a more virulent course. Sputum Gram stain would revel Gram positive diplococci.

Choice 2: *Haemophilus influenzae* is another common cause of community-acquired pneumonia. It presents like a typical pneumonia. Sputum Gram stain reveals small Gram negative bacilli.

Choice 3: *Legionella pneumophila* causes pneumonia with extra-pulmonary symptoms. Environmental water sources are often involved in its spread. It occurs most frequently in elderly patients and smokers. The urine *Legionella antigen* test is helpful in making the diagnosis.

Choice 5: Epstein Barr virus causes infectious mononucleosis, common in adolescents. Sore throat is the most common presenting symptom. Fatigue, malaise, headache and abdominal pain are also common. Concomitant pneumonia is not usually seen.

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A 24-year-old man attends the sexual health clinic complaining of urethral discharge and dysuria following an episode of unprotected intercourse. Examination of the external genitalia confirms purulent discharge but no other abnormalities. There is no previous history of sexually transmitted infection.

You undertake microscopy of the discharge which reveals many white blood cells but no organisms. A urine sample is sent for nucleic acid amplification tests (NAAT) for *Chlamydia trachomatis* and *Neisseria gonorrhoea*. You treat him empirically with a course of doxycycline for presumed chlamydial infection.

He returns for review a week later. You note both NAAT results to be negative. However, the patient tells you his symptoms have fully resolved.

What is the most likely explanation?

A. Mycoplasma genitalium infection
B. False negative NAAT for Chlamydia trachomatis
C. False negative NAAT for Neisseria gonorrhoea
D. Herpes simplex virus infection
E. Urinary tract infection

A. Mycoplasma genitalium infection

Urethritis in a male, negative for Gonorrhoea and Chlamydia →?Mycoplasma genitalium

The correct answer is ***Mycoplasma genitalium* infection**. This has emerged as the leading cause of non-gonococcal urethritis (NGU). Urethritis is evidenced in this case by the symptoms of dysuria and discharge combined with the presence of white cells in the discharge. NGU is diagnosed when features of urethritis exist in the absence of a positive test for gonorrhoea. Other explanations include infection with *Chlamydia* or organisms such as *Ureaplasma urealyticum*.

False negative NAAT for *Chlamydia trachomatis* is incorrect. Although improvement with doxycycline would fit with *Chlamydia*, the sensitivity of NAATs reaches 97% in some studies, making this a less likely explanation.

False negative NAAT for *Neisseria gonorrhoea* is incorrect. The sensitivity of NAATs for gonorrhoea reaches 98-99% in some studies. We would also not expect a symptomatic improvement with doxycycline.

Herpes simplex virus infection is incorrect. HSV most commonly presents with painful genital ulceration. Less commonly, patients may present with dysuria and discharge in the absence of visible ulceration. However, we are told this patient has had no previous STIs. The first episode of genital HSV is often severe and prolonged in the absence of anti-viral treatment and the spontaneous improvement in 7 days described here would be unusual. Subsequent recurrences are often less severe and more short-lived.

Urinary tract infection is incorrect. Urinary tract infection frequently causes dysuria but urethral discharge would be an unusual feature. It is also very unusual to see urinary tract infections in young males in the absence of a structural abnormality of the urinary tract or an iatrogenic cause such as catheterisation.

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A 28-year-old woman presents to the genitourinary medicine clinic with four days of dysuria and vaginal discharge. She has no past medical history and is on no regular medications. She had unprotected sexual intercourse with a casual partner two weeks previously.

Speculum examination revealed cervicitis and clear vaginal discharge. Examination of the genitalia was otherwise unremarkable.

A nucleic acid amplification test demonstrates the culprit organism.

Based on the clinical history, the patient is prescribed doxycycline and makes a full recovery.

Which organism is likely to be responsible for the clinical presentation?

A. Trichomonas vaginalis
B. Candida albicans
C. Chlamydia trachomatis
D. Gardnerella vaginalis
E. Neisseria gonorrhoeae

C. Chlamydia trachomatis

Chlamydia - treat with doxycycline

Chlamydia trachomatis is the correct answer. This sexually transmitted infection can either be asymptomatic or may present with dysuria, cervicitis and a clear vaginal discharge. It is diagnosed by a nucleic acid amplification test. The first line antibiotic is doxycycline.

Trichomonas vaginalis is incorrect. This can cause dysuria and cervicitis and is also diagnosed by a nucleic acid amplification test.. However, the discharge tends to be green-ish and the treatment is with metronidazole.

Candida albicans is incorrect. The clinical picture in this case tends to be a creamy, itchy discharge. It is a fungal infection and is treated with topical or oral anti-fungal agents.

Gardnerella vaginalis is incorrect. This is a typical cause of bacterial vaginosis. A 'fishy' odour is commonly described in this disorder and the treatment is with metronidazole.

Neisseria gonorrhoeae is incorrect. This shares many of the same symptoms as *Chlamydia trachomatis* and can also be diagnosed via a nucleic acid amplification test. However, the treatment is with ceftriaxone.

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A 28-year-old male presents to the emergency department complaining of an 8-day history of red eyes, fever, myalgia and headache. He has no significant past medical history and is on no regular medications. He has recently returned from a holiday with friends where they were kayaking and swimming in a freshwater lake. They had to abandon their trip earlier than expected as there was concerns about exposure to raw sewage in the lake.

His observations are heart rate 87 beats per minute, blood pressure 111/56 mmHg, respiratory rate 19/minute, oxygen saturation 96% on air and temperature 38.3ºC.

On examination, there is bilateral erythema of the conjunctiva. Cardiovascular and respiratory examinations are unremarkable. Abdominal examination reveals mild right upper quadrant tenderness and hepatomegaly. There is no photophobia or nuchal rigidity.

Urinalysis is unremarkable.

Plain radiography of the chest demonstrates clear lung fields.

Blood tests:

Hb	141 g/L	Male: (135-180)Female: (115 - 160)
Platelets	414 * 10 /L 9	(150 - 400)
WBC	11.8 * 10 /L 9	(4.0 - 11.0)
Na +	141 mmol/L	(135 - 145)
K +	4.1 mmol/L	(3.5 - 5.0)
Urea	9.2 mmol/L	(2.0 - 7.0)
Creatinine	141 µmol/L	(55 - 120)
CRP	68 mg/L	(< 5)
Bilirubin	38 µmol/L	(3 - 17)
ALP	167 u/L	(30 - 100)
ALT	190 u/L	(3 - 40)
Î³GT	111 u/L	(8 - 60)
Albumin	34 g/L	(35 - 50)

Given the clinical context, what is the most appropriate investigation to make the diagnosis?

A. Blood culture
B. Urine polymerase chain reaction (PCR)
C. Serology
D. Urinary antigens
E. Urine culture

C. Serology

Serology is the most commonly used test for the diagnosis of leptospirosis, however, antibodies may not be present until after 7 days

Serological testing is the correct answer. The diagnosis is leptospirosis, as evidenced by a history of fever, myalgia, headache and subconjunctival haemorrhage in association with hepatorenal failure in a patient with a potential geographic exposure to infected urine (freshwater exposure). The most commonly used test to diagnose leptospirosis is serological testing for IgM antibodies to leptospirosis. The disadvantages with this method of investigation are false positives, the inability for the test to diagnose early infection and the fact that antibodies can persist in the sera for months after initial infection, making interpretation of timing of infection difficult in cases of diagnostic uncertainty. Antibodies are not usually present until the infection has been present for seven days. In this case, the patient has presented with eight days of symptoms and therefore antibody testing should be positive.

Blood culture is the wrong answer. Culture testing for leptospirosis can take up to three months and is therefore predominantly used for retrospective diagnosis.

Urine polymerase chain reaction (PCR) testing is incorrect. While this technique is effective in identifying leptospira infection, it is not as commonly used as serological testing as its use is precluded by cost and technical factors in non-reference laboratories.

Urinary antigen testing is incorrect. This is used to diagnose pneumococcal and legionella pneumonia infections. The clinical history is not suggestive of a pneumonia.

Urine culture is incorrect. The use of this method of testing is precluded by the fact that cultures may take 8-12 weeks to form.

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A 34-year-old man is referred to the medical admissions unit with joint pain and swelling in his hands and feet. One month prior to his attendance, he had developed an illness while travelling in Colombia, which was characterized by fever, headache, rash and debilitating polyarthralgia. His treating physicians noted mild hepatomegaly at the time. He recovered well from this episode without intervention, except for his ongoing musculoskeletal symptoms.

Examination reveals a small joint polyarthritis of the hands and feet. Abdominal examination was unremarkable.

Serological testing is positive for an RNA virus.

What is the likely diagnosis?

A. Chikungunya virus
B. Dengue
C. Epstein Barr Virus (EBV)
D. Parvovirus
E. Zika virus

A. Chikungunya virus

Chikungunya can present with debilitating joint pain

Chikungunya is the correct answer. This is an RNA alpha-virus characterized by sudden onset fever accompanied by skin rashes and joint pain followed by persistent rheumatic symptoms. In contrast to the other diagnoses, which can all cause headache, fever and rash, the polyarthralgia is said to be far more debilitating and it is most associated with the development of persistent musculoskeletal symptoms. Furthermore, hepatomegaly can be a feature of this condition but this is not seen in Zika or dengue. While hepatomegaly is a feature of EBV, this is a DNA virus and is less likely to cause persistent musculoskeletal symptoms.

Zika virus is characterized by a pruritic maculopapular rash accompanied by other symptoms such as fever, headache, conjunctivitis, myalgia and arthralgia. It is an RNA virus but is much less likely to cause persistent musculoskeletal symptoms.

Dengue is incorrect. This is another RNA arbovirus that causes fever, rash and arthralgia in addition to other features such as thrombocytopenia. However, hepatomegaly is not a feature and it is not associated with the development of persistent arthritis.

Epstein Barr Virus (EBV) is incorrect. This is a DNA virus. Musculoskeletal manifestations are less prominent.

Parvovirus is incorrect. This can cause rash, hepatomegaly and polyarthritis but it is a DNA virus.

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A 38-year-old farmer is admitted to the acute medical unit with a 2-week history of fever and dry cough. He describes feeling increasingly more lethargic with generalised body aches and headaches. He has no past medical history of note and takes no regular medications.

On auscultation, there are scattered crackles. His cardiovascular examination is unremarkable. There is mild tenderness in the right upper quadrant, but his abdomen remains soft with no palpable organomegaly.

Laboratory tests:

Hb	139 g/L	(135-180)
Platelets	499 * 10 /L $_9$	(150 - 400)
WBC	16.1 * 10 /L $_9$	(4.0 - 11.0)
Na$^+$	144 mmol/L	(135 - 145)
K$^+$	4.6 mmol/L	(3.5 - 5.0)
Urea	6.1 mmol/L	(2.0 - 7.0)
Creatinine	110 µmol/L	(55 - 120)
Bilirubin	15 µmol/L	(3 - 17)
ALP	80 u/L	(30 - 100)
ALT	120 u/L	(3 - 40)
CRP	199 mg/L	(< 5)

What is the most likely causative organism for this patient's presentation?

A. Brucella melitensis
B. Coxiella burnetii
C. Mycoplasma pneumoniae
D. Staphylococcus aureus
E. Streptococcus pneumoniae

B. Coxiella burnetii

Coxiella burnetii is a cause of atypical pneumonia and Q fever. It is usually transmitted by infected cattle or sheep

This patient has an infection with ***Coxiella burnetii*** a zoonotic disease more commonly seen in those who work with animals such as farmers and veterinarians. Infection gives rise to Q fever that can present with a variety of symptoms ranging from fever, malaise and myalgia to atypical pneumonia or infective endocarditis. Auscultatory crackles and a dry cough with fever and fatigue are suggestive of atypical pneumonia. Furthermore, *Coxiella burnetii* can cause transaminitis as demonstrated by this patient's deranged liver function tests.

Brucella melitensis is another zoonotic disease secondary to close contact with infected animals or from ingestion of unpasteurised milk or undercooked meat. It is more common in the Middle East, farmers, vets, and abattoir workers. Symptoms include fever and malaise, as seen here. However, features of pneumonia are not common in Brucellosis making it a less likely diagnosis.

A ***Mycoplasma pneumoniae*** infection commonly presents with a preceding phase of flu-like symptoms before the onset of respiratory symptoms including shortness of breath and productive cough. Extra-pulmonary symptoms are common in patients with *Mycoplasma pneumoniae* and include erythema multiforme and haemolytic anaemia. The absence of these extra-pulmonary symptoms and the occupational history as a farmer make the diagnosis of Q fever more likely.

Staphylococcus aureus is a common cause of pneumonia in patients following infection with the influenza virus. Although this patient gives an initial history of flu-like symptoms, it does not explain his transaminitis and suggests an alternative diagnosis is more likely.

Streptococcus pneumoniae is the most common bacterial cause of community-acquired pneumonia. However, it does not explain this patient's transaminitis or prodrome of fever and myalgia. Furthermore, an occupational history as a farmer makes alternative zoonotic diseases, such as Q fever, more likely.

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A 49-year-old man is admitted to the acute medical unit with a 2-week history of pain and difficulty when swallowing. He reports having lost approximately 2-3kg in weight from reduced oral intake. His past medical history includes previous hospital admissions for lower respiratory tract infections and lower limb deep vein thrombosis.

On examination, he appears cachectic. There are multiple, well-defined white plaques over the oral mucosa. His abdomen is soft and non-tender with no palpable masses.

F. CD4 count	G. < 100 cells/mm^3

What is the most appropriate management for this patient?

A. Aciclovir
B. Caspofungin
C. Co-amoxiclav and metronidazole
D. Fluconazole
E. Nystatin and chlorhexidine mouthwash

D. Fluconazole

Candidiasis in immunocompromised patients should be treated using high dose fluconazole

This patient has a diagnosis of oesophageal candidiasis given the classical history of painful swallowing and visible white mucosal plaques on examination. It is particularly more common in immunocompromised individuals (e.g. such as patients with HIV). The treatment of choice for treating candidiasis in immunocompromised patients is with high-dose **fluconazole** or itraconazole.

Aciclovir is used to treat viral infections, including herpes simplex and herpes zoster. Typically viral rashes such as herpes simplex present with ulceration or vesicles. The presence of a white mucosal coating in an immunocompromised patient is highly suggestive of candidiasis, for which antifungal medication is needed, rather than antivirals like aciclovir.

Caspofungin is not a first-line treatment for oesophageal candidiasis. It is reserved for cases of refractory disease that do not respond to initial treatment with fluconazole. As first-line therapy has not yet been initiated, it would be inappropriate to start the patient on more invasive treatment such as caspofungin.

Co-amoxiclav and metronidazole would be indicated if there were concerns that this patient was suffering from a bacterial infection. However, the classical history of odynophagia and white mucosal coating in an immunocompromised patient makes candidiasis much more likely to be the diagnosis.

Nystatin and chlorhexidine mouthwash would normally form part of the initial management in immunocompetent patients. However, this patient has a CD4 count of < 100 cells/mm^3 suggesting significant immunocompromise. Therefore, stronger treatment such as high-dose fluconazole is needed.

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A 55 year old man attended his General Practitioner to complain of on-going fatigue and lethargy over recent months. He reported experiencing recurrent minor infections meaning that he had rarely been feeling well for any significant length of time. His GP records supported this story with the patient having been prescribed courses of antibiotics for a leg cellulitis, suppurative otitis media and sinusitis (twice) over the past 9 months. The patient reported a good appetite and no recent weight loss, fevers or night sweats. He denied any symptoms of arthritis, skin rashes, photophobia or dry eyes. There were no significant gastrointestinal or genitourinary symptoms.

Past medical history included obesity, hypertension, impaired fasting glucose tolerance and osteoarthritis of the knees. Drug therapy included ramipril 2.5 mg daily, bendroflumethiazide 2.5 mg daily and paracetamol as required. The patient was divorced with two grown up children. He was an ex-smoker who rarely drank alcohol. The patient had recently retired having previously worked as a salesperson for luxury yachts, an occupation that had involved extensive travel around the world.

On examination, the patient was significantly overweight (BMI 36 kg / m^2). Abdominal examination did not demonstrate any jaundice or signs of chronic liver disease and no lymphadenopathy. Cardiovascular, respiratory and musculoskeletal examination was unremarkable. Given the patients concerns, his General Practitioner arranged some basic blood tests.

Haemoglobin	16.0 g / dL
Mean cell volume	85 fL
White cell count	5.2 * 10 /l9
Neutrophils	4.2 * 10 /l9
Lymphocytes	0.6 * 10 /l9
Monocytes	0.1 * 10 /l9
Eosinophils	0.1 * 10 /l9
Basophils	0.2 * 10 /l9
Platelets	358 * 10 /l9
Urea	6.8 mmol / L
Creatinine	110 micromol / L
Sodium	141 mmol / L
Potassium	3.9 mmol / L
Albumin	34 g / L (reference 35-50)
Alkaline phosphatase	98 U / L (reference (35-100)
ALT	28 U / L (reference 3-36)

Bilirubin	21 micromol / L (reference < 26)
Total protein	85 g / L (reference 60-80)
B12	355 pmol / L (reference 74-516)
Folate	30 nmol / L (reference 7-36)
Serum immunoglobulins	Normal electrophoresis strip

Comparison with a routine blood test taken 6 months previously had shown a similar full blood count differential.

What is the most appropriate next line investigation?

A. Human immunodeficiency virus antibody testing
B. Anti-nuclear antibody
C. Rheumatoid factor
D. Epstein-Barr virus serology
E. Serum angiotensin-converting enzyme

A. Human immunodeficiency virus antibody testing

The patient has a persistent lymphopenia, raised total protein and low albumin in the setting of fatigue and recurrent minor infections. These findings could all be explained by nascent HIV infection. The patients history of international travel may place him at increased risk of HIV infection and a full sexual history would be required.

The other investigations listed would assess for other possible causes of isolated lymphopenia, however there is no clinical evidence of connective tissue disease, EBV infection or sarcoidosis to

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A 28-year-old man who has recently immigrated from Nigeria presents with a penile ulcer. He reports that it initially started as a small lump but then later progressed to a painful ulcer.

On examination, there is a 7mm diameter tender single ulcer with an undermined ragged edge just proximal to the glans of the penis. Examination of the testes and anal region is unremarkable. There is tender inguinal lymphadenopathy.

What is the most likely diagnosis?

A. Syphilis
B. Herpes simplex virus
C. Granuloma inguinale
D. Lymphogranuloma venereum
E. Chancroid

E. Chancroid

Chancroid is a bacterial sexually transmitted infection characterised by painful sores on the genitalia. It is caused by the gram-negative coccobacillus *Haemophilus ducreyi*. It is a disease found primarily in developing countries.

H. ducreyi enters the skin through microabrasions incurred during sexual intercourse. A local tissue reaction leads to the development of an erythematous papule, which progresses to a pustule in 4- 7 days. It then undergoes central necrosis to ulcerate.

The ulcer can range in size from 3 to 50 mm across. It is painful and has sharply defined undermined borders

Other features include:

- Irregular or ragged borders
- A base that is covered with a grey or yellowish-gray material
- A base that bleeds easily if traumatised or scraped
- Painful lymphadenopathy in 30 to 60% of patients.
- Dysuria and dyspareunia in females

About half of infected men have only a single ulcer. Women frequently have four or more ulcers, with fewer symptoms.

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The Medical Emergency Team (MET) is summoned to the Surgical Unit to assist with the management of an acutely unwell patient.

The patient is a 21-year-old male who underwent open surgery for perforated appendicitis 3 days ago. The Surgical Registrar informs you that faecal contamination of the abdomen was noted during the operation and that a peritoneal washout was performed.

24 hours later, the patient began to complain of worsening abdominal pain. He became febrile in the early hours of the morning and blood cultures were taken. Since then, he has become progressively more unwell. He was taken down for an urgent abdominal ultrasound midafternoon, but the nurses were so concerned about his condition when he arrived back on the ward that a MET call was put out.

On examination, the patient is responsive to voice. He is febrile at 38.9ºC, his pulse is 131bpm and his blood pressure is 72/53mmHg. His peripheries are warm and clammy. Palpation of the abdomen reveals localised tenderness and guarding in the right iliac fossa. The surgical wound appears clean with minimal surrounding erythema.

As you prepare to place a large bore IV cannula the Surgical FY1 passes you some results that have recently been phoned through:

Abdominal ultrasound	Anechoic fluid collection in the right iliac fossa
Blood culture	Gram-positive cocci both bottles - further information to follow

Which of the following organisms is most likely to be isolated from the blood culture?

A. Staphylococcus epidermidis
B. Escherichia coli
C. Streptococcus pyogenes
D. Enteroccocus faecalis
E. Staphylococcus aureus

D. Enteroccocus faecalis

Although on first glance this may appear to be a 'surgical case', it is important to remember that MET or 'crash' teams are called to attend a variety of patients, some of whom may be admitted under the care of other specialties. On-call medical doctors need to be able to apply their knowledge to a range of situations, particularly when they fall outwith the remit of their day-today practice.

A significant amount of surgical information is presented in this vignette, however, the underlying diagnosis is given to the candidate and the question is actually designed to test the candidate's knowledge of pathogenic bacteria and the illnesses they cause.

Strep. pyogenes is a common cause of pharyngitis and soft tissue infections, occasionally giving rise to septicaemia. It would be unlikely to cause illness in the case described above.

Staph. aureus and Staph. epidermidis are commensal organisms that commonly cause septicaemia in critically ill patients, often secondary to the colonisation of indwelling lines. This vignette makes no mention of any such devices and we are told that the surgical wound is clean, making primary staphylococcal wound infection unlikely.

Escherichia coli is a gram-negative bacillus and is, therefore, inconsistent with the blood culture results described.

Enterococcus faecalis is a group D streptococcus. It is a gut commensal and a well-known cause of intra-abdominal infections. In the case described, it is likely that faecal contamination of the abdomen lead to the formation of an abscess and consequent Enterococcus bacteraemia.

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A 32 yr old Indian lady presented at the clinic with history of a gross swelling on her right leg from more than four years with associated fever and pain in her groin. Swelling was not reversible at night. On examination, the whole leg was oedematous ,bluish in colour, deep skin fold at junction of middle and lower one third, non discharging sinus above the fold anteriorly. There were no nodules or warts on her limbs.

Hb	144g/l (130 - 180)
Platelets	310 * 10 /l (150 - 400)9
WBC	9.2 * 10 /l (4.0 - 11.0)9
Neutrophil	3.5 * 10 /l (1.5 - 7.0)9
Lymphocyte	2.1 * 10 /l (1.5 - 4.09
Eosinophil	2.4 * 10 /l (0.04 - 0.4)9
Monocyte	1.1 * 10 /l (0<0.8)9

What is the drug of choice for the most likely diagnosis for the scenario above?

A. Praziquantel
B. Niclosamide
C. Ivermectin
D. Diethylcarbamazine
E. Metronidazole

D. Diethylcarbamazine

Diethylcarbamazine is the drug of choice for lymphatic filariasis

Filariasis is a disease group caused by filariae that affects humans and animals. In lymphatic filariasis, repeated episodes of inflammation and lymphedema lead to lymphatic damage, chronic swelling, and elephantiasis of the legs. Eosinophilia is a feature of filariasis. Diethylcarbamazine is the drug of choice for lymphatic filariasis.

In contrast, Praziquantel is used to treat infections caused by Schistosoma worms, which enter the body through skin that has come into contact with contaminated water. Symptoms include abdominal pain, diarrhea, bloody stool, or blood in the urine. Niclosamide is an antihelminthic that is active against tapeworm infestation which is transmitted mainly by eating infected raw or uncooked fish or beef and usually have intestinal discomfort, diarrhea, vomiting, weakness, and weight loss. Metronidazole is useful in treating infective diarrhoea with susceptible organisms.

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Randy is a 45-year-old male presenting with a new fever. He works as a farmer and noted fevers up to 39.6ºC over the past 72 hours with a dry cough. He has also been struggling to manage his sheep due to the ongoing headache and worsening muscle aches that have accompanied the fever. The headache was significant but was not associated with any neck stiffness or photophobia.

Randy does not note any other symptoms. He has not been in contact with anyone unwell and he has not been travelling out of the country recently.

Physical examination was otherwise unremarkable with only mild crackles heard on auscultation on his chest. No insect bites were noted. No lymphadenopathy was palpable.

Blood tests are as follow:

Hb	150 g/L	Male: (135-180)Female: (115 - 160)
Platelets	140 * 10 /L 9	(150 - 400)
WBC	6.0 * 10 /L 9	(4.0 - 11.0)
Na +	140 mmol/L	(135 - 145)
K +	3.9 mmol/L	(3.5 - 5.0)
Urea	6.0 mmol/L	(2.0 - 7.0)
Creatinine	110 µmol/L	(55 - 120)
Bilirubin	13 µmol/L	(3 - 17)
ALP	90 u/L	(30 - 100)
ALT	110 u/L	(3 - 40)
AST	100 u/L	(5-40)
Albumin	37 g/L	(35 - 50)

What would be the likely organism causing the likely diagnosis?

A. Rickettsia prowazekii
B. Coxiella burnetti
C. Francisella tularensis
D. Legionella spp.
E. Bartonella henselae

B. Coxiella burnetti

Farmer, fever, transaminitis ?Q fever

Given that the patient is a farmer, this exposes him to zoonotic infections. The presence of fevers and associated transaminitis raises the suspicion for Q fever, caused by *Coxiella burnetti*. Its natural reservoir exists within cattle and sheep. It typically presents with fevers, headaches, fatigue and muscle aches. Diagnosis of this is often made by serological testing and is treated with doxycycline. Chronic Q-fever is possible and can present with endocarditis.

Francisella tularensis causes tularemia. This is less likely given the lack of risk of exposure to its natural vector, rabbits or deer. Also, this will tend to present with unilateral lymphadenopathy with associated skin ulcers at the site of inoculation.

Legionella spp. causes Legionnaires disease which presents with fevers and headaches. However, this is less likely given the lack of risk factors of exposure including recent repairs to water supplies and exposure to a known Legionnaires case. Also, this tends to cause a productive cough rather than a dry cough.

Bartonella henselae causes cat-scratch disease and this is not likely given the absence of lymphadenopathy or rash at the site of inoculation. Besides, there is no mention that he has been exposed to a cat.

Rickettsia prowazekii causes epidemic typhus. This typically occurs in overcrowded areas as the disease is spread by body lice. This is also typically associated with a generalized rash beginning at the trunk before spreading to the extremities (sparing of face, palms and soles).

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A 23-year-old female presents to her general practitioner complaining of a two-day history of rash at the back of her knee. She has a past medical history of eczema and asthma and is on regular Symbicort and emollients. She is a non-smoker and non-drinker and has recently returned from a camping holiday where she had been doing some hiking through a forested area.

On examination, there is a bulls-eye lesion just above her right popliteal fossa. She is otherwise systemically well and apyrexial.

What is the most appropriate initial management?

A. Oral doxycycline
B. Oral flucloxacillin
C. Serological testing for Borrelia antibodies
D. Skin biopsy
E. Topical steroid

A. Oral doxycycline

Lyme disease can be diagnosed clinically if erythema migrans is present → give antibiotics

Oral doxycycline is correct. This patient has a clinical diagnosis of Lyme disease and therefore treatment should be started as soon as possible. Doxycycline is the first-line antibiotic choice. NICE recommend a course length of 21 days. Second-line options include amoxicillin and azithromycin.

Serological testing for *Borrelia* antibodies is incorrect. NICE states that Lyme disease can be diagnosed clinically if erythema migrans is present. Given the typical appearance of the rash and the suggestive clinical history of a recent visit to a forest, we can make a clinical diagnosis in this case. This patient should receive antibiotics in the first instance and we do not need to wait for laboratory results.

Oral flucloxacillin is incorrect. This would be an appropriate choice for cellulitis but this is not the correct diagnosis.

Skin biopsy is incorrect. There is no uncertainty regarding diagnosis.

Topical steroid is the wrong answer. This would be a treatment for a flare of eczema but the description of the rash in association with the clinical history does not fit this description.

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A 54-year-old man is brought into the emergency department with a 4-day history of fever, headache and reduced appetite. His partner brought him into the hospital today as he became more lethargic and drowsy today. He has a past medical history of coeliac disease and HIV.

On examination, his GCS is 12 (E2V4M6) with equal and reactive pupils. He has a temperature of 38.1ºC with a heart rate of 99 beats/min and a blood pressure of 135/80mmHg. There are no visible rashes.

A lumbar puncture is performed, the CSF results are shown below:

Opening pressure	30 cmH20	(10 - 20)
White cell count	50 x10 /L mostly lymphocytes 6	(< 5)
Protein	3.5 g/L	(0.2 - 0.4)
Glucose	2 mmol/L	(2.8 - 4.2)

What is the most likely causative organism?

A. PassMedicine
B. *Cryptococcus neoformans*
C. *Herpes simplex virus*
D. *Listeria monocytes*
E. *Neisseria meningitidis*
F. *Streptococcus pneumoniae*

B. Cryptococcus neoformans

In cryptococcal meningitis, CSF shows high opening pressure and elevated protein. The white cell count may vary according to HIV status

Cryptococcus neoformans is correct. This patient has evidence of meningitis given the history of fever, headache and neurological dysfunction. The background of HIV suggests a likelihood of immunosuppression. In immunocompromised patients, it is important to consider alternative organisms that could cause this presentation rather than just those that are most prevalent in the general population. Looking at the lumbar puncture results from this patient, there is a significantly raised opening pressure, elevated protein and low glucose. The white cell count is only marginally raised and is predominantly lymphocytic. It is important to remember that the white cell count in CSF analysis can vary in patients with HIV depending on the severity of their immunocompromise. *Cryptococcus neoformans* is the most common fungal infection of the CNS in patients with HIV. It causes the lumbar puncture findings seen with this patient's presentation and is, therefore, the most likely causative organism.

Herpes simplex virus is incorrect. Viral meningitis can be seen in immunocompromised and immunocompetent individuals with common responsible viruses including enteroviruses (most common), herpesviruses, mumps and measles. Viral meningitis produces a CSF with elevated protein and a raised white cell count of mostly lymphocyte predominance as seen here. However, it would not cause a raised opening pressure and the CSF glucose is not reduced in patients with viral meningitis making this an unlikely diagnosis.

Listeria monocytes is incorrect. *Listeria monocytes* is an important organism to consider in patients with meningitic symptoms who are over the age of 50 years. Although the majority of bacteria cause neutrophil-dominant leucocytosis in the CSF, *Listeria monocytes* can also cause a lymphocytic predominance. However, the raised opening pressure points away from *Listeria monocytes* as the causative organism.

Neisseria meningitidis is incorrect. This is one of the more common causes of bacterial meningitis. Whilst *Neisseria meningitidis* can cause an elevated opening pressure, the white cell count is generally made up of neutrophils rather than lymphocytes as seen in this scenario.

Streptococcus pneumoniae is incorrect. Like *Neisseria meningitidis*, this is one of the most common causes of bacterial meningitis. However, the lymphocyte-predominant white cell count on CSF analysis makes this organism less likely to be responsible for this patient's presentation.

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A 29-year-old man presents to his first HIV clinic appointment. He has routine three month HIV tests. He received contact tracing notification last week and was tested and found to be HIV positive. He has just started antiretroviral treatment.

At clinic he mentions that he is due to go to South Africa. He hasn't yet had his travel vaccinations and is worried about what affect the new diagnosis of HIV will have on his travel plans. He is aware that currently immunosuppressed and at risk of infections and is committed to taking his tablets.

Investigations:

Haemoglobin	113 g/L (130-180)
White cell count	6.5 109/L (4.0-11.0)
Neutrophil count	5.4 109/L (1.5-7.0)
Platelet count	170 109/L (150-400)
CD4 count	180 cells/mm3 (600-1500)

Which of the following vaccinations is safe to be given?

A. Yellow fever
B. BCG
C. Oral polio
D. Meningococcal C
E. Varicella

D. Meningococcal C

Given this gentleman recent diagnosis with HIV he is immunocompromised which is reflected by his low CD4 count. He should not be given live vaccines as he is at risk of disseminated infection.

Yellow Fever, BCG, Oral Polio and Varicella are all live vaccines. Meningococcal C is a conjugate vaccine and can be given cautiously to an immunocompromised patient.

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A 34-year-old man with chronic immune thrombocytopenia has not responded to medical therapy and so is scheduled to undergo an elective splenectomy.

According to guidelines, he requires the following vaccinations:

A. Pneumovax (Pneumococcus), Haemophilus Influenzae, MMR (Measles, Mumps, Rubella), Annual influenza vaccine
B. Haemophilus Influenzae, Meningococcus C, Annual influenza vaccine
C. Pneumovax (Pneumococcus), Haemophilus Influenzae, Meningococcus C, Annual influenzavaccine
D. Pneumovax (Pneumococcus), Haemophilus Influenzae, Meningococcus C
E. Meningococcus C, Haemophilus Influenzae, MMR (Measles, Mumps, Rubella), Annualinfluenza vaccine

D. Pneumovax (Pneumococcus), Haemophilus Influenzae, Meningococcus C, Annual influenzavaccine

Risk of infection following splenectomy is with encapsulated bacteria e.g *Streptococcus pneumoniae*. *Haemophilus influenzae* type B, *Neisseria meningitidis*. Therefore patients should be immunised against these infections. MMR vaccination is not required.

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A 66 year old lady, originally from Pakistan, has lived in the UK for 10 years. She has type 2 diabetes mellitus which is controlled with metformin only.

For the last few years she has had multiple lumps over her whole body but this hasnt bothered her as it has been there for several years and not got too much worse. She has been seeing the diabetic foot care team due to several traumatic show healing ulcers on her feet.

Over the past few weeks she has been feeling increasingly unwell. She has had a fever, pain in her knees and elbows, sore painful eyes. She also complains of painful swelling in her ring and little fingers on her right hand.

On examination her temperature is 37.8C. Her pulse is 91 beats/minute and her RR is 18 breaths/minute. Her oxygen saturations are 97% on room air.

She has a multiple nodular lesions over her whole body in a symmetrical distribution. Sizes of these nodule range from 2cm - 4cm. These lesions are not painful, in fact with the larger ones she is unable to feel you touching them. On her shins there are several hot painful nodules which are uncomfortable to touch. She has a saddle deformity of her nose. There are several scars on her hands, and an ulcer on the lateral dorsum of her foot. Cardiorespiratory and abdominal examination is essentially normal. Both knees and elbow are slightly erythematous and swollen. She has weakness abducting her little and index finger on her left hand and has no sensation in her left little finger. There is a non-painful 1x4 cm lump two centimetres distal to her left medical epicondyle.

Urinalysis reveals ++ of protein and + of blood. Blood sugar: 8.8 mmol/L

On the clinical information alone what investigation is most likely to secure the diagnosis?

A. Serum cANCA antibody
B. Serum dsDNA antibody
C. Renal biopsy
D. Skin biopsy
E. Serum ANA antibody

D. Skin biopsy

With skin lesions and peripheral nerve involvement leprosy should certainly be on a differential diagnosis. Other give away features here are the anaesthetic skin lesions and nerve thickening. The erythema nodosum like lesions and the joint, renal and conjunctival involement indicate a type 2 reaction which occur in patients with lepromatous leprosy (usually following treatment).

Skin biopsy in this case will reveal the diagnosis. This would save her the potentially disastrous consequences of immunosupression.

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A 46 year old Indian gentleman who moved to the UK four years previously presents to the Emergency Department complaining of a numb left foot. He has a background history of type 2 diabetes mellitus, hypertension and gastro-oesophageal reflux disease. His latest haemoglobin A1c (HbA1c) is 6.4%. On examination he has reduced sensation over the left heel and plantar aspect of the foot and six hypoesthesic, hypopigmented patches over both legs.

What is the most appropriate management?

A. Referral to diabetes specialist clinic for management of peripheral neuropathy
B. Dapsone, clofazimine
C. Rifamipicin, clofazimine, isoniazid
D. Rifamipicin, dapsone, clofazimine
E. Dapsone, terbinafine, rifampicin

D. Rifamipicin, dapsone, clofazimine

The travel history, the distribution of sensory peripheral loss and the pattern of hypoesthesic skin patches are the clues to this patient's diagnosis. Mycobacterium leprae is one of the most common causes of peripheral neuropathy worldwide and has a vast spectrum of presentations from the typical skin manifestations described here to systemic involvement involving the respiratory tract, kidneys, testes and bones.

When there is neurological involvement, the posterior tibial nerve is most commonly affected, (the pattern of sensory loss being described here) however there may equally be involvement of other nerves eg. ulnar, median, lateral popliteal and facial.

Although he may have some underlying diabetic peripheral neuropathy, the pattern of sensory loss would be symmetrical and therefore targeting treatment towards his diabetes will not necessarily help here.

For the treatment of multibacillary leprosy, the WHO have recommended a multi-drug therapy regimen of dapsone, rifampicin and clofazimine for a two year period.

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A 36-year-old Sri-Lankan woman presents to the GP with a malar rash. On examination she has a erythematous rash that is warm to touch with some underlying oedema over both cheeks and the bridge of her nose.

She reports feeling low in energy over several months and describes a feeling of numbness in her feet and hands.

Investigations:

haemoglobin	125 g/L (115-165)
white cell count	9.4 Ã— 109/L (4.0-11.0)
platelet count	220 Ã— 109/L (150-400)
erythrocyte sedimentation rate	50 mm/1st h (<30)
serum urea	7.0 mmol/L (2.5-7.0)
serum creatinine	105 mol/L (60-110)
serum alanine aminotransferase	17 U/L (5-35)
serum aspartate aminotransferase	26 U/L (1-31)
serum complement C3	80 mg/dL (65-190)
serum complement C4	45 mg/dL (15-50)
serum C-reactive protein	145 mg/L (<10)
anti-double-stranded DNA antibodies	negative
c-ANCA	negative
p-ANCA	negative
antinuclear antibodies	1:20 (negative at 1:20 dilution)

An outpatient rheumatology appointment is arranged and she is started on a course of steroids in the interim. Her symptoms rapidly improve and the rash almost completely resolves however she is left with persistent numbness that appears to be worsening.

What is the most likely underlying diagnosis?

A. Granuloma annulare
B. Annular psoriasis
C. Systemic lupus erythematosus
D. Leprosy
E. Cutaneous leishmaniasis

D. Leprosy

The diagnosis here is leprosy. Leprosy can present along a spectrum from borderline to leprotamous and can often be mistaken for conditions like psoriasis, SLE and granuloma annulare.

The key distinguishing feature is the presence of anaesthetic patches that are present in leprosy but none of the other condition. A full clinical examination is not complete until sensation has been checked over the skin lesions with a microfilament.

The presence of anaesthetic patches should prompt you to look for other hallmarks of disease such as thickened nerves (often easiest to feel are the median nerve in the wrist the common peroneal nerve around the lateral head of the fibula and the ulnar nerve at the elbow.) the loss of hair (in and around lesions and often in the eyebrows) and the presence of neuropathy. Diagnosis is made by demonstrating acid fast bacilli within a cutaneous nerve on microscopy.

The severe skin changes seen with leprosy are not caused by the bacteria themselves but the bodys own immune response towards the bacilli. As a result steroids would improve the physical appearance of skin lesions but at the same time accelerate disease as the bacteria proliferate unhindered. This corresponds to the case outlined above where the patients neurological symptoms worsen yet the skin disease improves on starting steroids.

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A 34-year-old man presents to the emergency department with three days of fever, muscle ache, retro-orbital headache and a rash. He has returned from Honduras 5 days ago. He has no past medical history and does not take any regular medications.

Observations:

- Heart rate 101 beats/min
- Oxygen saturations 97% on room air
- Respiratory rate 20 breaths/min
- Temperature 38.5ºC
- Blood pressure 124/82 mmHg

On examination, his face appears flushed. There is a maculopapular rash covering most of his body. There is no splenomegaly. There is no synovitis.

Blood tests:

Hb	121 g/L	Male: (135-180)Female: (115 - 160)
Platelets	89 * 10 /L ^{9}	(150 - 400)
WBC	4.4 * 10 /L ^{9}	(4.0 - 11.0)
Na$^+$	137 mmol/L	(135 - 145)
K$^+$	4.2 mmol/L	(3.5 - 5.0)
Urea	5.2 mmol/L	(2.0 - 7.0)
Creatinine	88 µmol/L	(55 - 120)
CRP	22 mg/L	(< 5)
Bilirubin	14 µmol/L	(3 - 17)
ALP	111 u/L	(30 - 100)
ALT	143 u/L	(3 - 40)
Albumin	34 g/L	(35 - 50)

What is the most likely diagnosis?

A. Chikungunya
B. Dengue
C. Malaria
D. Typhoid
E. Zika

B. Dengue

Retro-orbital headache, fever, facial flushing, rash, thrombocytopenia in returning traveller → ?dengue

Dengue is the correct answer. Dengue virus is transmitted by the Aedes mosquito. It is endemic to Honduras. Facial flushing and a retro-orbital headache are characteristic along with severe myalgia (i.e 'breakbone fever'). Thrombocytopenia and deranged liver function tests are also common.

Chikungunya is incorrect. This can cause fever in the returning traveller and is another virus transmitted by the Aedes mosquito that is found in Honduras. While this condition can cause fever and myalgia, arthralgia and arthritis are almost ubiquitous in this condition and their absence makes this much less likely.

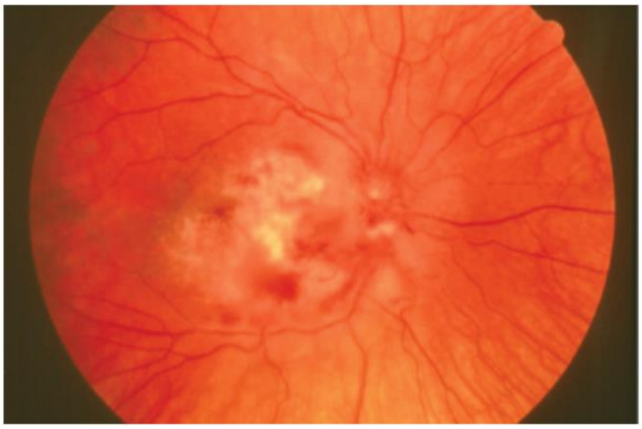
Malaria is incorrect. Malaria is present in Honduras and can cause fever and thrombocytopenia. However, malaria is typically not associated with a maculopapular rash and facial flushing and therefore dengue is more likely.

Typhoid is incorrect. This is another common cause of fever in the returning traveller. This can cause fever, thrombocytopenia and deranged liver function tests. However, it is often associated with abdominal pain and constipation. Additionally, the rash is typically described as 'rose spots' occurring on the lower chest and abdomen rather than a diffuse maculopapular rash and facial flushing. Severe myalgia is also less common. Therefore, dengue is more likely.

Zika is incorrect. This is another virus transmitted by the Aedes mosquito. However, it is not typically associated with a high fever (usually <38ºC) and is often associated with conjunctivitis and itching, which are absent in this case. Dengue is, therefore, more likely.

Question 260 of 277

A 42-year-old man who has recently emigrated to the UK is seen in the HIV clinic. He was diagnosed with HIV 5 years ago and has only sporadically taken anti-retroviral therapy during that period. He complains of blurred vision. Fundoscopy shows the following:



What is the most appropriate treatment?

A. Intravenous ganciclovir
B. Oral pyrimethamine and sulfadiazine, plus folinic acid
C. Intravenous methylprednisolone
D. Oral mebendazole
E. Oral co-trimoxazole

A. Intravenous ganciclovir

The fundoscopic image shows changes consistent with cytomegalovirus retinitis.

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A 35 year old known HIV positive patient presents with a gradual deterioration in his vision. He states that this has happened over the course of a few weeks. You note from his previous records that he is known to be poorly compliant with his antiretroviral therapy. His most recent CD4 count is 177 cells/mm3.

Given the most likely diagnosis, what would be the initial management strategy?

A. Oral aciclovir
B. Intravenous pentamidine

C. Oral pyrimethamine and sulfadiazine, plus folinic acid
D. Intravenous ganciclovir
E. Oral co-trimoxazole

D. Intravenous ganciclovir

This gentleman has developed cytomegalovirus retinitis treatment of which should be with intravenous ganciclovir or its prodrug oral valganciclovir.

CMV retinitis:

- Presents in those who are HIV +ve with CD4 counts typically <200 cells/mm3.
- Patients may be asymptomatic initially, but tend to present with a gradual deterioration in their visual acuity and visual blurring.
- Typical fundoscopic features described include the 'pizza pie' fundus. Flame hemorrhages may also be seen.
- Treatment is with intravenous ganciclovir or its prodrug oral valganciclovir.

Intravenous pentamidine and oral co-trimoxazole are used to treat *Pneumocystis jirovecii* (formely known as *Pneumocystis carinii*.

Oral pyrimethamine and sulfadiazine, plus folinic acid is used to treat toxoplasmosis.

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A 44-year-old woman presents to the emergency department with cough, haemoptysis, and episodes of epistaxis. Additionally, she reports a two week history of fatigue, fever and muscle aches.

On examination, she appears icteric. Coarse crackles are heard on her chest and she has right upper quadrant tenderness. There is tenderness in both her calves and in her lower back.

She is usually fit and well, working as a vet in a nearby town. She is a non-smoker and consumes around 20 units of alcohol a week. At home, she has a pet cat.

Which of the following is most appropriate for this patient?

A. Advise her to isolate for the course of her infection as she is at risk of transmission to others
B. Advise serology testing is most likely to confirm the diagnosis
C. After recovery she should be offered a vaccine as she has occupational risk
D. Offer a short course of oral ciprofloxacin
E. She should be warned that her cat was likely the source of her infection

B. Advise serology testing is most likely to confirm the diagnosis

Serology is the most commonly used test for the diagnosis of leptospirosis, however, antibodies may not be present until after 7 days

This patient has occupational and social history that increase the risk of zoonotic infections. She has presented with a febrile illness with components which include liver involvement and a coagulopathy. Together with myalgia classically affecting the calves and lower back this is suggestive of leptospirosis.

The diagnosis is most commonly made by serological testing for specific antibodies. It is difficult to isolate the organism by culture. The greatest diagnostic yield for culture is if it is done in the first 10 days of the illness. After this point, the organisms are only intermittently present in the blood. This patient has had symptoms for at least 2 weeks and therefore culture pick-up rates are now even lower.

An infected individual will shed leptospires in their urine for the period of the illness and for a time afterwards. Hence there is in theory a transmittable route to others but only in a specific way. Respiratory droplet transmission does not occur and so normal social interactions are safe.

There is no vaccine against leptospirosis available. Therefore, hand hygiene is important in those with occupational risk.

Most cases of leptospirosis are mild and self-resolve without pharmacological management. Where antibiotics are employed, doxycycline or azithromycin are often used. Amoxicillin is also effective.

Leptospirosis is classically associated with rats, which are the most common source of transmission, but other sources include dogs, cattle and pigs.

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A 24-year-old female presented to clinic with symptoms of dysuria and new vaginal discharge. She has been having unprotected sexual intercourse with a new male partner over the past eight weeks. Her nuclear acid amplification (NAAT) test was positive for *Chlamydia* and negative for gonorrhoea infection. She is currently menstruating.

What is the first-line treatment of choice for this infection?

A. Amoxicillin
B. Azithromycin
C. Ceftriaxone
D. Doxycycline
E. Erythromycin

D. Doxycycline

Chlamydia - treat with doxycycline

This patient has a *Chlamydia* infection. We know she is not pregnant as she is currently menstruating. The first-line treatment for *Chlamydia* infection is a 7-day course of doxycycline. Azithromycin was previously preferred as a first-line treatment as it can be given as a one-off dose, which increases compliance. However, recent evidence has raised concern for co-infection with *Chlamydia* and *Mycoplasma genitalium*, with growing resistance to macrolides. Therefore, doxycycline is now recommended as first-line treatment in non-pregnant patients.

Amoxicillin may be considered as a treatment for *Chlamydia* in pregnant patients. However, is not the first-line in this case.

Azithromycin is no longer considered first-line due to growing concerns around resistance. Azithromycin is also considered as a treatment option in pregnant patients with chlamydia infection.

Ceftriaxone is the first-line treatment for patients with *Gonorrhoea*, rather than *Chlamydia* infection. This patient was negative for *Gonorrhoea* infection on NAAT.

Erythromycin may also be considered as a treatment for *Chlamydia* in pregnant patients. However, is not the first-line in this case.

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A 23-year-old man presents to a sexual health clinic with a five-day history of dysuria, purulent urethral discharge and urinary frequency. He reports having a long-term partner. He has never previously had a sexual health screening. The patient has no known allergies.

A urine sample is sent for Nucleic Acid Amplification Testing (NAAT) which is negative for chlamydia and gonorrhoea. A urethral swab shows leucocytes. Blood-borne virus testing is negative and cultures of rectal and oral swabs are negative.

What is the most appropriate treatment?

A. Antibiotics not indicated
B. Intramuscular ceftriaxone
C. Oral aciclovir
D. Oral doxycycline
E. Oral metronidazole

D. Oral doxycycline

Urethritis in a male, negative for Gonorrhoea and Chlamydia →?Mycoplasma genitalium

Oral doxycycline is the correct answer. This patient has non-gonococcal urethritis (NGU) which can be caused, by organisms including *Chlamydia trachomatis* and *Mycoplasma genitalium*. NICE recommends doxycycline 100 mg twice a day for 7 days as first-line treatment. If a *Mycoplasma genitalium* NAAT is positive, BASHH recommends oral doxycycline 100mg bd for seven days followed by azithromycin 1g orally as a single dose then 500mg orally once daily for 2 days, where the organism is known to be macrolide-sensitive or where resistance status is unknown.

Antibiotics not indicated is the incorrect answer. Although NAAT testing for chlamydia and gonorrhoea is negative, this patient is symptomatic and requires treatment for urethritis.

Intramuscular ceftriaxone is the incorrect answer. This is the treatment for gonorrhoea and the patient has had a negative gonorrhoea NAAT.

Oral aciclovir is the incorrect answer. This is used to treat herpes simplex virus infection. There is no genital ulceration to suggest herpes infection.

Oral metronidazole is the incorrect answer. This would be the appropriate treatment if *Trichomonas vaginalis* is isolated, which is a cause of NGU. However, metronidazole would not be given first-line for a non-specified NGU.

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A 35-year-old woman presents to the acute medical unit with a 1-week history of worsening headache and fever. Her past medical history is unremarkable. On examination, you note nuchal rigidity and photophobia. A neurological examination is entirely normal. Observations are as follows: heart rate 115 beats per minute, blood pressure 125/75 mmHg, respiratory rate 16 breaths per minute, SpO2 96% (on air), and temperature 38.5ºC.

Blood results are as follows:

Hb	110 g/L	Male: (135-180)Female: (115 - 160)
Platelets	168 * 10 /L 9	(150 - 400)
WBC	4.2 * 10 /L 9	(4.0 - 11.0)
Na +	135 mmol/L	(135 - 145)
K +	3.6 mmol/L	(3.5 - 5.0)
Urea	2.4 mmol/L	(2.0 - 7.0)
Creatinine	52 µmol/L	(55 - 120)
CRP	78 mg/L	(< 5)
Glucose	7.8 mmol/L	(0.7-1.0)

HIV testing	HIV-1 antibody detected
HIV viral load	240,890 copies/mL

A lumbar puncture is performed:

Opening pressure	44 cm H2O	(7-18)
WBC count	250 cells/µL (90% lymphocytes)	(0-5)
Glucose	1.8 mmol/L	(0.7-1.0)
Protein	220 mg/dL	(<45)

What is the most likely diagnosis?

A. Bacterial meningitis
B. CNS lymphoma
C. Cerebral toxoplasmosis
D. Cryptococcal meningitis
E. TB meningitis

D. Cryptococcal meningitis

In cryptococcal meningitis, CSF shows high opening pressure and elevated protein. The white cell count may vary according to HIV status

It is important to note that this patient has a new diagnosis of HIV given the positive HIV-1 antibody result.

Cryptococcal meningitis is correct. Cryptococcal meningitis is the most common fungal CNS infection in patients with HIV. The lymphocytic pleocytosis, low glucose, raised protein, and markedly elevated opening pressure is characteristic of cryptococcal meningitis.

Bacterial meningitis is incorrect. The classic CSF results in bacterial meningitis is a high WBC count with a predominance of neutrophils (>90%).

CNS lymphoma is incorrect. This condition behaves as a space-occupying lesion which would usually be associated with focal neurology.

Cerebral toxoplasmosis is incorrect. Cerebral toxoplasmosis mostly presents as cerebral mass lesions with headache, confusion, fever, lethargy, seizures, cranial nerve palsies, psychomotor changes, hemiparesis and/or ataxia. The absence of neurological symptoms makes this a less likely diagnosis.

TB meningitis is incorrect. Distinguishing TB meningitis from cryptococcal meningitis based on clinical and laboratory features can be a real challenge. TB meningitis also results in a WBC pleocytosis (early neutrophils then lymphocytes), low glucose, and elevated protein. However, in this case, the markedly raised opening pressure favours the diagnosis of cryptococcal meningitis.

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A 49-year-old man presents to the emergency department with fever and dry cough for the last week. He also complains of myalgia and headaches which have been hampering his work on his farm. These headaches are variable in location, but usually worse behind his eyes. He is an exsmoker, with a 15 pack-year history. His alcohol intake is minimal and he has no significant past medical history of note.

On examination, he has coarse crackles in his right lung base. His abdomen is soft and non-tender. There is no rash. Heart sounds are normal, with no murmurs heard.

Initial investigations are done and a chest x-ray is requested.

Hb	146 g/L	Male: (135-180)Female: (115 - 160)
Platelets	452 * 10 /L 9	(150 - 400)
WBC	14.8 * 10 /L 9	(4.0 - 11.0)

Na +	138 mmol/L	(135 - 145)
K +	4.7 mmol/L	(3.5 - 5.0)
Urea	6.9 mmol/L	(2.0 - 7.0)
Creatinine	118 µmol/L	(55 - 120)
CRP	83 mg/L	(< 5)
Bilirubin	17 µmol/L	(3 - 17)
ALP	122 u/L	(30 - 100)
ALT	84 u/L	(3 - 40)
Î³GT	66 u/L	(8 - 60)
Albumin	39 g/L	(35 - 50)

What is the most appropriate treatment option?

A. Albendazole
B. Amoxicillin
C. Azithromycin
D. Co-trimoxazole
E. Doxycycline

E. Doxycycline

Farmer, fever, transaminitis ?Q fever

This patient is a farmer who has presented with evidence of pneumonia combined with a transaminitis. His occupation should make you think about potential zoonotic infections, which cause this presentation. Some examples of zoonotic infections include brucellosis, cryptosporidiosis, orf and Q fever.

Q fever is caused by *Coxiella burnetii*, a rickettsia. The source of infection is typically an abattoir (cattle/sheep). It may be inhaled from infected dust. It's features include a prodromal illness, atypical pneumonia and a transaminitis, as in this case. Treatment is most effective if given early, and doxycycline is the antibiotic of choice.

Amoxicillin may be used in cases of community-acquired pneumonia to cover typical organisms. However the transaminitis and occupation in the question point to a more atypical presentation.

Azithromycin is a potential treatment option for typhoid fever, which is more common in returning travellers from Asia. It often causes a prodromal illness and can affect the liver. In this case, there is no mention of travel and other features of typhoid such as a 'rose-spots' rash and bradycardia are absent.

Co-trimoxazole is a combination antibiotic, consisting of trimethoprim and sulfamethoxazole. It is the treatment of choice for pneumocystis pneumonia. However, this is rare in immunocompetent patients, classically occurring in those with HIV and a CD4 count below 200 cells/mm .

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Raymond is a 35-year-old man presenting with fevers. He had just returned from his overseas trip 7 days ago and he started experiencing fevers 3 days after his return. This was accompanied by joint pain along his wrists and ankles. The pain was so severe that he struggled to walk and lift objects with his hands due to the pain. Paracetamol and ibuprofen did not help the pain. He also felt generally fatigues over this time.

Raymond has recently travelled to Africa and India over the last 2 months. He states that he had gotten all of the recommended vaccinations before travelling. He notes that he had taken Malarone as his malaria prophylaxis throughout the trip. He had applied mosquito repellent during the 1st month of the trip but had forgotten to replenish this during the 2nd month of the trip.

Examination revealed swollen joints that were hot to touch but not overtly erythematous over both his wrist and ankle. An erythematous rash is also noted across the body with no clear discrete lesions identified. This was not pruritic in nature. Pyrexia was noted at 38.4ºC.

Blood tests results were as follow:

Hb	130 g/L	Male: (135-180)Female: (115 - 160)
Platelets	90 * 10 /L 9	(150 - 400)
WBC	5.0 * 10 /L 9	(4.0 - 11.0)
Neuts	3.7 * 10 /L 9	(2.0 - 7.0)
Lymphs	0.7 * 10 /L 9	(1.0 - 3.5)
Mono	0.5 * 10 /L 9	(0.2 - 0.8)
Eosin	0.1 * 10 /L 9	(0.0 - 0.4)

Na +	137 mmol/L	(135 - 145)
K +	4.0 mmol/L	(3.5 - 5.0)
Urea	6.0 mmol/L	(2.0 - 7.0)
Creatinine	110 µmol/L	(55 - 120)
Bilirubin	10 µmol/L	(3 - 17)
ALP	50 u/L	(30 - 100)
ALT	23 u/L	(3 - 40)
Î³GT	56 u/L	(8 - 60)
Albumin	36 g/L	(35 - 50)

Three thick and thin blood films were negative. \

What is the likely cause for his fevers?

A. Chikungunya fever
B. Dengue fever
C. Leptospirosis
D. Malaria
E. Yellow fever

A. Chikungunya fever

Chikungunya can present with debilitating joint pain

The history of travel to Africa and India with hints that the patient may not have engaged with adequate mosquito protection places him at risk for mosquito-borne illness. The combination of rash, fevers and significant joint pains makes chikungunya fever the most likely culprit. Its vector is the *Aedes* mosquito and is endemic within Africa and Asia. It can cause issues like lymphopenia and thrombocytopenia on blood tests. Treatment is supportive.

Dengue fever is less likely as it is not commonly associated with significant joint pains. Instead, it is usually associated with myalgic pains. It will also cause a circulatory collapse in its severe presentation, which is not common in chikungunya.

Yellow fever is also predominantly endemic in Africa. It is less likely in this case given the absence of any significant liver abnormalities. Also, hepatomegaly can be picked up during examination in these patients.

Leptospirosis is predominantly spread by rodents. There is no clear risk of exposure to its vector and it is also not usually associated with joint pains. It is more commonly seen with myalgia and as the disease progresses, liver and renal involvement start to become a prominent feature.

Lastly, malaria is a common disease in both these countries. However, anaemia and jaundice would be expected. Besides, the 3x negative blood films are reassuring and useful to rule out malaria as the underlying cause.

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A previously well 28-year-old man presents with shortness of breath and abdominal discomfort. He reports a dry cough for the previous 10 days. He works full time as a management consultant. He is a non-smoker and drinks approximately 20 units of alcohol a week. He went on a stag-do to Prague with a bunch of friends 2 weeks ago. In the last few days, he has noticed a widespread skin rash which he describes as lots of pink rings around a pale centre.

Bloods on admission:

Na +	128 mmol/l
K +	3.7 mmol/l
Urea	8.2 mmol/l
Creatinine	150 µmol/l

Chest x-ray: Diffuse reticular infiltrates and small right-sided pleural effusion.

What is the most likely causative organism?

A. Staphylococcus aureus
B. Pneumocystis jirovecii
C. Mycoplasma pneumoniae
D. Streptococcus pneumoniae
E. Haemophilus influenzae

C. Mycoplasma pneumoniae

Stereotypical history of mycoplasma pneumonia: worsening flu-like symptoms and a dry cough. Erythema multiforme is noted on examination

This patient has community-acquired pneumonia. Whilst *Streptococcus pneumoniae* and *Haemophilus influenzae* are the commonest causative organisms they tend to present with a shorter history. The longer duration of symptoms and unusual features of abdominal pain, dry cough and hyponatraemia should alert you to an atypical organism of which *Mycoplasma pneumoniae* is one of the commonest. The patient also describes erythema multiforme which is seen with *Mycoplasma pneumonia*.

Mycoplasma pneumoniae occurs in epidemics every three to four years and typically affects younger people. It tends to have an insidious onset with flu-like symptoms and atypical features. Hyponatraemia is often seen. Cold agglutinins can cause an autoimmune haemolytic anaemia. Diagnosis is confined with Mycoplasma serology. Treatment is with clarithromycin or a tetracycline or fluoroquinolone.

Staphylococcal pneumonia may complicate influenza infection and is seen most frequently in the elderly and in intravenous drug users or patients with underlying disease. It can result in a cavitating pneumonia.

Pneumocystis jirovecii is seen in immunocompromised patients. It typically presents with exertional breathlessness and patients are seen to desaturate on walking. Whilst a HIV test should be considered in the diagnostic workup of this patient, it is not the most likely diagnosis in this case.

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A 45-year-old man attends the general outpatient clinic having recently returned from hiking holiday in the Highlands of Scotland. Following his return, he describes how he has been feeling a bit unwell. He states that he has been experiencing general muscle aches, fatigue and a mild headache. On examination, they have a red papule on the posterior aspect of their calf with a patch of clear skin and a further red ring. It is non-tender and there is no warmth.

Further examination is unremarkable.

What is the most appropriate initial action?

A. Discharge with reassurance and worsening advice
B. ELISA antibody test B. Burgdorferi
C. Oral doxycycline
D. PCR test
E. Skin biopsy

C. Oral doxycycline

Lyme disease can be diagnosed clinically if erythema migrans is present → give antibiotics

The best initial option is to treat them empirically for Lyme disease. They have been out walking in an endemic area for Lyme disease and have features of early disease (erythema migrans and nonspecific viral features). Early treatment for suspected Lyme disease is appropriate in people who present with erythema migrans and other features of infection. Specific investigations for Lyme disease may be appropriate if the clinical presentation is delayed.

There is no requirement for further investigations as they have features of Lyme disease so a skin biopsy, ELISA PCR test is inappropriate as these would not change the management. Similarly, as they have presented with erythema migrans and there is good reason to suspect Lyme disease, discharging with no treatment is also inappropriate.

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A 30-year-old female presented to the emergency department with a 5-day history of fever, malaise, myalgia, joint pains, and headache. She thought she was recovering but over the past 2 days, her eyes had started to turn yellow. She had no past medical history and was on no regular medications. She had recently returned from Brazil 8 days ago where she had spent 2 weeks participating in many outdoor sporting events, including a forest marathon and wild swimming in lakes. She denied having a regular partner but reported that she had unprotected sexual intercourse on her last night in Brazil. She does not remember being bitten by insects.

On examination, she looked unwell.

Her observations were as follows:

- temperature 39.1ºC
- respiratory rate 22/min
- heart rate 109bpm
- blood pressure 105/67mmHg
- SpO2 98% on room air

She had scleral icterus and mild tender hepatomegaly. A faint petechial rash could be seen on her torso, hands, and feet. Blood tests results were as follows:

Hb	101 g/L	Male: (135-180)Female: (115 - 160)
Platelets	83 * 10 /L 9	(150 - 400)
WBC	15 * 10 /L 9	(4.0 - 11.0)
Neuts	11 * 10 /L 9	(2.0 - 7.0)
Lymphs	3 * 10 /L 9	(1.0 - 3.5)
Mono	0.6 * 10 /L 9	(0.2 - 0.8)
Eosin	0.4 * 10 /L 9	(0.0 - 0.4)
Bilirubin	80 µmol/L	(3 - 17)
ALP	231 u/L	(30 - 100)
ALT	265 u/L	(3 - 40)
AST	524 u/L	(5 - 40)

HBsAg - negative

Anti-HBs - positive

Anti-HBc - positive

Which test is most likely to reveal the diagnosis?

A. Skin scrapings for Leishmania braziliensis culture and PCR
B. Serum measurement of HBeAg
C. Urine microscopy for detection of Schistosoma haematobium
D. Treponema pallidum particle agglutination (TPPA) assay
E. Leptospira interrogans serum serology

E. Leptospira interrogans serum serology

Serology is the most commonly used test for the diagnosis of leptospirosis, however, antibodies may not be present until after 7 days

Leptospirosis is caused by zoonotic spirochete bacteria, spread via contact with infected animal urine. Animals such as rodents act as reservoirs of infection, shedding the bacteria in their urine which subsequently enter mucous membranes and broken skin. Freshwater swimming and other watersports are associated with leptospirosis infection. After an incubation period of 7 - 14 days, the disease presents in a biphasic fashion: acute fever, malaise, headache, and abdominal pain followed by severe systemic upset such as renal failure, pulmonary haemorrhage, and hepatic failure. Thrombocytopenia is also seen (suggested mechanisms include poor production by bone marrow and immune-mediated platelet destruction). Serology is the most commonly used test for the diagnosis of leptospirosis, however, antibodies may not be present until after 7 days.

Leishmania brasiliensis is the unicellular parasite responsible for cutaneous and mucosal leishmaniasis. Endemic in South America, this disease presents with skin and mucosal ulcerative lesions and is spread by the sandfly. Leishmaniasis is diagnosed by detecting Leishmania parasites (or DNA) in tissue specimens. This patient had no such skin lesions and no history of insect bites.

This patient's hepatitis serology demonstrates that she has been exposed to hepatitis B (positive anti-HBc and anti-HBs) but has cleared the virus (negative HBsAg). Hepatitis B e antigen (HBeAg) is an indicator of viral replication and correlates with hepatitis B virus infectivity. This would typically be measured if the patient had active hepatitis B. As this patient is HepBsAg negative, this test is not necessary.

Schistosoma haematobium is a parasitic fluke endemic to areas of Africa. The intermediate host is the freshwater snail; transmission risk is increased by swimming in such bodies of water. Whilst acute infection can cause a transient febrile illness (Katayama syndrome) the history typically includes pruritis (swimmers itch) with eosinophilia. Schistosomiasis is primarily a chronic disease with *Schistosoma haematobium* causing urological manifestations such as cystitis or obstructive uropathy. Examination of the urine for ova is the primary method used to diagnose suspected *Schistosoma haematobium* infections.

Treponema pallidum a sexually transmitted bacterial spirochete responsible for causing syphilis.

Primary syphilis is characterised by a painless self-limiting genital ulcer (chancre) and lymphadenopathy occurring 10-90 days after sexual transmission. Secondary syphilis occurs following the haematogenous spread of the bacteria and can present with a variety of symptoms including a widespread rash, adenopathy, arthralgia, hepatitis, and alopecia. This classically presents 4-8 weeks after the presentation of the chancre. Whilst this diagnosis should be considered given the patient's sexual history, there is no mention of a genital lesion in the question stem, and the very short period between the sexual encounter (8 days prior) and the onset of her symptoms is not typical of secondary syphilis. The Treponema pallidum particle agglutination (TPPA) assay is one of many tests that may be used to detect specific antitreponemal antibodies in serum. This is usually only requested after a positive non-treponemal screening test in the diagnostic workup of syphilis.

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A 25-year-old sexually active woman presents with abnormal vaginal discharge and burning during urination. A nucleic acid amplification test (NAAT) for sexually transmitted infections is performed.

NAAT	positive for *Chlamydia trachomatis*

She has no allergies, is using oral contraceptives, and is not pregnant or breastfeeding.

What antibiotic is most appropriate in this case?

A. Azithromycin
B. Ceftriaxone
C. Doxycycline
D. Erythromycin
E. Trimethoprim

C. Doxycycline

Chlamydia - treat with doxycycline

National Institute for Health and Care Excellence (NICE) guidance states that **doxycycline** is now the first-line treatment for anyone over the age of 13 diagnosed with *Chlamydia* who is not pregnant or breastfeeding. This is therefore the correct answer.

Azithromycin is the second-line treatment recommended by NICE for treating *Chlamydia* in patients for whom doxycycline is contraindicated or not tolerated.

Ceftriaxone is used in the treatment of *Gonorrhoea* but is not recommended for treating *Chlamydia*.

Erythromycin is a third-line agent recommended by NICE for treating *Chlamydia* in patients for whom both doxycycline and azithromycin are contraindicated or not tolerated.

Trimethoprim is indicated in treating urinary tract infections in some populations however is not used in the treatment of *Chlamydia*.

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A 47-year-old male presents to the emergency department with joint pains and rash developing over 48 hours. He describes feeling weak and tired with headaches, myalgia, and lumbar back pain.

Examination reveals painful tender distal inter-phalangeal (DIP) joints and tender wrists in a symmetric distribution. He tells you the pain is worse in the morning and improves throughout the day. He has an erythematous maculopapular rash involving his torso and upper limbs with multiple insect bites. During your assessment, he develops a fever of 39.7°C. His chest is clear on auscultation. There is no palpable lymphadenopathy. He denies any nausea, vomiting, or diarrhoea.

His medical history includes asthma, obstructive sleep apnoea, and depression. He tells you he returned from a family holiday in Mexico 5 days earlier.

Blood tests:

Hb	148g/L	Male: (135-180)Female: (115 - 160)
Platelets	98* 10 /L $_9$	(150 - 400)
WBC	7.8* 10 /L $_9$	(4.0 - 11.0)
Neuts	6.8* 10 /L $_9$	(2.0 - 7.0)
Lymphs	0.2* 10 /L $_9$	(1.0 - 3.5)
Mono	0.6* 10 /L $_9$	(0.2 - 0.8)
Eosin	0.2* 10 /L $_9$	(0.0 - 0.4)

Urea	6.8mmol/L	(2.0 - 7.0)
Creatinine	128 µmol/L	(55 - 120)
CRP	24mg/L	(< 5)

What is the most likely cause of this man's presentation?

A. Dengue
B. Rheumatoid arthritis
C. Malaria
D. Chikungunya
E. Psoriatic arthritis

D. Chikungunya

Chikungunya can present with debilitating joint pain

Chikungunya can present with debilitating joint pain. A rash develops in up to 75% of patients. The most common symptoms include fevers and malaise. It can be difficult to differentiate from dengue early in the disease. Two significant differences may help distinguish between them; 1) In chikungunya, rashes appear on the torso and the arms while in dengue, they appear on the arms and the face. 2) Joint pain in chikungunya is experienced in the hands, wrists, feet, and legs, while in dengue it is experienced in the shoulders and the knees.

Dengue is an important differential diagnosis. It is a mosquito-borne tropical disease caused by the dengue virus. Symptoms include a high fever, headache, vomiting, joint pains, and a characteristic skin rash. It may be indistinguishable from chikungunya early in the disease.

Rheumatoid arthritis is a symmetric polyarthritis involving large and small joints. This is unlikely given the acute presentation, and rheumatoid arthritis is not typically associated with fevers or rash.

Malaria should be considered in any patient with fevers and non-specific symptoms travelling from an endemic area. It may present with fevers, malaise, diaphoresis, headaches, cough, arthralgia. Chikungunya is more likely in our patient as it more commonly presents with joint pains and rash.

Psoriatic arthritis is associated with small joint involvement (DIPs) and a characteristic scaly rash. The acute presentation and fevers suggest an infectious cause.

Treatment: Relief of symptoms. No specific treatment.

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A 45-year-old man is known to be HIV positive but has a long history of poor compliance with anti-retroviral medication. He presents to the Emergency Department with a 4-day history of headache and vomiting. On examination, he has neck stiffness but no rash. He has a temperature of 38.6 ºC and is confused. A CT head is unremarkable. A lumbar puncture is performed. Examination of CSF with India ink is positive.

CD4 count	60 cells per mm 3	(500 - 1500)

What additional finding from the lumbar puncture would be most in keeping with the diagnosis?

A. Normal glucose
B. Normal protein
C. Opening pressure of 28 cm H2O
D. Presence of organism-specific antibodies in the CSF
E. Raised white cell count with predominantly neutrophils

C. Opening pressure of 28 cm H2O

In cryptococcal meningitis, CSF shows high opening pressure and elevated protein. The white cell count may vary according to HIV status

This patient has cryptococcal meningitis. *Cryptococcus neoformans* is an encapsulated yeast that is found in the environment and is a common cause of meningitis in patients with HIV with a CD4 count of less than 100. Examination of the CSF with India ink is positive in 70-90% of cases of HIVrelated cryptococcal meningitis.

Opening pressure of 28 cm H2O is correct. All patients with suspected cryptococcal meningitis should have manometry performed as intracranial pressure is often significantly raised. Patients can require serial lumbar punctures to lower intracranial pressure, or may even require neurosurgical devices such as extra-ventricular drains or ventriculoperitoneal shunts to manage their intracranial pressure.

Normal glucose is incorrect. CSF glucose is generally low in cryptococcal meningitis, although it can sometimes be normal.

Normal protein is incorrect. CSF protein is usually elevated in cryptococcal meningitis.

Presence of organism-specific antibodies in the CSF is incorrect. Antibodies to *Cryptococcus neoformans* are not used in diagnosis, either in the blood or CSF. Cryptococcal antigen (CrAg), however, is a useful test and can be carried out on both blood and CSF. A positive CSF CrAg has a 98% sensitivity for cryptococcal meningitis. High CSF CrAg is a poor prognostic marker.

Raised white cell count with predominantly neutrophils is incorrect. The white cell count is usually elevated, but the predominant cell type would be lymphocytes. Patients with HIV may not have an elevated CSF white cell count, and this is a poor prognostic marker.

Question 274 of 277

A 27-year-old male attends the emergency department with a 5-day history of fevers, myalgia, and headaches. He is a non-smoker and has no previous medical history.

On examination he appears dyspnoeic, his oxygen saturations are 92% with a respiratory rate of 24/minute. Heart sounds are normal and the abdomen is soft,non-tender with no organomegaly.

A chest x-ray reveals subtle bilateral interstitial infiltrates.

Blood tests:

Hb 153g/L Male: (135-180)

Female: (115 - 160)

Platelets 380* 10 /L (150 - 400)

WBC 15.1* 10 /L (4.0 - 11.0)

Na 138mmol/L (135 - 145)

K 4.1mmol/L (3.5 - 5.0)

Urea 6.7mmol/L (2.0 - 7.0)

Creatinine 96 µmol/L (55 - 120)

Bilirubin 12 µmol/L (3 - 17)

ALP 2721u/L (30 - 100)

ALT 354u/L (3 - 40)

Î³GT 468u/L (8 - 60)

Albumin 37g/L (35 - 50)

He works on a dairy farm with his father, who was recently treated in hospital for pneumonia. His father tested negative for *COVID19* while in hospital. He tells you he has had limited social contacts in the last number of weeks to protect his ageing father from COVID19.

Which of the following is the most likely cause for his presentation?

A. Q fever
B. Mycoplasma pneumonia
C. COVID19
D. Brucellosis
E. Psittacosis

A. Q fever

Farmer, fever, transaminitis ?Q fever

Q fever is the correct answer. Q fever *(Coxiella Burnetti)* is a zoonotic infection most commonly found in farmers and abattoir workers. It presents as a non-specific flu-like illness with fevers, headaches, fatigue, and myalgia. Q fever may present as pneumonia and is commonly associated with transaminitis. Occasionally it may present with endocarditis or other localized infections such as bone and joint infections or vascular infections (infected aneurysm). Q fever should be suspected in patients who present with symptoms and have risk factors (working closely with animals, exposure to infected dust, lab work).

Mycoplasma pneumonia may cause a similar presentation. Mycoplasma pneumonia is characterized by a non-specific flu-like illness with patchy infiltrates on chest x-ray, However, mycoplasma pneumonia is rarely associated with significant transaminitis and an alternative diagnosis should be considered.

COVID19 is less likely as he denies any potential exposure.

Brucellosis is incorrect. Brucellosis is associated with dairy farming and may have a similar presentation to q fever. However, brucellosis is more frequently associated with international travel. A minority of cases are transmitted within the UK and Ireland.

Psittacosis is typically associated with transmission from infected birds ('Parrot fever'). Transmission from dairy cattle has been reported but this is a much less likely occurrence. This patient does not disclose any contact with infected birds.

Question 275 of 277

A 45-year-old man presents with a 5-day history of fever and headache, followed by the onset of rash and drowsiness on the day of presentation. He has been working as a counsellor in a refugee camp in Ethiopia and returned to the UK 7 days ago. The camp is over capacity and the living conditions are very crowded. The facilities in the camp for personal hygiene and washing clothes are severely limited for both volunteers and residents. There are reports that many people in the camp have also become unwell.

On examination, he is mildly confused and drowsy with a Glasgow Coma Score of 13 (eyes 3, voice 4, motor 6.) He is visibly jaundiced with a palpable liver edge. He has a maculopapular rash on his trunk. A chest x-ray reveals bilateral patchy pulmonary infiltrates.

What antibiotic would be the first line in the management of this patient?

A. Benzylpenicillin
B. Ciprofloxacin
C. Doxycycline
D. Metronidazole
E. Rifampicin

C. Doxycycline

Doxycycline is typically used in the management of typhus

This patient has epidemic typhus, which is caused by *Rickettsia prowazekii*, which is a small bacterium transmitted by the bites of the human body louse. Epidemic typhus mainly occurs in outbreaks in conditions of severe upheaval and overcrowding, such as in conflict situations or after natural disasters. It is the most severe infection of the typhus group and has a mortality of 20-50% if untreated. It presents with abrupt onset of headache and fever, followed by the development of rash at day 5, and is commonly complicated by neurological symptoms or multi-organ failure.

The first-line treatment for all rickettsial infections is **doxycycline** . This includes infections in both the spotted fever group and the typhus group. Other management in the outbreak setting would include doxycycline prophylaxis for healthcare workers and mass delousing.

Benzylpenicillin is incorrect. Beta-lactam-based antibiotics are not effective against rickettsial infections. Benzylpenicillin can be used for bacterial meningitis, but this diagnosis is less likely in this patient who also has liver and respiratory involvement.

Ciprofloxacin is incorrect. Ciprofloxacin is not routinely used for the treatment of typhus. It could be considered for treating severe cholera, but this is unlikely in this patient given the lack of diarrhoea. (Doxycycline can also be used to treat cholera in addition to typhus).

Metronidazole is incorrect. Metronidazole is active against anaerobes but does not have any activity against rickettsia.

Rifampicin is incorrect. Rifampicin has some activity against rickettsia, but outcomes are significantly worse than with doxycycline so it is therefore not used in treatment.

Question 76 of 277

A 35-year-old farmer presents to the Emergency Department with a one week history of lethargy and subjective pyrexia. Over the past 24 hours he has developed right sided pleuritic chest pain which brought him to hospital.

He denies any past medical history and had been systemically well prior to this. He denies any recent overseas travel.

Examination reveals right sided coarse crackles in the lungs, there is no abnormality heard in the left side of the lung.

His observations revealed heart rate 105 beats per minute, oxygen saturation 96% in room air, temperature 38.1ºC, respiratory rate 18 breaths per minute, blood pressure 145/95 mmHg.

An ECG was performed which revealed sinus tachycardia and his chest radiograph revealed right sided consolidation.

His blood results taken on admission were as follows:

Hb	140 g/L	Male: (135-180)Female: (115 - 160)
Platelets	500 * 10 /L 9	(150 - 400)
WBC	16 * 10 /L 9	(4.0 - 11.0)
Na +	140 mmol/L	(135 - 145)
K +	4 mmol/L	(3.5 - 5.0)
Urea	6 mmol/L	(2.0 - 7.0)
Creatinine	110 µmol/L	(55 - 120)
CRP	159 mg/L	(< 5)

Bilirubin	16 µmol/L	(3 - 17)
ALP	78 u/L	(30 - 100)
ALT	80 u/L	(3 - 40)
Î³GT	55 u/L	(8 - 60)
Albumin	40 g/L	(35 - 50)

What is the most likely diagnosis?

A. Mycoplasma pneumoniae
B. Influenza
C. Legionnaires disease
D. Pulmonary embolism
E. Q fever

E. Q fever

Farmer, fever, transaminitis ?Q fever

This patient has Q fever. His possible exposure to sheep given that he is a farmer, pneumonia and mild transaminitis point towards Q fever as a diagnosis.

Influenza is a possibility as it may present with pyrexia and tachycardia along with respiratory symptoms, the clue in this history if that the patient is a farmer, thereby making Q fever more likely.

Legionnaires disease is usually spread through infected air conditioning units in aeroplanes or hotels. This patient has no history of travel.

Mycoplasma would classically present with hyponatraemia in the context of pneumonia, it is not usually caused by exposure to sheep or cattle. which this patients has most likely been exposed to.

A pulmonary embolism could be a differential with pleuritic chest pain and tachycardia, however the likely exposure to sheep, high temperature and consolidation on the chest radiograph makes Q fever more likely.

Question 42 of 277

A returning traveller presents to the emergency department with a 10 day history of fever, cough and abdominal pain. He has spent the last 2 weeks in Jakarta, Indonesia. His vital signs are: temperature: 40.1ºC, heart rate 85 beats/minute, blood pressure 120/80 mmHg

On examination his spleen is enlarged and there is a rose spot rash over the chest. Blood culture grows salmonella typhi and the on-call doctor diagnoses enteric fever (typhoid).

If left untreated, what is the most important and serious complication that can occur within the following 2 weeks?

A. Chronic carriage within gallbladder
B. Bowel perforation and haemorrhage
C. Splenic infarction and rupture
D. Acute liver failure
E. Bacterial meningitis

B. Bowel perforation and haemorrhage

The most serious and frequent complications of Typhoid are bowel perforation and haemorrhage. This is caused by bacterial destruction of the Peyer's patches in the small intestine and classically occurs late in the second week of illness or early in the third week. Other complications include myocarditis and endocarditis.

Chronic bacterial carriage in the gall bladder does occur, but this is not the most serious complication. It renders the patient chronically infective and capable of transmitting the disease. Acute liver failure does not occur, but mild jaundice may result from cholecystitis or hepatitis. Bacterial meningitis is a very rare complication not often reported.

passmedicine PART 2

ONCOLOGY

2024

A 20-year-old asymptomatic woman is referred to the genetics clinic. Her grandmother died of breast cancer aged 54 and her mother and older sister have both recently been diagnosed with the disease.

Breast examination does not reveal any lumps and her recent ultrasound of the breasts did not reveal any suspicious lesions.

What should she be told about her breast cancer risk?

- ○ Her chance of having a BRCA1 mutation is 55-65%
- ○ Her chance of having a BRCA2 mutation is 55-65%
- ○ If BRCA1 positive, she has a 55-65% chance of developing breast cancer
- ○ If BRCA2 positive, she has a 55-65% chance of developing breast cancer
- ○ Given her family history, she has a 55-65% chance of developing breast cancer

Submit answer

Reference ranges

Score: **0%**

1 -

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A 20-year-old asymptomatic woman is referred to the genetics clinic. Her grandmother died of breast cancer aged 54 and her mother and older sister have both recently been diagnosed with the disease.

Breast examination does not reveal any lumps and her recent ultrasound of the breasts did not reveal any suspicious lesions.

What should she be told about her breast cancer risk?

Her chance of having a BRCA1 mutation is 55-65%	7%
Her chance of having a BRCA2 mutation is 55-65%	3%
🟩 If BRCA1 positive, she has a 55-65% chance of developing breast cancer	49%
If BRCA2 positive, she has a 55-65% chance of developing breast cancer	15%
🟥 Given her family history, she has a 55-65% chance of developing breast cancer	25%

The BRCA1 and BRCA2 genes are tumour suppressor genes involved in DNA repair. Familial mutations in these genes are associated with an increased risk of breast and ovarian cancer. Carriers tend to develop these cancers at a younger age as they have an inherited germline mutation of one copy of the gene so only require a 'second hit' of damage to the second copy to inactivate it. BRCA mutations account for 20-25% of hereditary breast cancers and 5-10% of all breast cancer cases, They also account for 15% of all ovarian cancer.

If a woman with at least a 10% risk of breast or ovarian cancer (approximated from family history) will be offered genetic screening. This requires genetic counselling and living relative whose DNA can also be tested for the mutation, otherwise, the chance of identifying the mutation is too low. This has important ethical considerations with regard to consent.

The percentage risk of developing breast cancer by age 70 is 55-65% for BRCA1 and 45% for BRCA2 mutations.

The percentage risk of developing ovarian cancer by age 70 is 40% for BRCA1 and 15% for BRCA2 mutations.

Because of the high risks involved, women with a positive test result may choose to have enhanced screening for these cancers, prophylactic surgery (bilateral mastectomy and/or bilateral salpingo-oophorectomy) or chemoprevention with hormonal drugs.

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National Cancer Institute. BRCA1 and BRCA2. Cancer Risk and Genetic Testing. Updated 1 April 2015. Available at: http://www.cancer.gov/about-cancer/causes-prevention/genetics/brca-fact-sheet Accessed 7
July 2015

National Institute for Health and Care Excellence. Information for the Public: Familial Breast Cancer. Updated June 2013. Available at: http://publications.nice.org.uk/familial-breast-cancer-breast-cancer-in-the-family-ifp164/genetic-counselling-and-genetic-testing Accessed 7 July 2015

Discuss (2) Improve

Next question ›

Breast cancer: risk factors ★

Predisposing factors

- BRCA1, BRCA2 genes - 40% lifetime risk of breast/ovarian cancer
- 1st degree relative premenopausal relative with breast cancer (e.g. mother)
- nulliparity, 1st pregnancy > 30 yrs (twice risk of women having 1st child < 25 yrs)
- early menarche, late menopause
- combined hormone replacement therapy (relative risk increase * 1.023/year of use), combined oral contraceptive use
- past breast cancer
- not breastfeeding
- ionising radiation
- p53 gene mutations
- obesity
- previous surgery for benign disease (?more follow-up, scar hides lump)

+ Q 123

Next question ›

B I A

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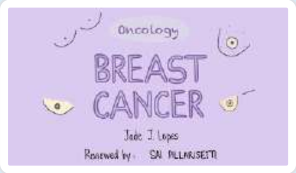
Oncology
BREAST CANCER
Jade J. Lopes
Reviewed by: SAI PILLARISETTI

Breast cancer

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BREAST CANCER
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42	✗
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You are working as a registrar on the palliative care ward. A 58-year-old lady is admitted for symptom control. She has a past medical history of metastatic pancreatic cancer and diabetic nephropathy. She has been suffering from severe abdominal pain which has been unresponsive to titration of morphine, gabapentin and duloxetine.

Blood results are as follows:

Hb	75 g/l	Na^+	138 mmol/l
Platelets	$224 * 10^9/l$	K^+	3.8 mmol/l
WBC	$14.9 * 10^9/l$	Urea	18.6 mmol/l
Neuts	$12.9 * 10^9/l$	Creatinine	288 µmol/l
eGFR	22 mL/min		

You decide to start an opioid which acts as a neuropathic agent by NMDA antagonism. What analgesic is most appropriate?

○ Methadone

○ Oxycodone

○ Buprenorphine

○ Paracetamol

○ Venlafaxine

Submit answer

Reference ranges

Score: **0%**

1 -

2	-

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Question 2 of 43 ✗

You are working as a registrar on the palliative care ward. A 58-year-old lady is admitted for symptom control. She has a past medical history of metastatic pancreatic cancer and diabetic nephropathy. She has been suffering from severe abdominal pain which has been unresponsive to titration of morphine, gabapentin and duloxetine.

Blood results are as follows:

Hb	75 g/l	Na^+	138 mmol/l
Platelets	224 * 10^9/l	K^+	3.8 mmol/l
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Neuts	12.9 * 10^9/l	Creatinine	288 µmol/l
eGFR	22 mL/min		

You decide to start an opioid which acts as a neuropathic agent by NMDA antagonism. What analgesic is most appropriate?

Methadone ✓	33%
Oxycodone	15%
Buprenorphine	40%
Paracetamol	0%
Venlafaxine ✗	12%

Methadone can be used as a third line opioid for patients with complex pain that is poorly responsive to other opioids and adjuvants

Important for me Less important

Methadone can be used as a third line opioid for patients with complex pain that is poorly responsive to other opioids and adjuvants. The patients pain has not responded to titration of morphine, combined with two neuropathic adjuvants. Methadone should therefore be considered.

The patients eGFR is les than 30 which also strengthens the decision to start methadone. Opioids which are safe in CKD 4 and 5 include fentanyl, buprenorphine and methadone.

Paracetamol and venlafaxine are not opioids.

👍 👎 💬 Discuss (8) Improve

Next question >

Palliative care prescribing: pain ★

NICE guidelines

In 2012 NICE published guidelines on the use of opioids in palliative care. Selected points are listed below. Please see the link for more details.

Starting treatment
- when starting treatment, offer patients with advanced and progressive disease regular oral modified-release (MR) or oral immediate-release morphine (depending on patient preference), with oral immediate-release morphine for breakthrough pain
- if no comorbidities use 20-30mg of MR a day with 5mg morphine for breakthrough pain. For example, 15mg modified-release morphine tablets twice a day with 5mg of oral morphine solution as required
- oral modified-release morphine should be used in preference to transdermal patches
- laxatives should be prescribed for all patients initiating strong opioids
- patients should be advised that nausea is often transient. If it persists then an antiemetic should be offered
- drowsiness is usually transient - if it does not settle then adjustment of the dose should be considered

SIGN guidelines

SIGN issued guidance on the control of pain in adults with cancer in 2008. Selected points
- the breakthrough dose of morphine is one-sixth the daily dose of morphine
- all patients who receive opioids should be prescribed a laxative
- opioids should be used with caution in patients with chronic kidney disease
  - oxycodone is preferred to morphine in palliative patients with mild-moderate renal impairment
  - if renal impairment is more severe, alfentanil, buprenorphine and fentanyl are preferred
- metastatic bone pain may respond to strong opioids, bisphosphonates or radiotherapy. The assertion that NSAIDs are particularly effective for metastatic bone pain is not supported by studies. Strong opioids have the lowest number needed to treat for relieving the pain and can provide quick relief, in contrast to radiotherapy and bisphosphonates*. All patients, however,

should be considered for referral to a clinical oncologist for consideration of further treatments such as radiotherapy

Other points

When increasing the dose of opioids the next dose should be increased by 30-50%.

In addition to strong opioids, bisphosphonates and radiotherapy, denosumab may be used to treat metastatic bone pain.

Opioid side-effects

Usually transient	**Usually persistent**
Nausea Drowsiness	Constipation

Conversion between opioids

From	**To**	**Conversion factor**
Oral codeine	Oral morphine	Divide by 10
Oral tramadol	Oral morphine	Divide by 10**

Oxycodone generally causes less sedation, vomiting and pruritis than morphine but more constipation.

From	**To**	**Conversion factor**
Oral morphine	Oral oxycodone	Divide by 1.5-2***

The current BNF gives the following conversion factors for transdermal perparations
- a transdermal fentanyl 12 microgram patch equates to approximately 30 mg oral morphine daily
- a transdermal buprenorphine 10 microgram patch equates to approximately 24 mg oral morphine daily.

From	To	Conversion factor
Oral morphine	Subcutaneous morphine	Divide by 2
Oral morphine	Subcutaneous diamorphine	Divide by 3
Oral oxycodone	Subcutaneous diamorphine	Divide by 1.5

*BMJ 2015;350:h315 Cancer induced bone pain

**this has previously been stated as 5 but the current version of the BNF states a conversion of 10

***historically a conversion factor of 2 has been used (i.e. oral oxycodone is twice as strong as oral morphine). The current BNF however uses a conversion rate of 1.5

+ Q 123

Next question >

Textbooks

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Links

NICE 👍 3 👎 1

2012 Opioids in palliative care guidelines

BNF 👍 3 👎 3

Prescribing opioids in palliative care

Suggest link

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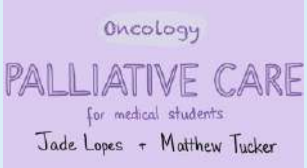
Media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for medical students
Jade Lopes + Matthew Tucker

Complete Guide to Palliative Care for Medical Students

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HEART ATTACK
REFERRED PAIN
ACUTE PAIN
CHRONIC PAIN
PAIN
BRAIN
SPINAL CORD
C FIBRES
A-DELTA FIBRES
RECEPTORS
PERIPHERAL NERVES

Understanding The Causes of Pain and Pain Management

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43	✗

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Question 3 of 43

You are asked to provide advice on a patient who has metastatic renal cell carcinoma with pulmonary and bone metastases. They are being managed in their own home with community palliative care input, but their swallow has started to deteriorate. It is now felt that they are approaching the terminal phase of their illness and a decision has been made to start a syringe driver. They report good pain control with their current analgesia regime.

Their current analgesia regime is 60mg oxycodone modified release capsules twice a day with oxycodone instant release, taken as required. The oxycodone modified release is converted into the equivalent subcutaneous dose for an oxycodone syringe driver.

What would be the most appropriate dose of breakthrough analgesia to prescribe based on the current syringe driver dose?

- ○ Oxycodone I/R oral solution 20mg
- ○ Oxycodone I/R oral solution 10mg
- ○ Oxycodone subcutaneous injection 10mg
- ○ Morphine sulphate for injection 10mg
- ○ Morphine sulphate injection 20mg

Submit answer

Reference ranges

Score: **0%**

1 -
2 -
3 -

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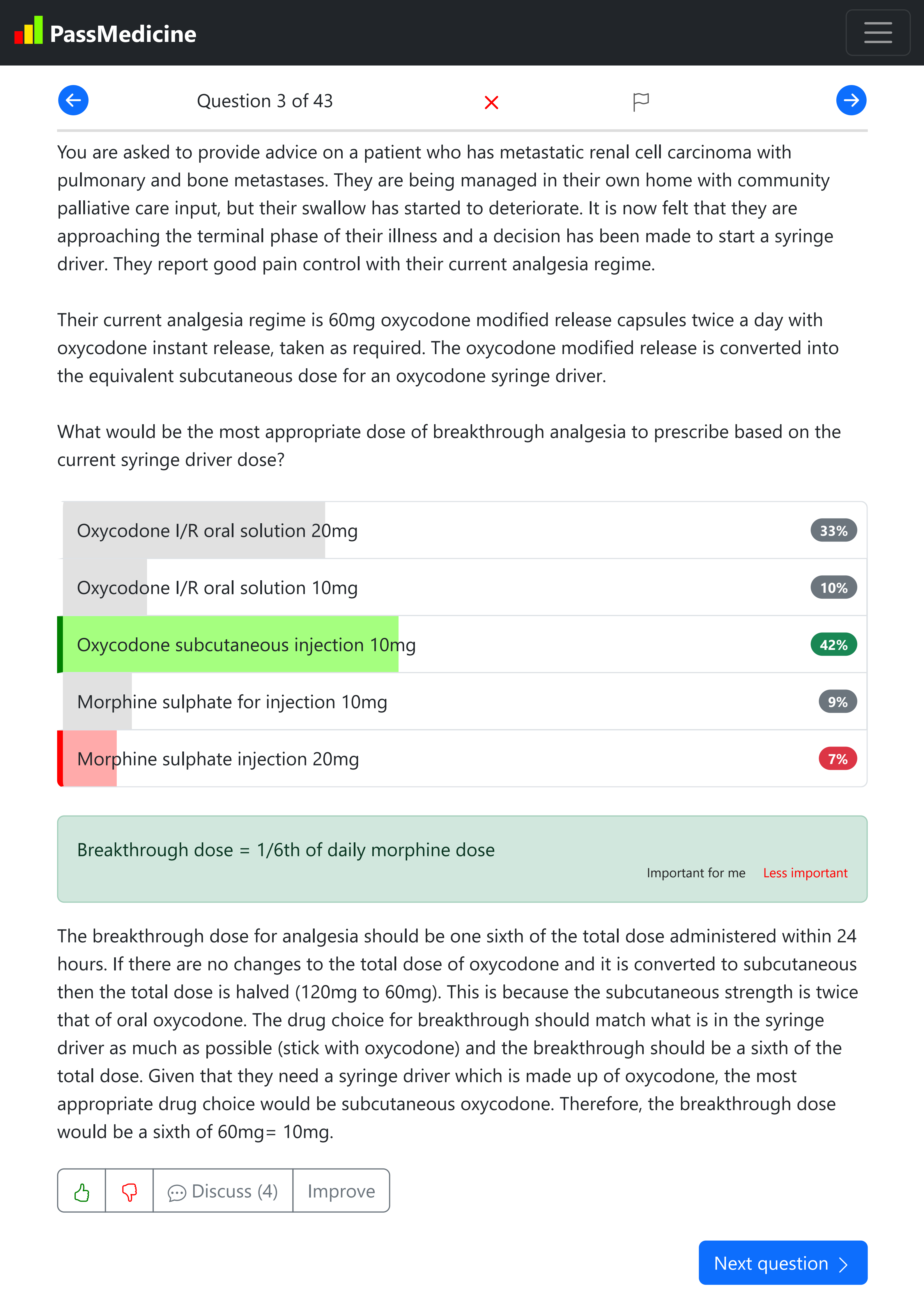

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Oxycodone I/R oral solution 20mg	33%
Oxycodone I/R oral solution 10mg	10%
Oxycodone subcutaneous injection 10mg	42%
Morphine sulphate for injection 10mg	9%
Morphine sulphate injection 20mg	7%

Breakthrough dose = 1/6th of daily morphine dose

Important for me   Less important

The breakthrough dose for analgesia should be one sixth of the total dose administered within 24 hours. If there are no changes to the total dose of oxycodone and it is converted to subcutaneous then the total dose is halved (120mg to 60mg). This is because the subcutaneous strength is twice that of oral oxycodone. The drug choice for breakthrough should match what is in the syringe driver as much as possible (stick with oxycodone) and the breakthrough should be a sixth of the total dose. Given that they need a syringe driver which is made up of oxycodone, the most appropriate drug choice would be subcutaneous oxycodone. Therefore, the breakthrough dose would be a sixth of 60mg= 10mg.

Palliative care prescribing: pain ★

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From	**To**	**Conversion factor**
Oral morphine	Oral oxycodone	Divide by 1.5-2***

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From	**To**	**Conversion factor**
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Oral morphine	Subcutaneous diamorphine	Divide by 3
Oral oxycodone	Subcutaneous diamorphine	Divide by 1.5

*BMJ 2015;350:h315 Cancer induced bone pain

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***historically a conversion factor of 2 has been used (i.e. oral oxycodone is twice as strong as oral morphine). The current BNF however uses a conversion rate of 1.5

123

Next question

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NICE 👍 3 👎 1

2012 Opioids in palliative care guidelines

BNF 👍 3 👎 3

Prescribing opioids in palliative care

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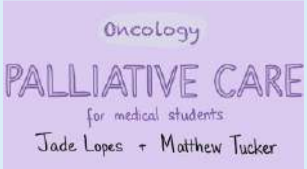
Oncology
PALLIATIVE CARE
for medical students
Jade Lopes + Matthew Tucker

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HEART ATTACK
REFERRED PAIN
ACUTE PAIN
CHRONIC PAIN
PAIN
BRAIN
SPINAL CORD
C FIBRES
A-DELTA FIBRES
RECEPTORS
PERIPHERAL NERVES

Understanding The Causes of Pain and Pain Management

Zero To Finals - YouTube 👍 1 👎 1

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You are looking after a 65-year-old man with a background of high-grade lymphoma who she is currently receiving R-CHOP chemotherapy for. She developed neutropaenic sepsis yesterday for which she was started on piperacillin/tazobactam, and gentamicin. She has been persistently pyrexial for the past 24 hours.

She has a background of complex pain for which she uses a 25micrograms/hour fentanyl patch. You are asked to review her due to confusion and visual hallucinations. On examination, she has pinpoint pupils, GCS 14 (M6, V4, E4), and you note myoclonic jerks.

Na^+	136 mmol/L	(135 - 145)
K^+	3.8 mmol/L	(3.5 - 5.0)
Bicarbonate	19 mmol/L	(22 - 29)
Urea	7.2 mmol/L	(2.0 - 7.0)
Creatinine	130 µmol/L	(55 - 120)

Bilirubin	14 µmol/L	(3 - 17)
ALP	95 u/L	(30 - 100)
ALT	82 u/L	(3 - 40)
Î³GT	86 u/L	(8 - 60)
Albumin	35 g/L	(35 - 50)

You suspect opioid toxicity. What is the most likely explanation for this?

○ Acidosis

○ Fentanyl and piperacillin/tazobactam drug interaction

○ Increased fentanyl absorption due to pyrexia

○ Liver dysfunction

○ Renal dysfunction

Submit answer

Reference ranges

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Question 4 of 43 ✗

You are looking after a 65-year-old man with a background of high-grade lymphoma who she is currently receiving R-CHOP chemotherapy for. She developed neutropaenic sepsis yesterday for which she was started on piperacillin/tazobactam, and gentamicin. She has been persistently pyrexial for the past 24 hours.

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ALT	82 u/L	(3 - 40)
Î³GT	86 u/L	(8 - 60)
Albumin	35 g/L	(35 - 50)

You suspect opioid toxicity. What is the most likely explanation for this?

Acidosis	6%
Fentanyl and piperacillin/tazobactam drug interaction	11%
Increased fentanyl absorption due to pyrexia	56%
Liver dysfunction	12%
Renal dysfunction	15%

Transdermal fentanyl absorption is increased by heat (e.g. hot water bottle) or pyrexia, potentially leading to opioid toxicity

Important for me Less important

Transdermal fentanyl absorption is increased by heat or pyrexia (which results in vasodilation and increased blood flow), potentially leading to opioid toxicity. This is therefore the correct option.

Acidosis is incorrect. Acidosis is not known to exacerbate opioid toxicity.

Renal dysfunction is incorrect. Fentanyl is hepatically metabolised by CYP450 3A4 with no active metabolites. Therefore it is the opioid of choice in renal failure.

Liver dysfunction is incorrect. Although the patient has a mild transaminitis, their synthetic liver function is normal, making it an unlikely contributing factor.

Piperacillin/tazobactam is incorrect. Fentanyl has no known interactions with piperacillin/tazobactam.

👍 👎 Discuss (4) Improve

Next question >

Palliative care prescribing: pain ★

NICE guidelines

In 2012 NICE published guidelines on the use of opioids in palliative care. Selected points are listed below. Please see the link for more details.

Starting treatment
- when starting treatment, offer patients with advanced and progressive disease regular oral modified-release (MR) or oral immediate-release morphine (depending on patient preference), with oral immediate-release morphine for breakthrough pain
- if no comorbidities use 20-30mg of MR a day with 5mg morphine for breakthrough pain. For example, 15mg modified-release morphine tablets twice a day with 5mg of oral morphine solution as required
- oral modified-release morphine should be used in preference to transdermal patches
- laxatives should be prescribed for all patients initiating strong opioids
- patients should be advised that nausea is often transient. If it persists then an antiemetic should be offered
- drowsiness is usually transient - if it does not settle then adjustment of the dose should be considered

SIGN guidelines

SIGN issued guidance on the control of pain in adults with cancer in 2008. Selected points

- the breakthrough dose of morphine is one-sixth the daily dose of morphine
- all patients who receive opioids should be prescribed a laxative
- opioids should be used with caution in patients with chronic kidney disease
    - oxycodone is preferred to morphine in palliative patients with mild-moderate renal impairment
    - if renal impairment is more severe, alfentanil, buprenorphine and fentanyl are preferred
- metastatic bone pain may respond to strong opioids, bisphosphonates or radiotherapy. The assertion that NSAIDs are particularly effective for metastatic bone pain is not supported by studies. Strong opioids have the lowest number needed to treat for relieving the pain and can provide quick relief, in contrast to radiotherapy and bisphosphonates*. All patients, however, should be considered for referral to a clinical oncologist for consideration of further treatments such as radiotherapy

Other points

When increasing the dose of opioids the next dose should be increased by 30-50%.

In addition to strong opioids, bisphosphonates and radiotherapy, denosumab may be used to treat metastatic bone pain.

Opioid side-effects

Usually transient	Usually persistent
Nausea Drowsiness	Constipation

Conversion between opioids

From	To	Conversion factor
Oral codeine	Oral morphine	Divide by 10
Oral tramadol	Oral morphine	Divide by 10**

Oxycodone generally causes less sedation, vomiting and pruritis than morphine but more constipation.

From	To	Conversion factor
Oral morphine	Oral oxycodone	Divide by 1.5-2***

The current BNF gives the following conversion factors for transdermal perparations

- a transdermal fentanyl 12 microgram patch equates to approximately 30 mg oral morphine daily
- a transdermal buprenorphine 10 microgram patch equates to approximately 24 mg oral morphine daily.

From	To	Conversion factor
Oral morphine	Subcutaneous morphine	Divide by 2
Oral morphine	Subcutaneous diamorphine	Divide by 3
Oral oxycodone	Subcutaneous diamorphine	Divide by 1.5

*BMJ 2015;350:h315 Cancer induced bone pain

**this has previously been stated as 5 but the current version of the BNF states a conversion of 10

***historically a conversion factor of 2 has been used (i.e. oral oxycodone is twice as strong as oral morphine). The current BNF however uses a conversion rate of 1.5

AI + Q 123

Next question >

B I A

Textbooks

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Links

NICE 👍 3 👎 1

2012 Opioids in palliative care guidelines

BNF 👍 3 👎 3

Prescribing opioids in palliative care

Suggest link

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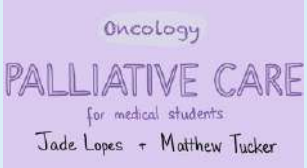
Media


Oncology
PALLIATIVE CARE
for medical students
Jade Lopes + Matthew Tucker

Complete Guide to Palliative Care for Medical Students

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HEART ATTACK
REFERRED PAIN
ACUTE PAIN
CHRONIC PAIN
PAIN
BRAIN
SPINAL CORD
C FIBRES
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RECEPTORS
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Question 5 of 43

A 63-year-old woman presents with unrelenting thoracic back pain. She finds that the pain is debilitating, especially as she helps look after her grandchildren. She has a past medical history of breast cancer diagnosed two years ago, for which she underwent a mastectomy with reconstruction. She also had postoperative radiotherapy and endocrine therapy. As she was found to be BRCA1 positive, she underwent a hysterectomy with bilateral oophorectomy as well. On examination, she is tender over the spine at T4, but neurological examination is unremarkable. What is the most appropriate imaging investigation for her back pain?

- ○ MRI of the whole spine within 7 days
- ○ MRI of the thoracic spine within 7 days
- ○ MRI of the whole spine within 24 hours
- ○ MRI of the thoracic spine within 24 hours
- ○ Bone scan of the whole body within 7 days

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MRI of the whole spine within 7 days	40%
MRI of the thoracic spine within 7 days	6%
MRI of the whole spine within 24 hours	45%
MRI of the thoracic spine within 24 hours	5%
Bone scan of the whole body within 7 days	4%

The correct answer is MRI whole spine within 7 days. This is a patient with known cancer presenting with highly suspicious back pain but no evidence of spinal cord compression. The most appropriate modality is MRI which can not only detect metastasis but also demonstrate patency or compression of the spinal cord. The whole spine should be scanned as to exclude further metastasis as 30-50% of patients have multi-level involvement by the time of presentation. The timing of the scan depends on suspicion of cord compression. For this patient, there is no evidence of neurological compromise and therefore the scan does not have to occur within 24 hours.

Discuss (4) | Improve

Next question >

Spinal metastases ★

Patients may present with spinal metastases before developing metastatic spinal cord compression. It is, therefore, important to detect these patients early before any neurological compromise develops.

Symptoms and findings

- Unrelenting lumbar back pain
- Any thoracic or cervical back pain
- Worse with sneezing, coughing or straining
- Nocturnal
- Associated with tenderness

If any neurological features are present then spinal cord compression must be suspected and acted on promptly. Without neurological features, a whole spine MRI should be completed within one week. The whole spine should be imaged as patients commonly present with multi-level disease.

Sources:
Al-Qurainy, Rasha, and Emily Collis. 'Metastatic Spinal Cord Compression: Diagnosis and Management.' Bmj (2016): I2539.

Metastatic Spinal Cord Compression in Adults: Risk Assessment, Diagnosis and Management Clinical Guideline [CG75]. National Institute of Clinical Excellence. N.p., Nov. 2008.

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Question 6 of 43

You are looking after a 68-year-old man with aggressive multiple myeloma. He has intractable vomiting which has not improved with regular subcutaneous metoclopramide.

Blood results are as follows:

Calcium	4.2 mmol/L	(2.1-2.6)

You decide to start him on a syringe driver. What would be your anti-emetic of choice?

○ Aprepitant

○ Cyclizine

○ Levomepromazine

○ Nabilone

○ Ondansetron

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You are looking after a 68-year-old man with aggressive multiple myeloma. He has intractable vomiting which has not improved with regular subcutaneous metoclopramide.

Blood results are as follows:

Calcium	4.2 mmol/L	(2.1-2.6)

You decide to start him on a syringe driver. What would be your anti-emetic of choice?

Aprepitant	9%
Cyclizine	13%
🟩 Levomepromazine	46%
Nabilone	1%
🟥 Ondansetron	31%

Metabolic causes of nausea and vomiting, for example uraemia and hypercalcaemia, cause nausea and vomiting by stimulating dopamine type 2 (D2) receptors on the chemoreceptor trigger zone. This pathway is most effectively blocked by using a central dopamine antagonist such as haloperidol

Important for me   Less important

Metabolic causes of nausea and vomiting (e.g. hypercalcaemia) are particularly sensitive to central dopamine antagonists such as **levomepromazine** making this the favoured choice.

Levomepromazine is a very broad spectrum anti-emetic that acts as an antagonist to multiple receptors including histamine type 1, muscarinic-cholinergic, dopaminergic 2, alpha-1 adrenoceptor, and 5HT-2 receptors. Thus it is a powerful anti-emetic that can be used in many scenarios.

Aprepitant is a substance P/neurokinin 1 receptor antagonist used to prevent chemotherapy-induced and postoperative nausea and vomiting. The patient in this clinical case has nausea secondary to a metabolic abnormality, and thus a dopamine antagonist is the preferred choice.

Cyclizine is a histamine H1-receptor antagonist which may have effects directly on the vestibular system and on the chemoreceptor trigger zone. It also exerts a central anticholinergic action The patient in this clinical case has nausea secondary to a metabolic abnormality, and thus a dopamine antagonist is the preferred choice.

Ondansetron is a specific 5HT3-receptor antagonist which blocks 5HT3 receptors in the gastrointestinal tract and in the CNS. The patient in this clinical case has nausea secondary to a metabolic abnormality, and thus a dopamine antagonist is the preferred choice.

Nabilone is a synthetic cannabinoid which is used to treat nausea and vomiting caused by cytotoxic chemotherapy, which is unresponsive to conventional antiemetics.

👍 👎 Discuss (3) Improve

Next question ›

Syringe drivers ★

A syringe driver should be considered in the palliative care setting when a patient is unable to take oral medication due to nausea, dysphagia, intestinal obstruction, weakness or coma. In the UK there are two main types of syringe driver:
- Graseby MS16A (blue): the delivery rate is given in mm per hour
- Graseby MS26 (green): the delivery rate is given in mm per 24 hours

The majority of drugs are compatible with water for injection although for the following drugs sodium chloride 0.9% is recommended:
- granisetron
- ketamine
- ketorolac
- octreotide
- ondansetron

Commonly used drugs
- nausea and vomiting: cyclizine, levomepromazine, haloperidol, metoclopramide
- respiratory secretions/bowel colic: hyoscine hydrobromide, hyoscine butylbromide, or glycopyrronium bromide.
- agitation/restlessness: midazolam, haloperidol, levomepromazine
- pain: diamorphine is the preferred opioid

Mixing and compatibility issues
- diamorphine is compatible with the majority of other drugs used including dexamethasone, haloperidol, hyoscine butylbromide, hyoscine hydrobromide, levomepromazine, metoclopramide, midazolam
    - cyclizine may precipitate with diamorphine when given at higher doses

- cyclizine is incompatible with a number of drugs including clonidine, dexamethasone, hyoscine butylbromide (occasional), ketamine, ketorolac, metoclopramide, midazolam, octreotide, sodium chloride 0.9%

+ Q 123

Next question >

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Syringe drivers

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Question 7 of 43

A 54-year-old-man with a history of granulomatosis with polyangiitis attends for his annual review in the rheumatology clinic. He was diagnosed at age 35 where he presented with rapidly progressive glomerulonephritis and pulmonary haemorrhage and was treated with cyclophosphamide and steroids. He required a period of haemofiltration on the intensive care unit but has remained well since.

Today, he feels well after recently quitting smoking but reports occasional blood in his urine. On examination, his abdomen is soft and non tender, there is no pallor or oedema and his chest is clear. His investigations are below and his urine appears clear.

Hb	105 g/l
Platelets	300* 10^9/l
WBC	5* 10^9/l
Urinalysis	blood 3+ protein -
Na$^+$	139 mmol/l
K$^+$	4.2 mmol/l
Urea	5.6 mmol/l
Creatinine	98 µmol/l

What is his likely diagnosis?

○ Recurrence of glomerulonephritis

○ Transitional cell carcinoma

○ Renal calculi

○ Faulty urinalysis recording

○ Rhabdomyolysis

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Urinalysis	blood 3+ protein -
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K^+	4.2 mmol/l
Urea	5.6 mmol/l
Creatinine	98 µmol/l

What is his likely diagnosis?

Recurrence of glomerulonephritis	12%
Transitional cell carcinoma	79%
Renal calculi	4%
Faulty urinalysis recording	3%
Rhabdomyolysis	2%

Use of cyclophosphamide in granulomatosis with polyangiitis is associated with increased risk of bladder cancer

Important for me Less important

The use of cyclophosphamide is linked to transitional cell carcinoma in patients with vasculitis. Given that his U&Es are normal, a recurrence of glomerulonephritis is unlikely. Renal calculi would cause loin to groin pain. Haematuria can often not be visible to the eye even when present. While rhabdomyolysis could give a positive dipstick, there are no other features to suggest this

Discuss (3) | Improve

Next question

Cyclophosphamide

Cyclophosphamide is an alkylating agent used in the management of cancer and autoimmune conditions. It works by causing cross-linking of DNA

Adverse effects

- haemorrhagic cystitis: incidence reduced by the use of hydration and mesna
- myelosuppression
- transitional cell carcinoma

Mesna

- 2-**me**rcaptoethane **s**ulfonate **Na**
- a metabolite of cyclophosphamide called acrolein is toxic to urothelium
- mesna binds to and inactivates acrolein helping to prevent haemorrhagic cystitis

123

Next question

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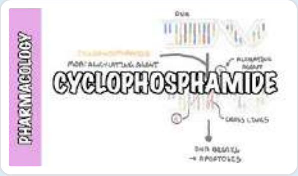
PHARMACOLOGY
CYCLOPHOSPHAMIDE

Cyclophosphamide - pharmacology, mechanism of action, adverse effects

Armando Hasudungan - YouTube 👍 2 👎 0

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A 68-year-old man is admitted to the unit with an exacerbation of severe pain around his abdomen. He describes it as a tight band like pain which is burning in character. He has a past medical history of metastatic prostate cancer. His drug history includes morphine modified release (100mg, twice daily), duloxetine (60mg, twice daily), pregabalin (200mg, three times per day). He states that the pain initially responded well to the analgesics he is on however over the past few weeks it has got progressively worse and culminated in a severe attack of pain today.

You suspect that he has got severe neuropathic pain secondary to spinal metastases. The pain is now poorly controlled with a large dose of opioids and two neuropathic agents. Your consultant decides to start him on ketamine.

What acute complication is it important to monitor for?

- ○ Bladder dysfunction
- ○ Liver dysfunction
- ○ Raised intracranial pressure
- ○ Cardiomyopathy
- ○ Pulmonary fibrosis

Submit answer

Reference ranges

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What acute complication is it important to monitor for?

Bladder dysfunction	27%
Liver dysfunction	20%
🟩 Raised intracranial pressure	42%
Cardiomyopathy	9%
🟥 Pulmonary fibrosis	2%

Ketamine can cause raised intracranial pressure. It is important to monitor for side effects such as headache, papilloedema, and vomiting

Important for me Less important

Ketamine can be used for neuropathic pain poorly responsive to titrated opioids and oral adjuvant analgesics (e.g. antidepressant and/or anticonvulsant) particularly when there is abnormal pain sensitivity (e.g. allodynia, hyperalgesia or hyperpathia). Ketamine is an NMDA receptor antagonists. Side effects to be aware of include hypertension, raised intracranial pressure and hallucinations.

Bladder and liver dysfunction can occur with long term use of ketamine. Therefore, if a patient is anticipated to have a reasonably good prognosis, alternative options should be considered to replace the ketamine, for example lidocaine infusions.

Discuss (6) Improve

Next question ›

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Syringe drivers

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Score: **9.3%**

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Question 9 of 43

A medical consult was sought from the nursing staff concerning a 68-year-old gentleman admitted with a urinary tract infection. He was diagnosed with terminal adenocarcinoma of the pancreas six months ago. He had complained of increasing severe abdominal pain for the last few weeks for which his GP commenced and then titrated morphine sulphate tablets (MST) achieving a temporary analgesic effect with each increment. By the time of admission he had been using 150mg BD and was unable to tolerate any further dose increases owing to drowsiness.

His drug history comprised of ciprofloxacin 250mg BD started four days ago for the management of urinary tract infection, paracetamol 1g QDS, tramadol 100mg QDS, MST 150mg BD, senna 2 tabs ON & Movicol one sachet BD.

In view of the continuing abdominal pain investigations were organised by the inpatient team revealing the following findings:

Hb	135 g/l
Platelets	224 * 10^9/l
WBC	11.2 * 10^9/l

Bilirubin	23 µmol/l
ALP	189 u/l
ALT	326 u/l
γGT	178 u/l
Albumin	34 g/l

Chest x-ray: Normal heart borders and lung fields, no evidence of subdiaphragmatic air.
Abdominal x-ray: presence of faeces, no evidence of dilatation.
USS abdomen: multiple hypoechoic lesions within the liver, nil else abnormal noted.

What is the next most appropriate management option?

- ○ Commence syringe driver containing diamorphine ×
- ○ Refer for immediate surgical consult ×
- ○ Refer for palliative radiotherapy ×
- ○ Convert MST to fentanyl ×

○ Commence trial with dexamethasone ×

Submit answer

Reference ranges

Score: **0%**

1	-
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Abdominal x-ray: presence of faeces, no evidence of dilatation.
USS abdomen: multiple hypoechoic lesions within the liver, nil else abnormal noted.

What is the next most appropriate management option?

Commence syringe driver containing diamorphine	20%
Refer for immediate surgical consult	4%
Refer for palliative radiotherapy	10%
Convert MST to fentanyl	19%

Commence trial with dexamethasone 47%

This gentleman has evidence of extensive hepatic metastases most probably arising from known adenocarcinoma of the pancreas. Despite an escalating opiate regimen he is unable to obtain satisfactory pain relief. One of the sources of pain associated with liver metastases is due to stretching and irritation of the liver capsule for which a trial of dexamethasone may provide an analgesic effect. Option 5 is, therefore, the most suitable option.

There is no evidence of obstruction or perforation and thus a surgical consult is not warranted, and palliative radiotherapy is not specifically indicated here (in contrast with bone metastases).

Switching to fentanyl will probably not provide any additional benefit in terms of tolerance compared with MST; it is however indicated in patients with poor renal failure owing to its predominant hepatic route of metabolism. For further information:

http://www.ruh.nhs.uk/ForClinicians/departmentsruh/PalliativeCare/documents/palliativecarehandbook.pd provides a helpful guide for prescribing in palliative settings.

Discuss (5) Improve

Next question >

Palliative care prescribing: pain

NICE guidelines

In 2012 NICE published guidelines on the use of opioids in palliative care. Selected points are listed below. Please see the link for more details.

Starting treatment
- when starting treatment, offer patients with advanced and progressive disease regular oral modified-release (MR) or oral immediate-release morphine (depending on patient preference), with oral immediate-release morphine for breakthrough pain
- if no comorbidities use 20-30mg of MR a day with 5mg morphine for breakthrough pain. For example, 15mg modified-release morphine tablets twice a day with 5mg of oral morphine solution as required
- oral modified-release morphine should be used in preference to transdermal patches
- laxatives should be prescribed for all patients initiating strong opioids
- patients should be advised that nausea is often transient. If it persists then an antiemetic should be offered
- drowsiness is usually transient - if it does not settle then adjustment of the dose should be considered

SIGN guidelines

SIGN issued guidance on the control of pain in adults with cancer in 2008. Selected points

- the breakthrough dose of morphine is one-sixth the daily dose of morphine
- all patients who receive opioids should be prescribed a laxative
- opioids should be used with caution in patients with chronic kidney disease
    - oxycodone is preferred to morphine in palliative patients with mild-moderate renal impairment
    - if renal impairment is more severe, alfentanil, buprenorphine and fentanyl are preferred
- metastatic bone pain may respond to strong opioids, bisphosphonates or radiotherapy. The assertion that NSAIDs are particularly effective for metastatic bone pain is not supported by studies. Strong opioids have the lowest number needed to treat for relieving the pain and can provide quick relief, in contrast to radiotherapy and bisphosphonates*. All patients, however, should be considered for referral to a clinical oncologist for consideration of further treatments such as radiotherapy

Other points

When increasing the dose of opioids the next dose should be increased by 30-50%.

In addition to strong opioids, bisphosphonates and radiotherapy, denosumab may be used to treat metastatic bone pain.

Opioid side-effects

Usually transient	**Usually persistent**
Nausea Drowsiness	Constipation

Conversion between opioids

From	**To**	**Conversion factor**
Oral codeine	Oral morphine	Divide by 10
Oral tramadol	Oral morphine	Divide by 10**

Oxycodone generally causes less sedation, vomiting and pruritis than morphine but more constipation.

From	To	Conversion factor
Oral morphine	Oral oxycodone	Divide by 1.5-2***

The current BNF gives the following conversion factors for transdermal perparations

- a transdermal fentanyl 12 microgram patch equates to approximately 30 mg oral morphine daily
- a transdermal buprenorphine 10 microgram patch equates to approximately 24 mg oral morphine daily.

From	To	Conversion factor
Oral morphine	Subcutaneous morphine	Divide by 2
Oral morphine	Subcutaneous diamorphine	Divide by 3
Oral oxycodone	Subcutaneous diamorphine	Divide by 1.5

*BMJ 2015;350:h315 Cancer induced bone pain

**this has previously been stated as 5 but the current version of the BNF states a conversion of 10

***historically a conversion factor of 2 has been used (i.e. oral oxycodone is twice as strong as oral morphine). The current BNF however uses a conversion rate of 1.5

123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

NICE 👍 3 👎 1

2012 Opioids in palliative care guidelines

BNF 👍 3 👎 3

Prescribing opioids in palliative care

Suggest link

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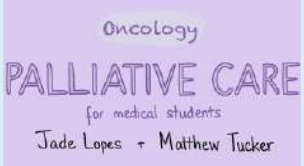
Media


Oncology
PALLIATIVE CARE
for medical students
Jade Lopes + Matthew Tucker

Complete Guide to Palliative Care for Medical Students

MedFlix - YouTube 👍 20 👎 2

HEART ATTACK
REFERRED PAIN
ACUTE PAIN
CHRONIC PAIN
PAIN
BRAIN
SPINAL CORD
C FIBRES
A-DELTA FIBRES
RECEPTORS
PERIPHERAL NERVES

Understanding The Causes of Pain and Pain Management

Zero To Finals - YouTube 👍 1 👎 1

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Score: **9.3%**

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A medical consult was sought regarding a 58-year-old gentleman admitted with pneumonia complaining of relentless hiccups. He was diagnosed with hepatocellular carcinoma six months ago and was deemed not for curative treatment owing to the extent of the disease. He was initially comfortable with no discernible symptoms but over the last four weeks developed hiccups. In conjunction with the palliative care nurse his GP had initially trialled domperidone and then haloperidol but with no improvement in his symptoms. He denied the presence of any other symptoms other than fatigue, and upon questioning denied the presence of abdominal pain, heartburn or early satiety.

His past medical history was comprised of alcohol dependence syndrome, chronic liver disease, hypertension and hypercholesterolaemia for which he was prescribed thiamine 100mg TDS, lactulose 10mls BD, spironolactone 100mg OD, propranolol 40mg OD, simvastatin 20mg OD as well as intravenous co-amoxiclav 625mg TDS for the treatment of his pneumonia.
Examination revealed the presence of a cachectic male. His blood pressure was 108/58 mmHg, heart rate 78 bpm, respiratory rate 18/min and temperature 36.6ºC. Examination of his gastrointestinal system revealed the presence of a palpable mass in the right upper quadrant inferior to the right sterna edge and ascites with no tenderness. Examination of his neurological system revealed the absence of any focal neurological symptoms; likewise, examination of cardiovascular and respiratory systems was unremarkable.

Recent investigations revealed the following results:

Bilirubin	22 µmol/l
ALP	221 u/l
ALT	278 u/l
Protein	62 g/l
Albumin	18 g/l

Chest x-ray: normal heart borders and lung fields

Surveillance upper GI endoscopy four months ago: normal stomach mucosal surface, no evidence of portal hypertension

Which of the following options is the best option with a view to managing his hiccups?

○ Commence treatment with proton pump inhibitor ×

○ Commence treatment with levomepromazine ×

○ Commence treatment with dexamethasone ×

○ Commence treatment with midazolam infusion ×

○ Commence treatment with baclofen ×

Submit answer

Reference ranges ∨

Score: **0%**

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Question 10 of 43 ✗

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Recent investigations revealed the following results:

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ALT	278 u/l
Protein	62 g/l
Albumin	18 g/l

Chest x-ray: normal heart borders and lung fields

Surveillance upper GI endoscopy four months ago: normal stomach mucosal surface, no evidence of portal hypertension

Which of the following options is the best option with a view to managing his hiccups?

Commence treatment with proton pump inhibitor	2%
Commence treatment with levomepromazine	38%

Commence treatment with dexamethasone	36%
Commence treatment with midazolam infusion	3%
Commence treatment with baclofen	22%

This gentleman has known advanced hepatocellular carcinoma and is complaining of distressing hiccups. He does not have any sign of gastro-oesophageal reflux and therefore a trial of proton pump inhibitor is unlikely to be of benefit. In the presence of hepatic or cerebral cancer a trial of dexamethasone may induce some remission and is therefore suggested as an appropriate next line given he has already had a trial of treatment with a prokinetic and a typical antipsychotic with no remission; see http://www.palliativecareguidelines.scot.nhs.uk/guidelines/symptom-control/Hiccups.aspx for further information. There is also limited evidence for treatment with baclofen and levomepromazine; subcutaneous midazolam should be reserved if all else proves futile. Note also the BNF has a very useful section regarding prescribing in palliative care scenarios; in this instance it also suggests the use of antacids or nifedipine in addition to the above options.

Discuss (6) Improve

Next question

Palliative care prescribing: hiccups

Management of hiccups

- chlorpromazine is licensed for the treatment of intractable hiccups
- haloperidol, gabapentin are also used
- dexamethasone is also used, particularly if there are hepatic lesions

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

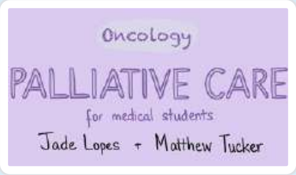
Links

NHS Scotland 👍 7 👎 4

Scottish Palliative Care Guidelines - hiccups

Suggest link Report broken link

Media


Oncology
PALLIATIVE CARE
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Complete Guide to Palliative Care for Medical Students

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Score: **9.3%**

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A 47-year-old woman presents to the emergency department with sudden onset of shortness of breath and pleuritic chest pain. Observations demonstrate marked hypoxia and she is given oxygen and analgesia and undergoes a chest X-ray followed by a CT pulmonary angiogram which shows a pulmonary embolism. There is also an incidental finding of a nodule suspicious of metastatic disease.

She is started on a treatment dose of low molecular weight heparin and admitted under the medical team for further assessment. During a more detailed history, she explains that she has lost one stone in weight over two months and been feeling progressively more tired as well. She denies any other symptoms. She has a past medical history of asthma and also underwent a caesarean section three years ago. She has two children, of which she breastfed both, and has a long history of use of oral contraceptive use. She is an ex-smoker of 5 pack-years and has minimal alcohol intake. Her grandfather died of prostate cancer, and her sister developed breast cancer at the age of 52.

A complete further examination was completed and no abnormalities were found. Routine blood tests are currently pending, and a flexible bronchoscopy for biopsy of the lung nodule has been arranged. How should this patient be further investigated?

○ Tumour markers AFP and hCG, and mammography

○ CT abdomen and pelvis, tumour markers AFP and hCG, and a myeloma screen

○ Ultrasound abdomen and pelvis, and tumours marker AFP and hCG

○ CT abdomen and pelvis, and tumour markers AFP and hCG

○ CT abdomen and pelvis, myeloma screen, and colonoscopy

Submit answer

Reference ranges

Score: **0%**

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A complete further examination was completed and no abnormalities were found. Routine blood tests are currently pending, and a flexible bronchoscopy for biopsy of the lung nodule has been arranged. How should this patient be further investigated?

Tumour markers AFP and hCG, and mammography	22%
CT abdomen and pelvis, tumour markers AFP and hCG, and a myeloma screen	21%
Ultrasound abdomen and pelvis, and tumours marker AFP and hCG	5%
CT abdomen and pelvis, and tumour markers AFP and hCG	46%
CT abdomen and pelvis, myeloma screen, and colonoscopy	7%

The correct answer is CT abdomen and pelvis, and tumour markers AFP and hCG. These investigations complete all those recommended by NICE for an unknown primary. This question highlights how to investigate a malignancy of unknown origin. This patient has a likely metastatic disease causing a pulmonary embolism with unclear clinical findings as to the location of the primary malignancy.

NICE recommends the following investigations for all patients:

- FBC, U&E, LFT, calcium, urinalysis, LDH
- Chest X-ray
- CT of chest, abdomen and pelvis
- AFP and hCG

NICE recommends the following investigations for specific patients:
- Myeloma screen (if lytic bone lesions)
- Endoscopy (directed towards symptoms)
- PSA (men)
- CA 125 (women with peritoneal malignancy or ascites)
- Testicular US (in men with germ cell tumours)
- Mammography (in women with clinical or pathological features compatible with breast cancer)

A is incorrect because it does not include CT scanning of the abdomen and pelvis. Mammography is tempting but NICE does not recommend it without any clinical findings which are suggestive of a breast malignancy. B and E are incorrect because a myeloma screen is not indicated without lytic bone lesions. E is also incorrect as colonoscopy should be symptom directed. C is incorrect as US is not an appropriate investigation.

👍 👎 Discuss (7) Improve

Next question >

Investigating metastatic disease of unknown primary ★

NICE recommends the following investigations for all patients:
- FBC, U&E, LFT, calcium, urinalysis, LDH
- Chest X-ray
- CT of chest, abdomen and pelvis
- AFP and hCG

NICE recommends the following investigations for specific patients:
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- Mammography (in women with clinical or pathological features compatible with breast cancer)

Source:
Metastatic Malignant Disease of Unknown Origin in Adults: Diagnosis and Management Clinical guideline [CG 104]. National Institute of Clinical Excellence. N.p., Jul. 2010.

+ Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

NICE 👍 0 👎 1

2010 Metastatic malignant disease of unknown primary origin in adults

Suggest link Report broken link

Score: **9.3%**

1 ✗
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Question 12 of 43

A 62-year-old lady with metastatic breast cancer is admitted with a general decline. She is found to have a pneumonia and treated with IV fluids and antibiotics. She makes limited improvement with these and antibiotics are stopped. The decision is made to focus on control of her symptoms.

She is started on a syringe driver of morphine sulphate to treat breathlessness and pain related to liver metastases. She then develops nausea and hiccups.

Which medication is the best choice to add to the syringe driver?

○ Cyclizine

○ Domperidone

○ Midazolam

○ Metoclopramide

○ Ondansetron

Submit answer

Reference ranges

Score: **0%**

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Question 12 of 43

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She is started on a syringe driver of morphine sulphate to treat breathlessness and pain related to liver metastases. She then develops nausea and hiccups.

Which medication is the best choice to add to the syringe driver?

Cyclizine	16%
Domperidone	16%
Midazolam	14%
Metoclopramide	40%
Ondansetron	15%

Cyclizine, domperidone and ondansetron are anti-emetics, though domperidone does not come in a subcutaneous preparation for syringe driver. Midazolam is an anxiolytic.

Metoclopramide is the first choice to treat hiccup as well as nausea. Other options include baclofen, nifedipine and chlorpromazine.

Reference:
British National Formulary. Guidance on prescribing in palliative care. 2015.

Discuss (4) Improve

Next question

Palliative care prescribing: hiccups

Management of hiccups
- chlorpromazine is licensed for the treatment of intractable hiccups
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123

Next question >

B *I* A

Textbooks

High-yield textbook

Extended textbook

Links

NHS Scotland 👍 7 👎 4

Scottish Palliative Care Guidelines - hiccups

Suggest link

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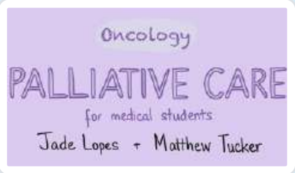
Media


Oncology
PALLIATIVE CARE
for medical students
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Complete Guide to Palliative Care for Medical Students

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Score: **9.3%**

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Question 13 of 43

A 60-year-old man has been admitted under urology with difficulty passing urine and back pain. A post-void bladder scan shows 980mls in his bladder. He was catheterised by the admitting doctor and now has haematuria.

He has a background of metastatic prostate cancer, atrial fibrillation, previous NSTEMI, hypertension, hypercholesterolaemia and hypothyroidism. His medications include warfarin and modified-release morphine 30mg every 12 hours.

He has been seen by the urology consultant who has said to put a 3-way catheter in if the haematuria does not settle and refer to the palliative care team for pain management. The palliative care specialist advised increasing the modified release morphine to 45mg every 12 hours.

What dose of immediate-release morphine should the patient be prescribed for breakthrough pain?

- ○ 5mg
- ○ 7.5mg
- ○ 10mg
- ○ 15mg
- ○ 20mg

Submit answer

Reference ranges

Score: **0%**

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Question 13 of 43

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What dose of immediate-release morphine should the patient be prescribed for breakthrough pain?

5mg	3%
7.5mg	6%
10mg	10%
15mg	80%
20mg	2%

Breakthrough dose = 1/6th of daily morphine dose

Important for me Less important

To answer this question, you need to calculate the total daily dose of morphine. The amount of immediate-release morphine for breakthrough pain should be one-sixth (1/6) of the total daily dose.

The patient was previously on 60mg of morphine per day. One-sixth of this would be 10mg of immediate-release morphine for breakthrough pain.

The modified release morphine has now been up titrated to 90mg per day. Whenever background morphine is increased, it is important to calculate the new breakthrough pain dosage for patients to receive the maximal benefit. In this case, one-sixth of 90 is 15mg. Therefore the correct answer is 15mg of immediate-release morphine.

👍 👎 Discuss (1) Improve

Next question ›

Palliative care prescribing: pain

NICE guidelines

In 2012 NICE published guidelines on the use of opioids in palliative care. Selected points are listed below. Please see the link for more details.

Starting treatment

- when starting treatment, offer patients with advanced and progressive disease regular oral modified-release (MR) or oral immediate-release morphine (depending on patient preference), with oral immediate-release morphine for breakthrough pain
- if no comorbidities use 20-30mg of MR a day with 5mg morphine for breakthrough pain. For example, 15mg modified-release morphine tablets twice a day with 5mg of oral morphine solution as required
- oral modified-release morphine should be used in preference to transdermal patches
- laxatives should be prescribed for all patients initiating strong opioids
- patients should be advised that nausea is often transient. If it persists then an antiemetic should be offered
- drowsiness is usually transient - if it does not settle then adjustment of the dose should be considered

SIGN guidelines

SIGN issued guidance on the control of pain in adults with cancer in 2008. Selected points

- the breakthrough dose of morphine is one-sixth the daily dose of morphine
- all patients who receive opioids should be prescribed a laxative
- opioids should be used with caution in patients with chronic kidney disease
  - oxycodone is preferred to morphine in palliative patients with mild-moderate renal impairment
  - if renal impairment is more severe, alfentanil, buprenorphine and fentanyl are preferred
- metastatic bone pain may respond to strong opioids, bisphosphonates or radiotherapy. The assertion that NSAIDs are particularly effective for metastatic bone pain is not supported by studies. Strong opioids have the lowest number needed to treat for relieving the pain and can provide quick relief, in contrast to radiotherapy and bisphosphonates*. All patients, however,

should be considered for referral to a clinical oncologist for consideration of further treatments such as radiotherapy

Other points

When increasing the dose of opioids the next dose should be increased by 30-50%.

In addition to strong opioids, bisphosphonates and radiotherapy, denosumab may be used to treat metastatic bone pain.

Opioid side-effects

Usually transient	**Usually persistent**
Nausea Drowsiness	Constipation

Conversion between opioids

From	**To**	**Conversion factor**
Oral codeine	Oral morphine	Divide by 10
Oral tramadol	Oral morphine	Divide by 10**

Oxycodone generally causes less sedation, vomiting and pruritis than morphine but more constipation.

From	**To**	**Conversion factor**
Oral morphine	Oral oxycodone	Divide by 1.5-2***

The current BNF gives the following conversion factors for transdermal perparations

- a transdermal fentanyl 12 microgram patch equates to approximately 30 mg oral morphine daily
- a transdermal buprenorphine 10 microgram patch equates to approximately 24 mg oral morphine daily.

From	To	Conversion factor
Oral morphine	Subcutaneous morphine	Divide by 2
Oral morphine	Subcutaneous diamorphine	Divide by 3
Oral oxycodone	Subcutaneous diamorphine	Divide by 1.5

*BMJ 2015;350:h315 Cancer induced bone pain

**this has previously been stated as 5 but the current version of the BNF states a conversion of 10

***historically a conversion factor of 2 has been used (i.e. oral oxycodone is twice as strong as oral morphine). The current BNF however uses a conversion rate of 1.5

+ Q 123

Next question >

B *I*

Textbooks

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Links

NICE 👍 3 👎 1

2012 Opioids in palliative care guidelines

BNF 👍 3 👎 3

Prescribing opioids in palliative care

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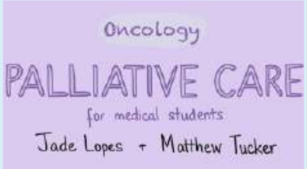
Media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BRAIN
SPINAL CORD
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Question 14 of 43

You are looking after an 82-year-old man with a background of metastatic colorectal carcinoma. He is in significant pain and is on a syringe driver containing 6mg of alfentanil. He has end-stage renal failure.

What breakthrough analgesic will you prescribe?

- ○ 1mg subcutaneous alfentanil
- ○ 25mg subcutaneous morphine
- ○ 25mg subcutaneous oxycodone
- ○ 2mg subcutaneous alfentanil
- ○ 50mg subcutaneous oxycodone

Submit answer

Reference ranges

Score: **0%**

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← Question 14 of 43 ✗ →

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What breakthrough analgesic will you prescribe?

1mg subcutaneous alfentanil	74%
25mg subcutaneous morphine	4%
25mg subcutaneous oxycodone	12%
2mg subcutaneous alfentanil	7%
50mg subcutaneous oxycodone	2%

Breakthrough dose = 1/6th of daily morphine dose

Important for me Less important

Palliative care guidelines recommend using 1/6 of the daily dose as a breakthrough. Alfentanil is a potent drug and is particularly useful in renal failure as it is metabolised by the liver, has no active metabolites, and has a very short half-life.

Discuss (2) Improve

Next question >

Palliative care prescribing: pain

NICE guidelines

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Usually transient	**Usually persistent**
Nausea Drowsiness	Constipation

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From	To	Conversion factor
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BNF 👍 3 👎 3

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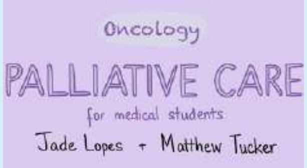
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Question 15 of 43

An 82-year-old man is reviewed by the palliative care team on the ward. He has a past medical history of metastatic lung cancer. He is not for any further chemotherapy, surgery or radiotherapy. He has deteriorated rapidly in the prior few days and is considered to be in the last days of life. He is suffering from intractable nausea, the cause of which is not clear.

What is the most appropriate medication to manage his symptoms?

- ○ Buscopan
- ○ Levomepromazine
- ○ Metoclopramide
- ○ Morphine
- ○ Ondansetron

Submit answer

Reference ranges

Score: **0%**

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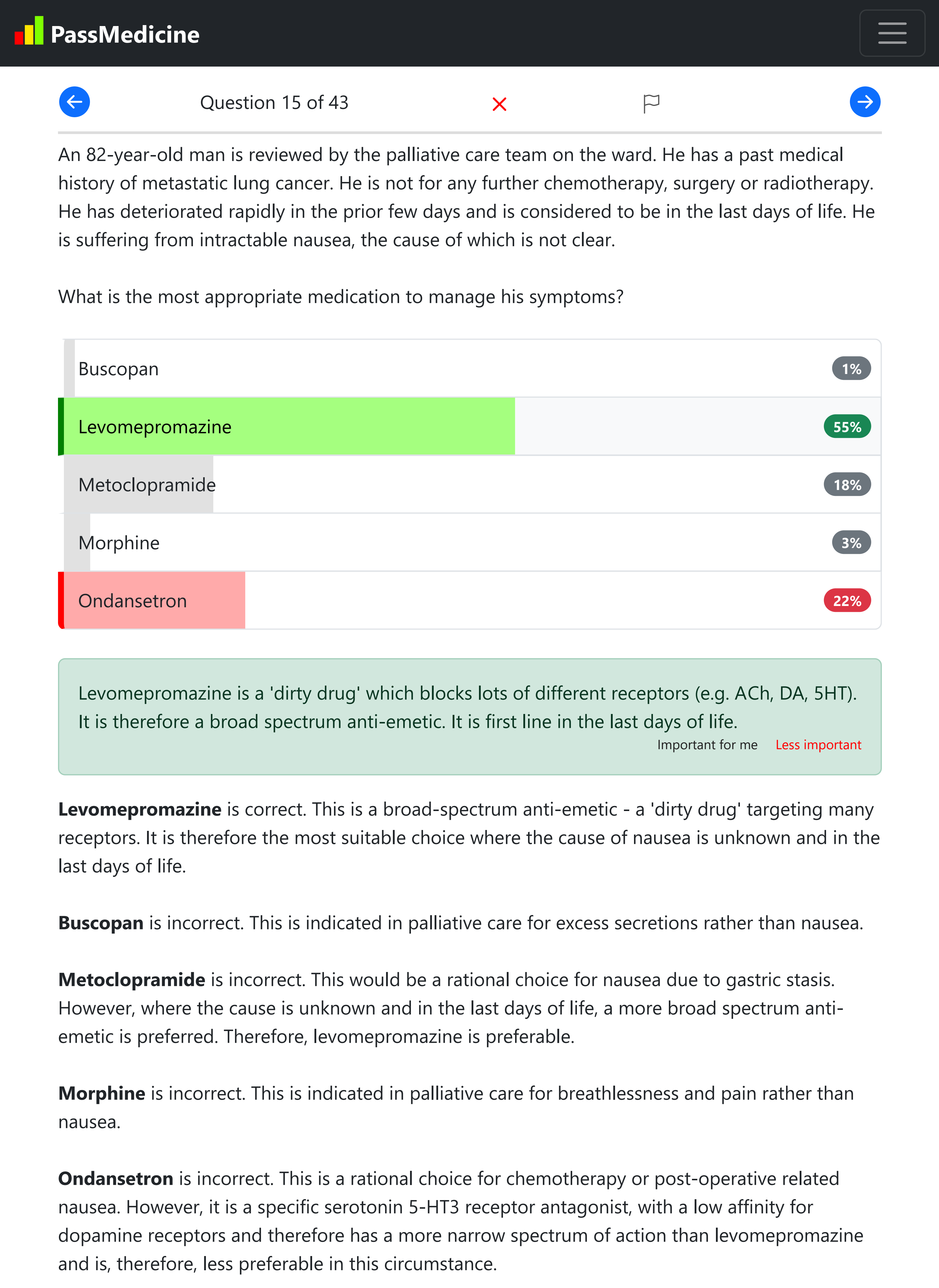

Question 15 of 43

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What is the most appropriate medication to manage his symptoms?

Buscopan	1%
Levomepromazine	55%
Metoclopramide	18%
Morphine	3%
Ondansetron	22%

Levomepromazine is a 'dirty drug' which blocks lots of different receptors (e.g. ACh, DA, 5HT). It is therefore a broad spectrum anti-emetic. It is first line in the last days of life.

Levomepromazine is correct. This is a broad-spectrum anti-emetic - a 'dirty drug' targeting many receptors. It is therefore the most suitable choice where the cause of nausea is unknown and in the last days of life.

Buscopan is incorrect. This is indicated in palliative care for excess secretions rather than nausea.

Metoclopramide is incorrect. This would be a rational choice for nausea due to gastric stasis. However, where the cause is unknown and in the last days of life, a more broad spectrum anti-emetic is preferred. Therefore, levomepromazine is preferable.

Morphine is incorrect. This is indicated in palliative care for breathlessness and pain rather than nausea.

Ondansetron is incorrect. This is a rational choice for chemotherapy or post-operative related nausea. However, it is a specific serotonin 5-HT3 receptor antagonist, with a low affinity for dopamine receptors and therefore has a more narrow spectrum of action than levomepromazine and is, therefore, less preferable in this circumstance.

Discuss (1) Improve

Next question

Syringe drivers

A syringe driver should be considered in the palliative care setting when a patient is unable to take oral medication due to nausea, dysphagia, intestinal obstruction, weakness or coma. In the UK there are two main types of syringe driver:

- Graseby MS16A (blue): the delivery rate is given in mm per hour
- Graseby MS26 (green): the delivery rate is given in mm per 24 hours

The majority of drugs are compatible with water for injection although for the following drugs sodium chloride 0.9% is recommended:

- granisetron
- ketamine
- ketorolac
- octreotide
- ondansetron

Commonly used drugs

- nausea and vomiting: cyclizine, levomepromazine, haloperidol, metoclopramide
- respiratory secretions/bowel colic: hyoscine hydrobromide, hyoscine butylbromide, or glycopyrronium bromide.
- agitation/restlessness: midazolam, haloperidol, levomepromazine
- pain: diamorphine is the preferred opioid

Mixing and compatibility issues

- diamorphine is compatible with the majority of other drugs used including dexamethasone, haloperidol, hyoscine butylbromide, hyoscine hydrobromide, levomepromazine, metoclopramide, midazolam
    - cyclizine may precipitate with diamorphine when given at higher doses
- cyclizine is incompatible with a number of drugs including clonidine, dexamethasone, hyoscine butylbromide (occasional), ketamine, ketorolac, metoclopramide, midazolam, octreotide, sodium chloride 0.9%

Next question

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Patient.info 👍 11 👎 12

Syringe drivers

Suggest link Report broken link

Score: **9.3%**

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Question 16 of 43

A 65-year-old woman presents to the emergency department with reduced consciousness. Her husband reports that she has been unwell for the past 3 days with coryzal symptoms. She has a past medical history of hypertension and metastatic pancreatic cancer for which she is under the palliative care team. Her regular medications include amlodipine, bisoprolol, paracetamol and a fentanyl patch for pain.

On examination, her temperature is 38.1ºC with a heart rate of 101 beats/min and a blood pressure of 118/66mmHg. Auscultation of the chest is clear with a respiratory rate of 8 breaths/min and oxygen saturations of 92% on air. Her abdomen is soft and non-tender and she is visibly jaundiced. She has a GCS of 11/15 (E2V4M5).

What is the most likely cause of this patient's reduced consciousness?

- ○ Delirium ×
- ○ Hepatic encephalopathy ×
- ○ Hypoglycaemia ×
- ○ Meningitis ×
- ○ Opioid toxicity ×

Submit answer

Reference ranges

Score: **0%**

1 -
2 -
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What is the most likely cause of this patient's reduced consciousness?

Delirium	8%
Hepatic encephalopathy	16%
Hypoglycaemia	2%
Meningitis	3%
Opioid toxicity	72%

Transdermal fentanyl absorption is increased by heat (e.g. hot water bottle) or pyrexia, potentially leading to opioid toxicity

Important for me Less important

Opioid toxicity is correct. This patient has signs of opioid toxicity including drowsiness and a slow respiratory rate. There are multiple differential diagnoses for an acutely unresponsive patient and, therefore, it is key to take a thorough history and examination. From this patient's collateral history it is learned that she uses a fentanyl patch for pain control. It is important to note that transdermal fentanyl absorption is increased by heat whether that be secondary to the application of a hot water bottle or pyrexia. This patient has a fever of 38.1ºC which is a likely contributing factor to her toxicity.

Delirium is incorrect. This is a very reasonable differential diagnosis given the history of fever in a patient with a frail comorbid status. However, delirium is less likely to cause this patient's reduced respiratory rate compared to opioid toxicity. Generally, fever is associated with tachypnoea and the opposite response should raise suspicion.

Hepatic encephalopathy is incorrect. Although this patient is jaundiced, it is likely to be cholestatic jaundice secondary to the underlying malignancy rather than decompensated liver disease. Furthermore, the acute onset of symptoms and lack of signs of hepatic encephalopathy makes the diagnosis less likely.

Hypoglycaemia is incorrect. This is an important differential diagnosis given the history of advanced malignancy and the likelihood of reduced oral intake. However, the presence of bradypnoea and opioid use makes opioid toxicity more likely.

Meningitis is incorrect. Fever and reduced consciousness are features of meningitis. However, the presence of bradypnoea and the use of strong opioid analgesics make opioid toxicity more likely.

👍 👎 Discuss (2) Improve

Next question >

Palliative care prescribing: pain ★

NICE guidelines

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Other points

When increasing the dose of opioids the next dose should be increased by 30-50%.

In addition to strong opioids, bisphosphonates and radiotherapy, denosumab may be used to treat metastatic bone pain.

Opioid side-effects

Usually transient	**Usually persistent**
Nausea Drowsiness	Constipation

Conversion between opioids

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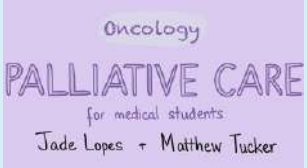
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Question 17 of 43

A 56-year-old man with metastatic small cell lung carcinoma is admitted to hospital with vomiting, ankle swelling and pruritus. He recently completed a course of palliative chemotherapy. He has a background of chronic obstructive pulmonary disease and hypertension and takes morphine sulphate (MST) for pain relief.

On examination, he is cachectic with peripheral oedema and skin excoriations. Heart rate is 96 beats per minute and blood pressure is 140/85 mmHg.

Tests show:

Hb	134 g/l
Platelets	185 * 10^9/l
WBC	5.5 * 10^9/l

Na^+	146 mmol/l
K^+	5.4 mmol/l
Urea	23 mmol/l
Creatinine	420 µmol/l

Urine dip:	Blood +
Urine osmolality	350 mOsm/L
Urinary sodium	45 mEqL
Microscopy:	red cells; casts.

What is the most appropriate pain relief for this patient?

- ○ Fentanyl patch ×
- ○ Diamorphine ×
- ○ Oxycodone ×
- ○ Pregabalin ×
- ○ Tramadol ×

Submit answer

Reference ranges

Score: **0%**

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A 56-year-old man with metastatic small cell lung carcinoma is admitted to hospital with vomiting, ankle swelling and pruritus. He recently completed a course of palliative chemotherapy. He has a background of chronic obstructive pulmonary disease and hypertension and takes morphine sulphate (MST) for pain relief.

On examination, he is cachectic with peripheral oedema and skin excoriations. Heart rate is 96 beats per minute and blood pressure is 140/85 mmHg.

Tests show:

Hb	134 g/l
Platelets	185 * 10^9/l
WBC	5.5 * 10^9/l

Na^+	146 mmol/l
K^+	5.4 mmol/l
Urea	23 mmol/l
Creatinine	420 µmol/l

Urine dip:	Blood +
Urine osmolality	350 mOsm/L
Urinary sodium	45 mEqL
Microscopy:	red cells; casts.

What is the most appropriate pain relief for this patient?

Fentanyl patch	56%
Diamorphine	5%
Oxycodone	36%
Pregabalin	1%
Tramadol	1%

This patient has an acute kidney injury secondary to cisplatin chemotherapy. As a result, he needs adjustments to his pain relief. He is on regular, long-acting MST, so the most appropriate substitution is a fentanyl patch.

Oxycodone can be used in moderate renal failure, but only as breakthrough pain relief. Modified release preparations should be avoided. Diamorphine and tramadol should be avoided in renal failure. Pregabalin can be administered at reduced dose, but would not be an appropriate first-line substitution for MST.

Alfentanil and methadone are safe even in severe renal impairment and are administered subcutaneously. Alfentanil would be appropriate for this patient if he had unstable pain and required dose titration.

👍 👎 Discuss (7) Improve

Next question >

Palliative care prescribing: pain ★

NICE guidelines

In 2012 NICE published guidelines on the use of opioids in palliative care. Selected points are listed below. Please see the link for more details.

Starting treatment
- when starting treatment, offer patients with advanced and progressive disease regular oral modified-release (MR) or oral immediate-release morphine (depending on patient preference), with oral immediate-release morphine for breakthrough pain
- if no comorbidities use 20-30mg of MR a day with 5mg morphine for breakthrough pain. For example, 15mg modified-release morphine tablets twice a day with 5mg of oral morphine solution as required
- oral modified-release morphine should be used in preference to transdermal patches
- laxatives should be prescribed for all patients initiating strong opioids
- patients should be advised that nausea is often transient. If it persists then an antiemetic should be offered
- drowsiness is usually transient - if it does not settle then adjustment of the dose should be considered

SIGN guidelines

SIGN issued guidance on the control of pain in adults with cancer in 2008. Selected points

- the breakthrough dose of morphine is one-sixth the daily dose of morphine
- all patients who receive opioids should be prescribed a laxative
- opioids should be used with caution in patients with chronic kidney disease
    - oxycodone is preferred to morphine in palliative patients with mild-moderate renal impairment
    - if renal impairment is more severe, alfentanil, buprenorphine and fentanyl are preferred
- metastatic bone pain may respond to strong opioids, bisphosphonates or radiotherapy. The assertion that NSAIDs are particularly effective for metastatic bone pain is not supported by studies. Strong opioids have the lowest number needed to treat for relieving the pain and can provide quick relief, in contrast to radiotherapy and bisphosphonates*. All patients, however, should be considered for referral to a clinical oncologist for consideration of further treatments such as radiotherapy

Other points

When increasing the dose of opioids the next dose should be increased by 30-50%.

In addition to strong opioids, bisphosphonates and radiotherapy, denosumab may be used to treat metastatic bone pain.

Opioid side-effects

Usually transient	**Usually persistent**
Nausea Drowsiness	Constipation

Conversion between opioids

From	**To**	**Conversion factor**
Oral codeine	Oral morphine	Divide by 10
Oral tramadol	Oral morphine	Divide by 10**

Oxycodone generally causes less sedation, vomiting and pruritis than morphine but more constipation.

From	**To**	**Conversion factor**
Oral morphine	Oral oxycodone	Divide by 1.5-2***

The current BNF gives the following conversion factors for transdermal perparations

- a transdermal fentanyl 12 microgram patch equates to approximately 30 mg oral morphine daily
- a transdermal buprenorphine 10 microgram patch equates to approximately 24 mg oral morphine daily.

From	To	Conversion factor
Oral morphine	Subcutaneous morphine	Divide by 2
Oral morphine	Subcutaneous diamorphine	Divide by 3
Oral oxycodone	Subcutaneous diamorphine	Divide by 1.5

*BMJ 2015;350:h315 Cancer induced bone pain

**this has previously been stated as 5 but the current version of the BNF states a conversion of 10

***historically a conversion factor of 2 has been used (i.e. oral oxycodone is twice as strong as oral morphine). The current BNF however uses a conversion rate of 1.5

+ Q 123

Next question >

Textbooks

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Extended textbook

Links

NICE	👍 3	👎 1

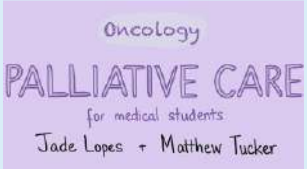
2012 Opioids in palliative care guidelines

BNF 👍 3 👎 3

Prescribing opioids in palliative care

Suggest link Report broken link

Media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PALLIATIVE CARE
for medical students
Jade Lopes + Matthew Tucker

Complete Guide to Palliative Care for Medical Students

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HEART ATTACK
REFERRED PAIN
ACUTE PAIN
CHRONIC PAIN
PAIN
BRAIN
SPINAL CORD
C FIBRES
A-DELTA FIBRES
RECEPTORS
PERIPHERAL NERVES

Understanding The Causes of Pain and Pain Management

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Question 18 of 43

A 69-year-old male patient presents with worsening mouth soreness and painful swallowing, progressively getting worse over the last week. He started radical radiotherapy for a base of mouth squamous cell cancer three weeks ago. He also has a PEG in situ. He was recently seen by the dietician and is now having fluid and Ensure nutritional supplements via his PEG. He had been given soluble paracetamol by the specialist nurse, but this has failed to control his pain.

On examination, his oral cavity appears inflamed with multiple small ulcers posterior to his lower lip. There are no patches or plaques inside. You change the paracetamol to liquid co-codamol and give him a topical lidocaine gel at 2% strength. What is the most appropriate next step in management to control his symptoms?

- ○ Benzydamine mouthwash
- ○ Menthol mouthwash
- ○ Chlorhexidine mouthwash
- ○ Fluconazole
- ○ Metronidazole

Submit answer

Reference ranges

Score: **0%**

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On examination, his oral cavity appears inflamed with multiple small ulcers posterior to his lower lip. There are no patches or plaques inside. You change the paracetamol to liquid co-codamol and give him a topical lidocaine gel at 2% strength. What is the most appropriate next step in management to control his symptoms?

Benzydamine mouthwash	60%
Menthol mouthwash	2%
Chlorhexidine mouthwash	31%
Fluconazole	6%
Metronidazole	1%

The correct answer is benzydamine mouthwash. This gentleman is suffering from radiotherapy-induced mucositis. Given the location of his tumour and the high dose of radiotherapy necessary for curative therapy, mucositis is not an uncommon side effect. Many of these patients will have a PEG inserted prophylactically in anticipation of the difficulties in maintaining an oral diet with mucositis and oesophageal involvement. For this patient, already on co-codamol, benzydamine mouthwash can be very helpful in treating his symptoms. Menthol and chlorhexidine mouthwashes, often bought over the counter, would increase oral irritation. There is no evidence of fungal or bacterial infection currently.

Discuss (6) Improve

Next question

Radiotherapy-induced mucositis

Many patients will suffer from mucositis following radiotherapy to the head and neck. Often these patients will have a PEG tube inserted prior to treatment to help maintain oral intake. Management of pain involves topical treatment such as local anaesthetic gels and benzydamine mouthwashes. Standard WHO based pain ladder should be followed, but medications should be in liquid form.

Sources:
Peterson, D. E., C. B. Boers-Doets, R. J. Bensadoun, and J. Herrstedt. 'Management of Oral and Gastrointestinal Mucosal Injury: ESMO Clinical Practice Guidelines for Diagnosis, Treatment, and Follow-up.' Ann Oncol Annals of Oncology 26.Suppl 5 (2015): V139-151.

Tanay, T., R. Logan, D. Riesenbeck, and A. Hovan. 'Mouth Care Guidance and Support in Cancer and Palliative Care.' UK Oral Mucositis in Cancer Group (2015): n. pag.

+ Q 123

Next question >

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Score: **9.3%**

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Question 19 of 43

A 75-year-old man is admitted to the palliative care unit for end of life care. He has a past medical history of metastatic prostate cancer. His drug history includes a syringe driver with morphine, levomepromazine and midazolam. During personal care the nurses have stated that he suffers excruciating pain when being turned onto his side. His pain throughout the day is otherwise well controlled.

What is the most appropriate way to manage his pain?

- ○ Titrate the syringe driver morphine ×
- ○ Morphine sulphate modified release tablets ×
- ○ Oxycodone modified release tablets ×
- ○ Morphine sulphate immediate release liquid ×
- ○ Paracetamol ×

Submit answer

Reference ranges

Score: **0%**

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What is the most appropriate way to manage his pain?

Titrate the syringe driver morphine	15%
Morphine sulphate modified release tablets	7%
Oxycodone modified release tablets	2%
Morphine sulphate immediate release liquid	74%
Paracetamol	2%

Incident pain is defined as pain which comes on as a result of an action or activity, for example during personal care. Sublingual fentanyl has rapid onset and is a short-acting opioid, and is therefore a good choice for treating this type of pain

Important for me Less important

Incident pain is defined as pain which comes on as a result of an action or activity, for example during personal care. Sublingual fentanyl has rapid onset and is a short-acting opioid, and is therefore a good choice for treating this type of pain. Similarly, morphine sulphate immediate release liquid is also suitable.

The patients pain during the day is otherwise well controlled therefore titrating up the syringe driver with more morphine is not appropriate. Not only will this not be particularly effective at treating the incident pain, it will also give rise to unnecessary side effects such as sedation and constipation.

Similarly, morphine sulphate modified release tablets have an action time of approximately 12 hours so they are therefore inappropriate for breakthrough pain as they will cause accumulation of morphine in the system predisposing to toxicity.

Discuss (4) Improve

Next question ›

Palliative care prescribing: pain

NICE guidelines

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SIGN guidelines

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Other points

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In addition to strong opioids, bisphosphonates and radiotherapy, denosumab may be used to treat metastatic bone pain.

Opioid side-effects

Usually transient	**Usually persistent**
Nausea Drowsiness	Constipation

Conversion between opioids

From	**To**	**Conversion factor**
Oral codeine	Oral morphine	Divide by 10
Oral tramadol	Oral morphine	Divide by 10**

Oxycodone generally causes less sedation, vomiting and pruritis than morphine but more constipation.

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*BMJ 2015;350:h315 Cancer induced bone pain

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***historically a conversion factor of 2 has been used (i.e. oral oxycodone is twice as strong as oral morphine). The current BNF however uses a conversion rate of 1.5

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Next question >

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Links

NICE 👍 3 👎 1

2012 Opioids in palliative care guidelines

BNF 👍 3 👎 3

Prescribing opioids in palliative care

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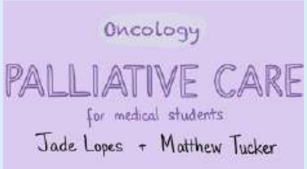
Media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HEART ATTACK
REFERRED PAIN
ACUTE PAIN
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PAIN
BRAIN
SPINAL CORD
C FIBRES
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RECEPTORS
PERIPHERAL NERVES

Understanding The Causes of Pain and Pain Management

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Question 20 of 43

A 64-year-old cachectic lady presents to the emergency department with difficulty in breathing. She reports smoking since she was 14 years old. Chest X-Ray reveals a large right sided pleural effusion. A sample is obtained under ultrasound-guidance for evaluation. CT does not identify a cause. The results of the pleural aspiration demonstrate an exudative effusion and histology is suggestive of an adenocarcinoma with positive TTF-1 stain. What is the most likely cause for the effusion?

- ○ Non-small cell lung cancer ×
- ○ Small cell lung cancer ×
- ○ Ovarian carcinoma ×
- ○ Breast carcinoma ×
- ○ Mesothelioma ×

Submit answer

Reference ranges

Score: **0%**

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Question 20 of 43 ✗

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Non-small cell lung cancer	43%
Small cell lung cancer	17%
Ovarian carcinoma	24%
Breast carcinoma	5%
Mesothelioma	10%

TTF-1 (thyroid transcription factor 1) is a protein which regulates the transcription of genes specific for the thyroid and lung. Lung adenocarcinomas and small cell carcinomas are usually TTF-1 positive. The histology in the above example is suggestive of an adenocarcinoma and therefore this is mostly likely a lung primary given the TTF-1 stain and history of smoking.

Non-small cell lung cancers include adenocarcinoma, large cell and squamous cell carcinoma.

Discuss (5) Improve

Next question >

Lung cancer: non-small cell

There are three main subtypes of non-small cell lung cancer:

Squamous cell cancer

- typically central
- associated with parathyroid hormone-related protein (PTHrP) secretion → hypercalcaemia
- strongly associated with finger clubbing
- cavitating lesions are more common than other types
- hypertrophic pulmonary osteoarthropathy (HPOA)

Adenocarcinoma

- typically peripheral
- most common type of lung cancer in non-smokers, although the majority of patients who develop lung adenocarcinoma are smokers

Large cell lung carcinoma

- typically peripheral
- anaplastic, poorly differentiated tumours with a poor prognosis
- may secrete β-hCG

123

Next question >

Textbooks

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Links

Radiopaedia 👍 4 👎 3

Lung cancer review

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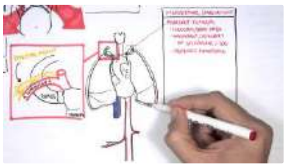
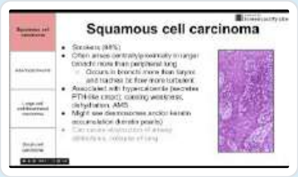
Media



Lung cancer

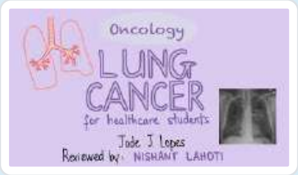
Armando Hasudungan - YouTube 👍 1 👎 0



Squamous cell carcinoma

Cancers of the lung

MedLecturesMadeEasy - YouTube 👍 1 👎 0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Question 21 of 43

A 76-year-old woman with metastatic transitional cell carcinoma of the bladder is admitted to hospice with a 3-day history of hallucinations, confusion and pruritus. Her only other symptoms are intermittent vomiting and pelvic discomfort. She has a past medical history of hypertension and has recently completed a course of radiotherapy for bone metastases. She takes paracetamol, morphine sulphate, cyclizine, dexamethasone and a herbal flower remedy for anxiety.

On examination, she is cachectic and frail. The lungs are clear and heart sounds are normal. Pulse rate is 85 beats per minute and blood pressure 130/80mmHg. She is afebrile.

Neurological examination reveals myoclonus. She is mildly hypotonic and all reflexes are diminished. There is miosis on cranial nerve examination.

Blood tests show:

Hb	12.1 g/dl
Platelets	250 * 10^9/l
WBC	12 * 10^9/l

Na^+	135 mmol/l
K^+	4.3 mmol/l
Urea	5 mmol/l
Creatinine	80 µmol/l
Calcium (unadjusted)	2.1 mmol/l

Bilirubin	15 µmol/l
ALP	45 u/l
ALT	15 u/l
AST	20 u/l
Albumin	28 g/l

Urine dip: no abnormality

What is the most likely diagnosis?

○ Hypercalcaemia

○ Opioid toxicity

○ Sepsis

○ Corticosteroid psychosis

○ Brain metastases

Submit answer

Reference ranges

Score: **0%**

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Creatinine	80 µmol/l
Calcium (unadjusted)	2.1 mmol/l

Bilirubin	15 µmol/l
ALP	45 u/l
ALT	15 u/l
AST	20 u/l
Albumin	28 g/l

Urine dip: no abnormality

What is the most likely diagnosis?

Hypercalcaemia 8%

Opioid toxicity	60%
Sepsis	1%
Corticosteroid psychosis	16%
Brain metastases	15%

This lady is presenting with opioid toxicity. She has vomiting, confusion, hallucinations and pruritus. Her symptoms have been precipitated by a reduction in her opioid requirement. This is a common occurrence in patients who have radiotherapy for bone metastases if their medication dose is not adjusted.

Hypercalcaemia could cause a similar presentation, but the corrected calcium is 2.4 mmol/L. This lady is hypoalbuminaemic due to her advanced cancer. The patient no symptoms of infection. She is apyrexial and her slightly raised white cell count is due to her advanced disease and dexamethasone treatment. Corticosteroid psychosis is possible, but would not cause the neurological signs or pruritus. Transitional cell carcinomas of the urothelial tract only rarely metastasise to the brain; the neurological signs suggest opioid toxicity rather than a focal lesion.

Discuss (5) | Improve

Next question >

Palliative care prescribing: pain

NICE guidelines

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Starting treatment

- when starting treatment, offer patients with advanced and progressive disease regular oral modified-release (MR) or oral immediate-release morphine (depending on patient preference), with oral immediate-release morphine for breakthrough pain
- if no comorbidities use 20-30mg of MR a day with 5mg morphine for breakthrough pain. For example, 15mg modified-release morphine tablets twice a day with 5mg of oral morphine solution as required
- oral modified-release morphine should be used in preference to transdermal patches
- laxatives should be prescribed for all patients initiating strong opioids
- patients should be advised that nausea is often transient. If it persists then an antiemetic should be offered

- drowsiness is usually transient - if it does not settle then adjustment of the dose should be considered

SIGN guidelines

SIGN issued guidance on the control of pain in adults with cancer in 2008. Selected points

- the breakthrough dose of morphine is one-sixth the daily dose of morphine
- all patients who receive opioids should be prescribed a laxative
- opioids should be used with caution in patients with chronic kidney disease
  - oxycodone is preferred to morphine in palliative patients with mild-moderate renal impairment
  - if renal impairment is more severe, alfentanil, buprenorphine and fentanyl are preferred
- metastatic bone pain may respond to strong opioids, bisphosphonates or radiotherapy. The assertion that NSAIDs are particularly effective for metastatic bone pain is not supported by studies. Strong opioids have the lowest number needed to treat for relieving the pain and can provide quick relief, in contrast to radiotherapy and bisphosphonates*. All patients, however, should be considered for referral to a clinical oncologist for consideration of further treatments such as radiotherapy

Other points

When increasing the dose of opioids the next dose should be increased by 30-50%.

In addition to strong opioids, bisphosphonates and radiotherapy, denosumab may be used to treat metastatic bone pain.

Opioid side-effects

Usually transient	**Usually persistent**
Nausea Drowsiness	Constipation

Conversion between opioids

From	**To**	**Conversion factor**
Oral codeine	Oral morphine	Divide by 10
Oral tramadol	Oral morphine	Divide by 10**

Oxycodone generally causes less sedation, vomiting and pruritis than morphine but more constipation.

From	To	Conversion factor
Oral morphine	Oral oxycodone	Divide by 1.5-2***

The current BNF gives the following conversion factors for transdermal perparations

- a transdermal fentanyl 12 microgram patch equates to approximately 30 mg oral morphine daily
- a transdermal buprenorphine 10 microgram patch equates to approximately 24 mg oral morphine daily.

From	To	Conversion factor
Oral morphine	Subcutaneous morphine	Divide by 2
Oral morphine	Subcutaneous diamorphine	Divide by 3
Oral oxycodone	Subcutaneous diamorphine	Divide by 1.5

*BMJ 2015;350:h315 Cancer induced bone pain

**this has previously been stated as 5 but the current version of the BNF states a conversion of 10

***historically a conversion factor of 2 has been used (i.e. oral oxycodone is twice as strong as oral morphine). The current BNF however uses a conversion rate of 1.5

123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

NICE 👍 3 👎 1

2012 Opioids in palliative care guidelines

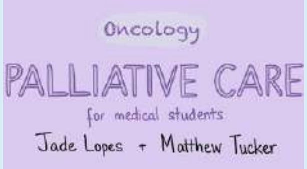
BNF 👍 3 👎 3

Prescribing opioids in palliative care

Suggest link

Report broken link

Media


Oncology
PALLIATIVE CARE
for medical students
Jade Lopes + Matthew Tucker

Complete Guide to Palliative Care for Medical Students

MedFlix - YouTube 👍 20 👎 2

HEART ATTACK
REFERRED PAIN
ACUTE PAIN
CHRONIC PAIN
PAIN
BRAIN
SPINAL CORD
C FIBRES
A-DELTA FIBRES
RECEPTORS
PERIPHERAL NERVES

Understanding The Causes of Pain and Pain Management

Zero To Finals - YouTube 👍 1 👎 1

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Score: **9.3%**

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43 ✕

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Back to top

A 65-year-old man is referred to the oncology team after presenting to the emergency department. The patient described a three-week history of progressive thoracic back pain. The pain was localised to a point between the patient's shoulder blades, and was exacerbated by movement and coughing.

In addition, over the previous 6 hours, the patient reported that he had been struggling to stand up unassisted, and was having difficultly walking. The sudden change in his mobility had been the event that had prompted his attendance at hospital. When questioned directly, the patient described intermittent tingling sensations in his lower legs, but denied any loss of sensation. There had been no dysfunction of his bladder or bowels.

The patient had been diagnosed with prostate cancer five years previously. This had been treated with a radical prostatectomy and hormone therapy. The patient had moved area two years previously, and had subsequently been lost to urological follow-up. Despite his previous cancer treatment, the patient stated that he was very fit and active, playing golf three times a week and taking an active role in the care of his grandchildren.

Peripheral nerve examination of lower limbs: tone generally increased bilaterally; power generally slightly reduced (4+/5) bilaterally; normal coordination; no impairment of light touch or sharp touch sensation; knee and ankle jerks brisk bilaterally, plantar responses up going bilaterally.

Digital rectal examination: preserved peri-anal sensation and normal tone.

Bed-side bladder scan: 50 ml

MRI whole spine: metastatic invasion and collapse of T10 vertebral with spinal cord compression.

The patient was treated with analgesia and dexamethasone while definitive treatment was planned.

What is most appropriate first-line treatment of patient's spinal cord compression?

- ○ Surgical tumour resection and spinal cord stabilisation with adjuvant radiotherapy ×
- ○ Radiotherapy: single dose 8 Gy ×
- ○ Chemotherapy: docetaxel ×
- ○ Best supportive care ×

○ Chemotherapy: cabazitaxel ×

Submit answer

Reference ranges

Score: **0%**

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Bed-side bladder scan: 50 ml

MRI whole spine: metastatic invasion and collapse of T10 vertebral with spinal cord compression.

The patient was treated with analgesia and dexamethasone while definitive treatment was planned.

What is most appropriate first-line treatment of patient's spinal cord compression?

Surgical tumour resection and spinal cord stabilisation with adjuvant radiotherapy	55%
Radiotherapy: single dose 8 Gy	39%
Chemotherapy: docetaxel	2%
Best supportive care	4%

Chemotherapy: cabazitaxel 1%

The best predictor of neurological outcome with treatment in metastatic spinal cord compression is neurological status at the initiation of treatment. The patient in the question has presented very promptly and has only limited neurological compromise. Therefore, it is imperative that definitive treatment is provided promptly (ideally, within 24 hours) to optimise long-term outcome.

Definite treatment of metastatic spinal cord compression can include any combination of radiotherapy, surgery and chemotherapy. Recent evidence indicates that surgery is superior to radiotherapy in retaining or regaining neurological function and relieving pain. In the case of this patient, who has an excellent performance status, surgery followed by radiotherapy would likely offer a better outcome than radiotherapy alone.

Radiotherapy may be considered as first-line treatment in some circumstances. Recommended regimen vary, for example 20 Gy in five daily fractions. The radiotherapy regime offered as a answer to the question would be appropriate as palliative treatment for pain-control, making it an incorrect answer for this patient.

Chemotherapy have a role in the management of spinal-cord compression secondary to chemo-sensitive malignancies (for example, small-cell lung cancer or germ cell tumours), but would not be optimal treatment for this patient.

Al-Qurainy, Collis E. Metastatic spinal cord compression: diagnosis and management. BMJ 2016;353:i2539.

👍 👎 Discuss (5) Improve

Next question

Neoplastic spinal cord compression

Spinal cord compression is an oncological emergency and affects up to 5% of cancer patients. Extradural compression accounts for the majority of cases, usually due to vertebral body metastases. It is more common in patients with lung, breast and prostate cancer

Features

- back pain
    - the earliest and most common symptom
    - may be worse on lying down and coughing
- lower limb weakness
- sensory changes: sensory loss and numbness
- neurological signs depend on the level of the lesion. Lesions above L1 usually result in upper motor neuron signs in the legs and a sensory level. Lesions below L1 usually cause lower

motor neuron signs in the legs and perianal numbness. Tendon reflexes tend to be increased below the level of the lesion and absent at the level of the lesion

Investigation

- urgent MRI: the 2019 NICE guidelines recommend a whole MRI spine within 24 hours of presentation

Management

- high-dose oral dexamethasone
- urgent oncological assessment for consideration of radiotherapy or surgery

+ Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

St Luke's Alliance 7 9

Suspected metastatic spinal cord compression pathway

NICE 7 1

2008 Metastatic spinal cord compression guidelines

Suggest link

Report broken link

Media

ANATOMY
SPINAL CORD
CLINICAL ANATOMY

Spinal Cord - Clinical Anatomy and Physiology (dermatomes, blood supply, shingles, lumbar puncture)

Armando Hasudungan - YouTube 👍 2 👎 0

ONCOLOGY
SPINAL CORD
COMPRESSION

Spinal Cord Compression

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Oncology
Metastatic
Spinal Cord
Compression
Oncological Emergencies

Metastatic spinal cord compression

Oncology for Medical Students - YouTube 👍 2 👎 1

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Score: **9.3%**

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A 56-year-old patient presents to hospital with confusion. He is a long-term smoker. He has a past-medical history of chronic obstructive pulmonary disease, hypertension, bipolar disease and long-term muscular back pain.

On examination he is confused with a Glasgow Coma Scale score of 14 out of 15. He has a pulse rate of 97 beats per minute with a blood pressure of 145/97 mmHg. Respiratory rate is 14 per minute with oxygen saturations of 95% on room air. He has bilateral vesicular breath sounds on auscultation with normal percussion. His abdomen is soft and non-tender with no masses. He has no focal neurology.

Investigations

Hb	102 g/dL
WCC	8.2 *10^9/l
Platelets	133 *10^9/l

Na+	146 mmol/L
K+	5.4 mmol/L
Cr	132 µmol/L
Ur	10.1 mmol/L

Albumin	26 g/L
Adjusted Calcium	3.78 mmol/L
Alkaline Phosphatase	76 IU/L
PTH	Undetectable
Serum ACE	45U/L (10-50)
Serum Electrophoresis	IgG 18 g/L (5-13 g/L) - Polyclonal
Urinary Electrophoresis	Undetectable

Chest X-ray	Right parahilar opacification 3x4cm
Lumbar Spine X-ray	Normal

What is the likely underlying diagnosis?

○ Squamous Cell Carcinoma ×

○ Multiple Myeloma ×

○ Sarcoidosis ×

○ Drug induced hypercalcaemia ×

○ Prostate carcinoma ×

Submit answer

Reference ranges ∨

Score: **0%**

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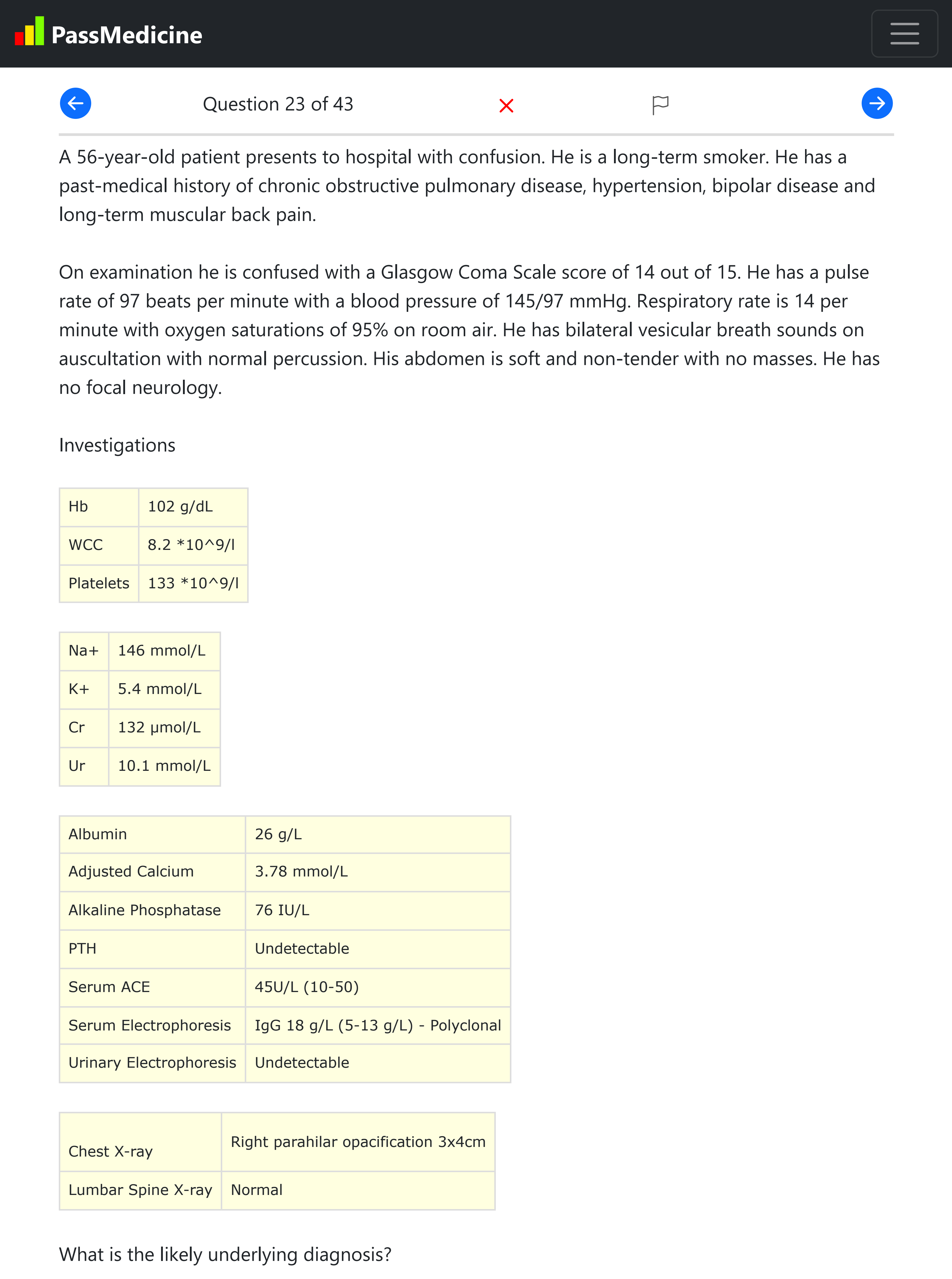

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Serum Electrophoresis	IgG 18 g/L (5-13 g/L) - Polyclonal
Urinary Electrophoresis	Undetectable

Chest X-ray	Right parahilar opacification 3x4cm
Lumbar Spine X-ray	Normal

What is the likely underlying diagnosis?

Squamous Cell Carcinoma	63%
Multiple Myeloma	22%
Sarcoidosis	13%
Drug induced hypercalcaemia	1%
Prostate carcinoma	1%

The patient has severe hypercalcaemia. The commonest causes are hyperparathyroidism and cancer. The patient has a normal alkaline phosphatase without evidence of fracture or bone disease making bone metastases unlikely. The patient has a normal serum ACE with an atypical chest X-ray for sarcoidosis, although unilateral chest findings are possible.

The patient has features of multiple myeloma including anaemia, renal insufficiency and hypercalcaemia. However, there is a polyclonal, rather than a monoclonal increase in paraprotein.

The chest x-ray findings with a background of smoking suggest an underlying respiratory malignancy. This patient has squamous cell carcinoma of the lung with parathyroid related protein secretion causing hypercalcaemia. PrPTH is not deteced on a PTH assay. Hypercalcaemia can induce confusion and renal impairment.

👍 👎 Discuss (9) Improve

Next question >

Lung cancer: non-small cell ★

There are three main subtypes of non-small cell lung cancer:

Squamous cell cancer
- typically central
- associated with parathyroid hormone-related protein (PTHrP) secretion → hypercalcaemia
- strongly associated with finger clubbing
- cavitating lesions are more common than other types
- hypertrophic pulmonary osteoarthropathy (HPOA)

Adenocarcinoma
- typically peripheral
- most common type of lung cancer in non-smokers, although the majority of patients who develop lung adenocarcinoma are smokers

Large cell lung carcinoma

- typically peripheral
- anaplastic, poorly differentiated tumours with a poor prognosis
- may secrete β-hCG

123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Radiopaedia 👍 4 👎 3

Lung cancer review

Suggest link

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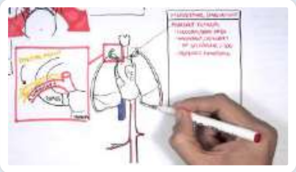
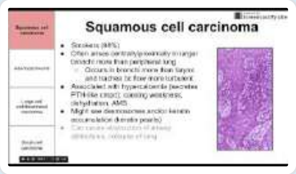
Media



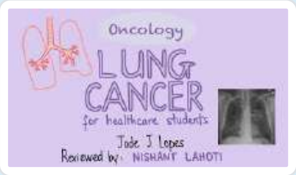
Lung cancer

Armando Hasudungan - YouTube 👍 1 👎 0



Cancers of the lung

MedLecturesMadeEasy - YouTube 👍 1 👎 0


Oncology
LUNG CANCER
for healthcare students
Jode J Lopes
Reviewed by: NISHANT LAHOTI

Lung cancer

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Oncology
LUNG CANCER:
An Overview

Lung Cancer: An Overview

Oncology for Medical Students - YouTube 👍 0 👎 0

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Score: **9.3%**

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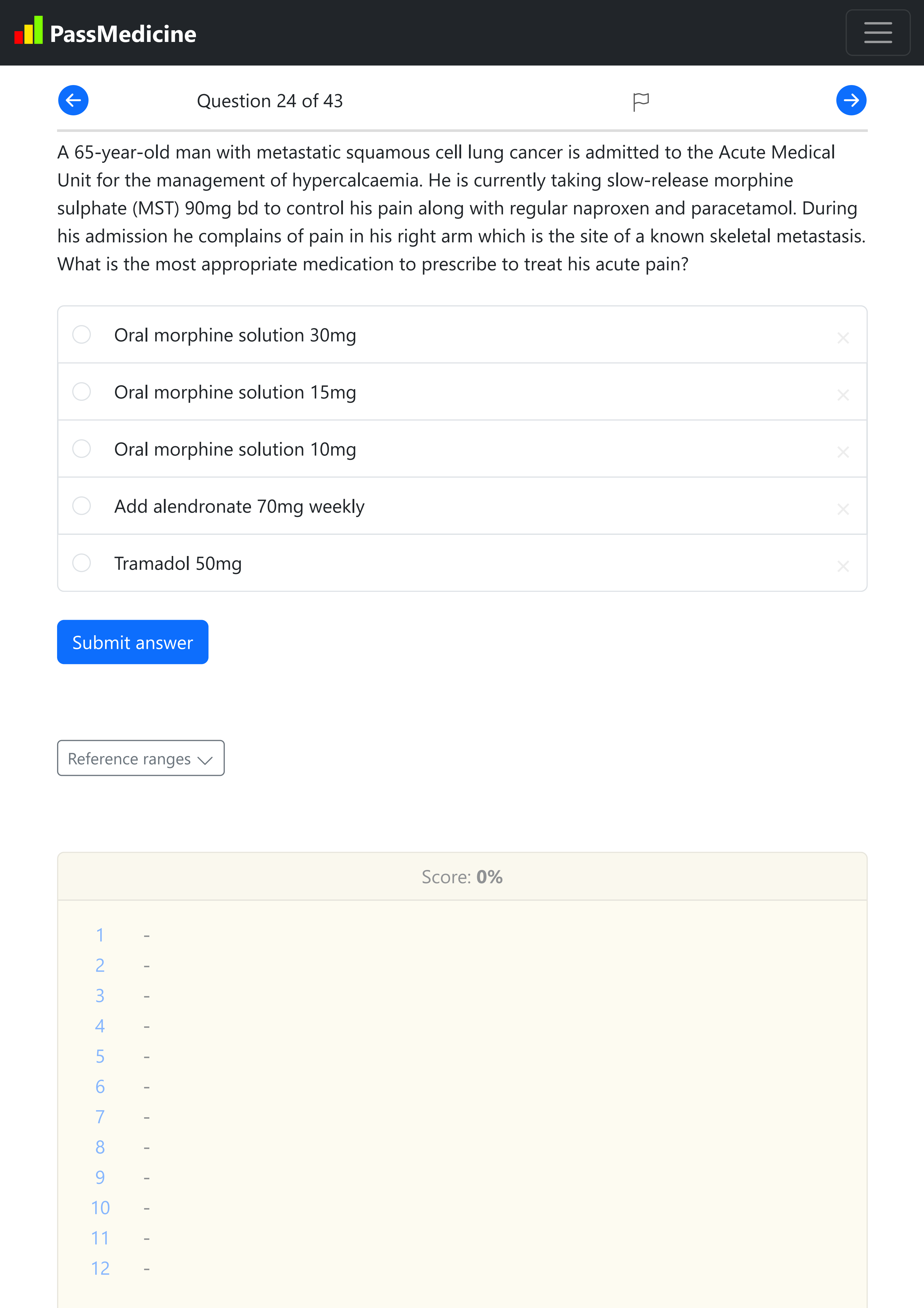

A 65-year-old man with metastatic squamous cell lung cancer is admitted to the Acute Medical Unit for the management of hypercalcaemia. He is currently taking slow-release morphine sulphate (MST) 90mg bd to control his pain along with regular naproxen and paracetamol. During his admission he complains of pain in his right arm which is the site of a known skeletal metastasis. What is the most appropriate medication to prescribe to treat his acute pain?

- ○ Oral morphine solution 30mg
- ○ Oral morphine solution 15mg
- ○ Oral morphine solution 10mg
- ○ Add alendronate 70mg weekly
- ○ Tramadol 50mg

Score: **0%**

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Question 24 of 43 ✗

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Oral morphine solution 30mg	63%
Oral morphine solution 15mg	13%
Oral morphine solution 10mg	2%
Add alendronate 70mg weekly	19%
Tramadol 50mg	2%

This patient is describing break-through pain. Whilst bisphosphonates have a role in bone metastases they are not suitable for acute pain. A suitable break-through dose is therefore 1/6th of the total daily morphine dose i.e. 1/6th of 180mg = 30mg.

Discuss (2) Improve

Next question ›

Palliative care prescribing: pain

NICE guidelines

In 2012 NICE published guidelines on the use of opioids in palliative care. Selected points are listed below. Please see the link for more details.

Starting treatment

- when starting treatment, offer patients with advanced and progressive disease regular oral modified-release (MR) or oral immediate-release morphine (depending on patient preference), with oral immediate-release morphine for breakthrough pain
- if no comorbidities use 20-30mg of MR a day with 5mg morphine for breakthrough pain. For example, 15mg modified-release morphine tablets twice a day with 5mg of oral morphine

solution as required
- oral modified-release morphine should be used in preference to transdermal patches
- laxatives should be prescribed for all patients initiating strong opioids
- patients should be advised that nausea is often transient. If it persists then an antiemetic should be offered
- drowsiness is usually transient - if it does not settle then adjustment of the dose should be considered

SIGN guidelines

SIGN issued guidance on the control of pain in adults with cancer in 2008. Selected points
- the breakthrough dose of morphine is one-sixth the daily dose of morphine
- all patients who receive opioids should be prescribed a laxative
- opioids should be used with caution in patients with chronic kidney disease
  - oxycodone is preferred to morphine in palliative patients with mild-moderate renal impairment
  - if renal impairment is more severe, alfentanil, buprenorphine and fentanyl are preferred
- metastatic bone pain may respond to strong opioids, bisphosphonates or radiotherapy. The assertion that NSAIDs are particularly effective for metastatic bone pain is not supported by studies. Strong opioids have the lowest number needed to treat for relieving the pain and can provide quick relief, in contrast to radiotherapy and bisphosphonates*. All patients, however, should be considered for referral to a clinical oncologist for consideration of further treatments such as radiotherapy

Other points

When increasing the dose of opioids the next dose should be increased by 30-50%.

In addition to strong opioids, bisphosphonates and radiotherapy, denosumab may be used to treat metastatic bone pain.

Opioid side-effects

Usually transient	**Usually persistent**
Nausea Drowsiness	Constipation

Conversion between opioids

From	To	Conversion factor
Oral codeine	Oral morphine	Divide by 10
Oral tramadol	Oral morphine	Divide by 10**

Oxycodone generally causes less sedation, vomiting and pruritis than morphine but more constipation.

From	To	Conversion factor
Oral morphine	Oral oxycodone	Divide by 1.5-2***

The current BNF gives the following conversion factors for transdermal perparations
- a transdermal fentanyl 12 microgram patch equates to approximately 30 mg oral morphine daily
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*BMJ 2015;350:h315 Cancer induced bone pain

**this has previously been stated as 5 but the current version of the BNF states a conversion of 10

***historically a conversion factor of 2 has been used (i.e. oral oxycodone is twice as strong as oral morphine). The current BNF however uses a conversion rate of 1.5

+ Q 123

Next question ›

B I A

Textbooks

High-yield textbook

Extended textbook

Links

NICE 👍 3 👎 1

2012 Opioids in palliative care guidelines

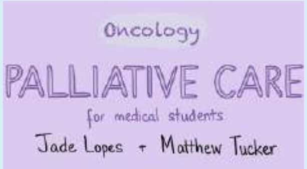
BNF 👍 3 👎 3

Prescribing opioids in palliative care

Suggest link

Report broken link

Media


Oncology
PALLIATIVE CARE
for medical students
Jade Lopes + Matthew Tucker

Complete Guide to Palliative Care for Medical Students

MedFlix - YouTube 👍 20 👎 2

HEART ATTACK
REFERRED PAIN
ACUTE PAIN
CHRONIC PAIN
PAIN
BRAIN
SPINAL CORD
C FIBRES
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PERIPHERAL NERVES

Understanding The Causes of Pain and Pain Management

Zero To Finals - YouTube 👍 1 👎 1

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Score: **9.3%**

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Question 25 of 43

A 77-year-old female is electively admitted for investigation of bilateral lower limb swelling onset over the past 3 years associated with limitation of exercise as a result. Her past medical history includes bilateral pulmonary emboli in 2010 and type 2 diabetes mellitus. On examination, non-pitting bilateral lower limb oedema is noted to the high thighs without abdominal involvement. Her heart sounds are normal with no added sounds, her chest is clear. Her abdomen is soft and non-tender without ascites or superficial swelling. A transthoracic echocardiogram demonstrates normal left ventricular function but was unable to produce good views of the right heart. A CT pulmonary angiogram demonstrates no embolus, a ventilation-perfusion scan demonstrates a low probability of pulmonary emboli. Ultrasound abdomen is unremarkable. The patient undergoes a right heart catheter, demonstrating a mean pulmonary arterial pressure of 14mmHg. What is the likely cause of her lower limb swelling?

- ○ Left heart failure ×
- ○ Idiopathic pulmonary arterial hypertension ×
- ○ Right heart failure ×
- ○ Pelvic malignancy ×
- ○ Lymphoedema ×

Submit answer

Reference ranges

Score: **0%**

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A 77-year-old female is electively admitted for investigation of bilateral lower limb swelling onset over the past 3 years associated with limitation of exercise as a result. Her past medical history includes bilateral pulmonary emboli in 2010 and type 2 diabetes mellitus. On examination, non-pitting bilateral lower limb oedema is noted to the high thighs without abdominal involvement. Her heart sounds are normal with no added sounds, her chest is clear. Her abdomen is soft and non-tender without ascites or superficial swelling. A transthoracic echocardiogram demonstrates normal left ventricular function but was unable to produce good views of the right heart. A CT pulmonary angiogram demonstrates no embolus, a ventilation-perfusion scan demonstrates a low probability of pulmonary emboli. Ultrasound abdomen is unremarkable. The patient undergoes a right heart catheter, demonstrating a mean pulmonary arterial pressure of 14mmHg. What is the likely cause of her lower limb swelling?

Left heart failure	1%
Idiopathic pulmonary arterial hypertension	7%
Right heart failure	14%
Pelvic malignancy	22%
Lymphoedema	56%

Interpretation of the various investigations this patient has undertaken allows lymphoedema as a diagnosis of exclusion. The patient would have been referred to this standard set of investigations for pulmonary hypertension after clinically presenting as progressive exercise limitation and peripheral oedema, with the primary concern being right heart failure secondary to an increased pressure within the pulmonary vasculature.

Most importantly, the pulmonary arterial pressure is normal on the right heart catheter, ruling idiopathic pulmonary hypertension. Left heart dysfunction is excluded by a normal echocardiogram evaluation of the left ventricular function. CTPA and normal abdominal ultrasound, predominantly investigating the portal circulation, rule out causes of pulmonary hypertension. Lastly, the non-pitting nature of the oedema suggests a non-cardiac cause, making lymphoedema the most likely cause of lower limb swelling.

Discuss (7) | Improve

Next question

Lymphoedema

Causes of lymphoedema include:

- primary: inherited
- secondary e.g. surgery, radiation, infection (classically filariasis) or injury resulting in damage to the lymphatic system

+ Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Media

Lymphoedema

Osmosis - YouTube 3 0

Report broken media

Score: **9.3%**

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Question 26 of 43

A 65-year-old man is admitted to the hospital for palliative radiotherapy. He has a squamous cell carcinoma of the tongue that has metastasized to his neck and surrounds the carotid artery. There are no surgical options to treat his disease.

On day 2 of the admission, he experiences a catastrophic bleed from his neck.

What is the most appropriate medication to administer in this circumstance?

- ○ Buscopan
- ○ Chlorpromazine
- ○ Haloperidol
- ○ Midazolam
- ○ Morphine

Submit answer

Reference ranges

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Question 26 of 43 ✗

A 65-year-old man is admitted to the hospital for palliative radiotherapy. He has a squamous cell carcinoma of the tongue that has metastasized to his neck and surrounds the carotid artery. There are no surgical options to treat his disease.

On day 2 of the admission, he experiences a catastrophic bleed from his neck.

What is the most appropriate medication to administer in this circumstance?

Buscopan	4%
Chlorpromazine	3%
Haloperidol	4%
Midazolam	79%
Morphine	10%

In a massive terminal haemorrhage, a large dose of midazolam (10mg) can be given as part of 'crisis management' to relieve distress. Red or green towels or blankets should be available to soak up and mask the colour of blood

Important for me Less important

Midazolam is the correct answer. The patient has experienced a terminal haemorrhage as a consequence of his tumour eroding through the carotid artery. Given the lack of surgical options available to treat his condition, urgent symptom control is warranted to manage this distressing event. Intravenous midazolam is indicated in this horrific situation as a consequence of its anxiolytic and sedative effects, along with the rapidity of its onset.

Haloperidol is incorrect. This can be used to treat agitation in palliative care. However, intravenous midazolam is preferred in this catastrophic situation due to its rapidity of onset.

Morphine is incorrect. This is typically used to treat pain and breathlessness in a palliative care setting, rather than agitation.

Chlorpromazine is incorrect. This can also be used to treat agitation in a palliative care setting. However, midazolam is preferred in terminal haemorrhages.

Buscopan is incorrect. This is used to decrease the burden of secretions in palliative care. It is not used to treat agitation.

Discuss (3) | Improve

Next question ›

Palliative care prescribing: agitation and confusion

Underlying causes of confusion need to be looked for and treated as appropriate, for example hypercalcaemia, infection, urinary retention and medication. If specific treatments fail then the following may be tried:

- first choice: haloperidol
- other options: chlorpromazine, levomepromazine

In the terminal phase of the illness then agitation or restlessness is best treated with midazolam

+ Q 123

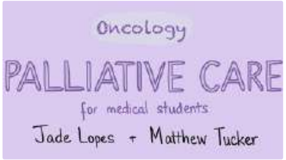
Next question ›

Textbooks

High-yield textbook

Extended textbook

Media


Oncology
PALLIATIVE CARE
for medical students
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Complete Guide to Palliative Care for Medical Students

MedFlix - YouTube 👍 1 👎 2

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A 64-year-old Nepalese woman who emigrated to the United Kingdom six months prior arrives to her GP. She has known multinodular goitre which has been untreated. However, her daughter who translates for her reports that she has noticed a tight sensation like she is being strangled. This is worse in the mornings and her daughter has noticed that her face does appear puffy. She has no weight loss, fevers or night sweats. She is otherwise well and has never smoked.

On examination, there is a large multinodular goitre that occupies a large part of the neck and measures 15-20cm in width. There is no audible stridor but there are several neck veins visible. You ask her to stretch out her hands up high to observe for a tremor and she starts to complain of the tightness. becoming worse. Her chest sounds are quiet and her chest is clear. There is dull percussion under the top third of the sternum.

TSH	0.07 mU/L (normal range 0.05-5.0)
T4	10 pmol/L (normal range 9-50)
T3	4.0 pmol/L (normal range 3.5-7.8)
D-dimer	0.15 (normal range 0-0.25)
Chest x-ray	Widened mediastinum, no focal lung lesion

What is the underlying cause of these new symptoms?

○ Multinodular goitre ×

○ Lymphoma ×

○ Thoracic artery aneurysm ×

○ Lung carcinoma ×

○ Superior vena cava thrombus ×

Submit answer

Reference ranges

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Question 27 of 43 ✗

A 64-year-old Nepalese woman who emigrated to the United Kingdom six months prior arrives to her GP. She has known multinodular goitre which has been untreated. However, her daughter who translates for her reports that she has noticed a tight sensation like she is being strangled. This is worse in the mornings and her daughter has noticed that her face does appear puffy. She has no weight loss, fevers or night sweats. She is otherwise well and has never smoked.

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TSH	0.07 mU/L (normal range 0.05-5.0)
T4	10 pmol/L (normal range 9-50)
T3	4.0 pmol/L (normal range 3.5-7.8)
D-dimer	0.15 (normal range 0-0.25)
Chest x-ray	Widened mediastinum, no focal lung lesion

What is the underlying cause of these new symptoms?

Multinodular goitre	28%
Lymphoma	13%
Thoracic artery aneurysm	4%
Lung carcinoma	4%
Superior vena cava thrombus	50%

Enlarged goitre can be a rare cause of SVC obstruction

Important for me Less important

This presentation is classical for superior vena cava obstruction and the worsening of her symptoms when she lifts her hands up is Pemberton's test which is positive in this case. The next decision is to seek the cause. The obvious cause here is a huge goitre which is recognised as a rare cause of superior cava obstruction. Lung carcinoma is possible but this lady is a non smoker and there is no cough or weight loss. Thoracic artery aneurysm would show a widened mediastinum but there would often be a history of connective tissue disease or uncontrolled hypertension. Lymphoma causing SVC obstruction would typically have mediastinal lymphadenopathy on chest x-ray. SVC thrombus would be unlikely given that the D-dimer is negative

Discuss (8) | Improve

Next question

Superior vena cava obstruction

Superior vena cava (SVC) obstruction is an oncological emergency caused by compression of the SVC. It is most commonly associated with lung cancer.

Features
- dyspnoea is the most common symptom
- swelling of the face, neck and arms - conjunctival and periorbital oedema may be seen
- headache: often worse in the mornings
- visual disturbance
- pulseless jugular venous distension

Causes
- common malignancies: small cell lung cancer, lymphoma
- other malignancies: metastatic seminoma, Kaposi's sarcoma, breast cancer
- aortic aneurysm
- mediastinal fibrosis
- goitre
- SVC thrombosis

Management is dependant on the individual patient and malignancy and advice should be taken from the oncology team. Options include:
- endovascular stenting is often the treatment of choice to provide symptom relief
- certain malignancies such as lymphoma, small cell lung cancer may benefit from radical chemotherapy or chemo-radiotherapy rather than stenting
- the evidence base supporting the use of glucocorticoids is weak but they are often given

AI + Q 123

Next question

Textbooks

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Media

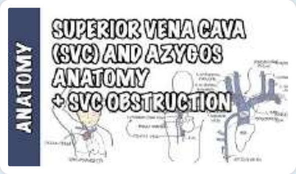
SVC SYNDROME

Superior Vena Cava Syndrome in 3 Minutes

Townsend Teaching - YouTube 👍 2 👎 1

Superior vena cava syndrome

Osmosis - YouTube 👍 2 👎 1


ANATOMY
SUPERIOR VENA CAVA
(SVC) AND AZYGOS
ANATOMY
+SVC OBSTRUCTION

Superior vena cava and the azygos system clinical anatomy - SVC obstruction (oncology emergency)

Armando Hasudungan - YouTube 👍 1 👎 2

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Score: **9.3%**

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A 78-year-old gentleman with known metastatic prostate cancer is admitted due to lower back pain that is worse on sitting upright. He denies any sensory changes or weakness but is woken at night and cannot sleep. He takes aspirin, paracetamol and bicalutamide regularly.

On examination, he has normal tone, full power and downgoing plantars in both lower legs. He is exquisitely tender in the midline at L2 and L3.

Bilirubin	34 µmol/l
ALP	450 u/l
ALT	56 u/l
Albumin	32 g/l
Calcium	3.1mmol/l
MRI spine	sclerotic lesions seen in T9, L2 and L3

What medical therapy would offer greatest benefit to him?

- ○ Intravenous fluids ×
- ○ Denosumab ×
- ○ Spinal decompressive surgery ×
- ○ Omeprazole ×
- ○ Transcutaneous electrical nerve stimulation (TENS) ×

Submit answer

Reference ranges

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Question 28 of 43 ✗

A 78-year-old gentleman with known metastatic prostate cancer is admitted due to lower back pain that is worse on sitting upright. He denies any sensory changes or weakness but is woken at night and cannot sleep. He takes aspirin, paracetamol and bicalutamide regularly.

On examination, he has normal tone, full power and downgoing plantars in both lower legs. He is exquisitely tender in the midline at L2 and L3.

Bilirubin	34 µmol/l
ALP	450 u/l
ALT	56 u/l
Albumin	32 g/l
Calcium	3.1mmol/l
MRI spine	sclerotic lesions seen in T9, L2 and L3

What medical therapy would offer greatest benefit to him?

Intravenous fluids	13%
Denosumab	62%
Spinal decompressive surgery	19%
Omeprazole	0%
Transcutaneous electrical nerve stimulation (TENS)	6%

Denosumab is an unlicensed medication for bony metastasis pain

Important for me   Less important

This gentleman has bony pain associated with bony metastases. This is a key question in identifying the problem and how to help him. There is no sign of nerve compression so spinal surgery will not benefit him. He is asymptomatic from his hypercalcaemia so IV fluids would not benefit. Omeprazole will help protect his stomach from ulceration. For bony pain, NSAIDs usually are a good first line. Bisphosphonates and radiotherapy help but are not an answer. Denosumab is a third line when the above fail. TENS can be useful for all pains but is not evidence based for bony pain.

👍 👎 Discuss (10) Improve

Next question ›

Palliative care prescribing: pain ★

NICE guidelines

In 2012 NICE published guidelines on the use of opioids in palliative care. Selected points are listed below. Please see the link for more details.

Starting treatment

- when starting treatment, offer patients with advanced and progressive disease regular oral modified-release (MR) or oral immediate-release morphine (depending on patient preference), with oral immediate-release morphine for breakthrough pain
- if no comorbidities use 20-30mg of MR a day with 5mg morphine for breakthrough pain. For example, 15mg modified-release morphine tablets twice a day with 5mg of oral morphine solution as required
- oral modified-release morphine should be used in preference to transdermal patches
- laxatives should be prescribed for all patients initiating strong opioids
- patients should be advised that nausea is often transient. If it persists then an antiemetic should be offered
- drowsiness is usually transient - if it does not settle then adjustment of the dose should be considered

SIGN guidelines

SIGN issued guidance on the control of pain in adults with cancer in 2008. Selected points

- the breakthrough dose of morphine is one-sixth the daily dose of morphine
- all patients who receive opioids should be prescribed a laxative
- opioids should be used with caution in patients with chronic kidney disease
    - oxycodone is preferred to morphine in palliative patients with mild-moderate renal impairment
    - if renal impairment is more severe, alfentanil, buprenorphine and fentanyl are preferred
- metastatic bone pain may respond to strong opioids, bisphosphonates or radiotherapy. The assertion that NSAIDs are particularly effective for metastatic bone pain is not supported by studies. Strong opioids have the lowest number needed to treat for relieving the pain and can provide quick relief, in contrast to radiotherapy and bisphosphonates*. All patients, however,

should be considered for referral to a clinical oncologist for consideration of further treatments such as radiotherapy

Other points

When increasing the dose of opioids the next dose should be increased by 30-50%.

In addition to strong opioids, bisphosphonates and radiotherapy, denosumab may be used to treat metastatic bone pain.

Opioid side-effects

Usually transient	Usually persistent
Nausea Drowsiness	Constipation

Conversion between opioids

From	To	Conversion factor
Oral codeine	Oral morphine	Divide by 10
Oral tramadol	Oral morphine	Divide by 10**

Oxycodone generally causes less sedation, vomiting and pruritis than morphine but more constipation.

From	To	Conversion factor
Oral morphine	Oral oxycodone	Divide by 1.5-2***

The current BNF gives the following conversion factors for transdermal perparations
- a transdermal fentanyl 12 microgram patch equates to approximately 30 mg oral morphine daily
- a transdermal buprenorphine 10 microgram patch equates to approximately 24 mg oral morphine daily.

From	To	Conversion factor
Oral morphine	Subcutaneous morphine	Divide by 2
Oral morphine	Subcutaneous diamorphine	Divide by 3
Oral oxycodone	Subcutaneous diamorphine	Divide by 1.5

*BMJ 2015;350:h315 Cancer induced bone pain

**this has previously been stated as 5 but the current version of the BNF states a conversion of 10

***historically a conversion factor of 2 has been used (i.e. oral oxycodone is twice as strong as oral morphine). The current BNF however uses a conversion rate of 1.5

AI + Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

NICE 👍 3 👎 1

2012 Opioids in palliative care guidelines

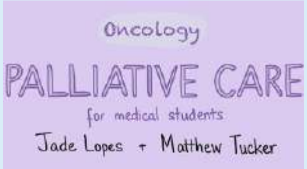
BNF 👍 3 👎 3

Prescribing opioids in palliative care

Suggest link

Report broken link

Media


Oncology
PALLIATIVE CARE
for medical students
Jade Lopes + Matthew Tucker

Complete Guide to Palliative Care for Medical Students

MedFlix - YouTube 👍 20 👎 2

HEART ATTACK
REFERRED PAIN
ACUTE PAIN
CHRONIC PAIN
PAIN
BRAIN
SPINAL CORD
C FIBRES
A-DELTA FIBRES
RECEPTORS
PERIPHERAL NERVES

Understanding The Causes of Pain and Pain Management

Zero To Finals - YouTube 👍 1 👎 1

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Score: **9.3%**

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Question 29 of 43

You are fast-bleeped to the oncology ward to review a 72-year-old man with a background of metastatic, fungating buccal cancer. During initial MDT, it was deemed inoperable due to carotid artery involvement. He was initially admitted with hypercalcaemia but has deteriorated during his admission and is on the end-of-life pathway.

On your arrival, you note a large amount of blood around the patient with staff using towels and applying pressure to the neck. Nursing staff report that there was a sudden 'pop' and large volume bleeding began at the site of the fungating cancer. They state that bleeding has occurred before but not to this extent.

What is the most appropriate next step in management?

- ○ Adrenaline ×
- ○ Midazolam ×
- ○ Morphine ×
- ○ Terlipressin ×
- ○ Vascular surgery opinion ×

Submit answer

Reference ranges

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On your arrival, you note a large amount of blood around the patient with staff using towels and applying pressure to the neck. Nursing staff report that there was a sudden 'pop' and large volume bleeding began at the site of the fungating cancer. They state that bleeding has occurred before but not to this extent.

What is the most appropriate next step in management?

Adrenaline	8%
Midazolam	74%
Morphine	5%
Terlipressin	6%
Vascular surgery opinion	7%

In a massive terminal haemorrhage, a large dose of midazolam (10mg) can be given as part of 'crisis management' to relieve distress. Red or green towels or blankets should be available to soak up and mask the colour of blood

Important for me Less important

This is an example of a catastrophic, terminal haemorrhage likely arising from carotid artery involvement. In these circumstances, patients can become very distressed and unfortunately, often there is very little that can be done. To aid in relieving stress it is advised to use red or green towels to help soak up blood as well as obscuring the colour of the blood, as well as giving a large dose of midazolam for relaxation.

Adrenaline can be used in specific circumstances of major haemorrhage but is not used in terminal haemorrhage. Given that the history explains that the patient is on the end of life pathway and that he is not for surgical intervention, adrenaline is not appropriate.

Morphine can be used for pain and breathlessness in palliative care but is not used in terminal haemorrhages.

Terlipressin can be used for major haemorrhage, particularly of gastrointestinal bleeding, however again, this is not appropriate in terminal haemorrhages.

The MDT has already decided that surgical intervention is not appropriate and therefore a vascular surgery opinion at this time would not be appropriate.

Discuss (4) Improve

Next question

Palliative care prescribing: agitation and confusion

Underlying causes of confusion need to be looked for and treated as appropriate, for example hypercalcaemia, infection, urinary retention and medication. If specific treatments fail then the following may be tried:

- first choice: haloperidol
- other options: chlorpromazine, levomepromazine

In the terminal phase of the illness then agitation or restlessness is best treated with midazolam

+ Q 123

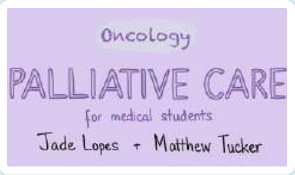
Next question

Textbooks

High-yield textbook

Extended textbook

Media


Oncology
PALLIATIVE CARE
for medical students
Jade Lopes + Matthew Tucker

Complete Guide to Palliative Care for Medical Students

MedFlix - YouTube 👍 1 👎 2

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Score: **9.3%**

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Question 30 of 43

A 60-year-old woman presented to the emergency department with recurrent episodes of severe headache and vomiting over past 3-4 days. She was known to have underlying metastatic breast cancer for which she was receiving palliative chemotherapy. She was also known to have underlying type 2 diabetes mellitus and osteoarthritis of bilateral knee joints.

On examination, her pulse rate was 105/min, respiratory rate was 16/min, blood pressure was 120/80 mmHg. Fundoscopy revealed early papilloedema.

A contrast enhanced CT brain was done which showed a solitary mass of 3 cm in the left cerebral hemisphere.

What is the next appropriate management?

○ Start 16 mg of i.v dexamethasone and prophylactic anticonvulsants

○ Start 16 mg of dexamethasone and arrange urgent MRI brain

○ Refer to neurosurgery for biopsy of the brain lesion

○ Refer to radiation oncologist for Whole brain radiotherapy

○ Stereotactic radiosurgery

Submit answer

Reference ranges

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A 60-year-old woman presented to the emergency department with recurrent episodes of severe headache and vomiting over past 3-4 days. She was known to have underlying metastatic breast cancer for which she was receiving palliative chemotherapy. She was also known to have underlying type 2 diabetes mellitus and osteoarthritis of bilateral knee joints.

On examination, her pulse rate was 105/min, respiratory rate was 16/min, blood pressure was 120/80 mmHg. Fundoscopy revealed early papilloedema.

A contrast enhanced CT brain was done which showed a solitary mass of 3 cm in the left cerebral hemisphere.

What is the next appropriate management?

Start 16 mg of i.v dexamethasone and prophylactic anticonvulsants	37%
Start 16 mg of dexamethasone and arrange urgent MRI brain	57%
Refer to neurosurgery for biopsy of the brain lesion	1%
Refer to radiation oncologist for Whole brain radiotherapy	2%
Stereotactic radiosurgery	2%

High dose dexamethasone is the mainstay of immediate management of brain metastasis. For lesions identified as solitary on contrast enhanced CT brain, MRI with gadolinium is indicated to assess the extent of intracranial disease. Patients presenting with seizure activity should be given anticonvulsant therapy. Patients with limited number of lesions(<4) can be considered for more aggressive treatment like neurosurgery or stereotactic radiotherapy but not the immediate step. Biopsy of the lesion is indicated if primary is unknown.Whole brain radiotherapy is indicated for multiple brain metastasis (>4).
Reference: Clinical medicine 2014 Vol 14, No 5: 535-7

Discuss (4) | Improve

Next question

Breast cancer: management

The management of breast cancer depends on the staging, tumour type and patient background. It may involve any of the following:

- surgery
- radiotherapy
- hormone therapy
- biological therapy
- chemotherapy

Surgery

The vast majority of patients who have breast cancer diagnosed will be offered surgery. An exception may be a very frail, elderly lady with metastatic disease who may be better managed with hormonal therapy.

Prior to surgery, the presence/absence of axillary lymphadenopathy determines management:

- women with no palpable axillary lymphadenopathy at presentation should have a pre-operative axillary ultrasound before their primary surgery
    - if negative then they should have a sentinel node biopsy to assess the nodal burden
- in patients with breast cancer who present with clinically palpable lymphadenopathy, axillary node clearance is indicated at primary surgery
    - this may lead to arm lymphedema and functional arm impairment

Depending on the characteristics of the tumour women either have a wide-local excision or a mastectomy. Around two-thirds of tumours can be removed with a wide-local excision. The table below lists some of the factors determining which operation is offered:

Mastectomy	**Wide Local Excision**
Multifocal tumour	Solitary lesion
Central tumour	Peripheral tumour
Large lesion in small breast	Small lesion in large breast
DCIS > 4cm	DCIS < 4cm

Women should be offered breast reconstruction to achieve a cosmetically suitable result regardless of the type of operation they have. For women who've had a mastectomy this may be done at the initial operation or at a later date.

Radiotherapy

Whole breast radiotherapy is recommended after a woman has had a wide-local excision as this may reduce the risk of recurrence by around two-thirds. For women who've had a mastectomy radiotherapy is offered for T3-T4 tumours and for those with four or more positive axillary nodes

Hormonal therapy

Adjuvant hormonal therapy is offered if tumours are positive for hormone receptors. For many years this was done using tamoxifen for 5 years after diagnosis. Tamoxifen is still used in pre- and peri-menopausal women. In post-menopausal women, aromatase inhibitors such as anastrozole are used for this purpose*. This is important as aromatisation accounts for the majority of oestrogen production in post-menopausal women and therefore anastrozole is used for ER +ve breast cancer in this group.

Important side effects of tamoxifen include an increased risk of endometrial cancer, venous thromboembolism and menopausal symptoms.

Biological therapy

The most common type of biological therapy used for breast cancer is trastuzumab (Herceptin). It is only useful in the 20-25% of tumours that are HER2 positive.

Trastuzumab cannot be used in patients with a history of heart disorders.

Chemotherapy

Cytotoxic therapy may be used either prior to surgery ('neoadjuvanant' chemotherapy) to downstage a primary lesion or after surgery depending on the stage of the tumour, for example, if there is axillary node disease - FEC-D is used in this situation.

+ Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

NICE 👍 1 👎 1

Early and locally advanced breast cancer: Diagnosis and treatment

Suggest link

Report broken link

Media

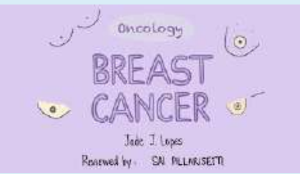
Pharmacology - CANCER DRUGS - HORMONAL THERAPY

Speed Pharmacology - YouTube 👍 2 👎 0

BREAST
CANCER
Osmosis.org

Breast cancer

Osmosis - YouTube 👍 1 👎 0


Oncology
BREAST
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Breast cancer

MedFlix - YouTube 👍 0 👎 0

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Score: **9.3%**

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Question 31 of 43

A 48-year-old woman is currently undergoing various cycles of chemotherapy for breast cancer. She asks the oncology team for advice as, 2-3 days post-chemotherapy, she experiences severe nausea and vomiting that lasts for several days. This is debilitating, despite taking antiemetics during the chemotherapy and afterward.

What is the most appropriate drug to prevent this delayed reaction?

- ○ Dexamethasone
- ○ Levopromazine
- ○ Lorazepam
- ○ Metoclopramide
- ○ Ondansetron

Submit answer

Reference ranges

Score: **0%**

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What is the most appropriate drug to prevent this delayed reaction?

Dexamethasone	42%
Levopromazine	12%
Lorazepam	2%
Metoclopramide	8%
Ondansetron	36%

Dexamethasone is the most useful agent in preventing the delayed phase of chemotherapy-induced emesis

Important for me Less important

Dexamethasone has been shown by numerous trials to be the most effective agent in preventing delayed nausea and vomiting. As opposed to more immediate nausea and vomiting, for which antiemetics are useful, the delayed reaction (between 24 hours and 1-week post-chemotherapy) is not as effectively reduced by antiemetics.

Levopromazine is a 'dirty drug', acting at multiple receptor sites. It is a useful antiemetic, particularly parenterally, but sedates patients quite significantly and is generally reserved for palliative care and intractable symptoms.

Lorazepam, a benzodiazepine, has mild antiemetic properties and is not considered effective at reducing the delayed phase. It is generally used for its anti-anxiolytic properties.

Metoclopramide is a dopamine antagonist and an effective antiemetic. It may be used first-line for immediate nausea and vomiting. However, in the context of the delayed phase, dexamethasone is more useful.

Ondansetron is a serotonin receptor antagonist and another effective antiemetic used for chemotherapy-induced nausea and vomiting. With regards to the delayed phase, however, dexamethasone is the most effective agent.

Discuss (1) Improve

Next question >

Chemotherapy side-effects: nausea and vomiting

Nausea and vomiting are common side-effects of chemotherapy. Risk factors for the development of symptoms include:

- anxiety
- age less than 50 years old
- concurrent use of opioids
- the type of chemotherapy used

For patients at low-risk of symptoms then drugs such as metoclopramide may be used first-line. For high-risk patients then 5HT3 receptor antagonists such as ondansetron are often effective, especially if combined with dexamethasone

+ Q 123

Next question >

Textbooks

High-yield textbook

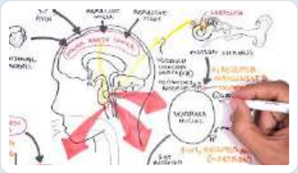
Extended textbook

Media

VOMITING
REFLEX

Physiology of Vomiting - Vomiting reflex

Armando Hasudungan - YouTube 👍 3 👎 0



Pharmacology - Antiemetics

Armando Hasudungan - YouTube 👍 1 👎 0

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Score: **9.3%**

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Question 32 of 43

A 68-year-old man with metastatic prostate cancer is due to go for radiotherapy in an hour. The nurses tell you that he complained of pain during the transfer yesterday. His pain has been otherwise well controlled on a total of 60mg morphine daily. How is it best to manage his pain control for the transfer today?

○ Nothing as it is only temporary pain

○ An additional dose of 10mg morphine 30 minutes prior to his transfer

○ An additional dose of 15mg morphine 1 hour prior to his transfer

○ An additional dose of 10mg morphine 1 hour prior to his transfer

○ An additional dose of 15mg morphine 30 minutes prior to his transfer

Submit answer

Reference ranges

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Question 32 of 43

A 68-year-old man with metastatic prostate cancer is due to go for radiotherapy in an hour. The nurses tell you that he complained of pain during the transfer yesterday. His pain has been otherwise well controlled on a total of 60mg morphine daily. How is it best to manage his pain control for the transfer today?

Nothing as it is only temporary pain	2%
An additional dose of 10mg morphine 30 minutes prior to his transfer	68%
An additional dose of 15mg morphine 1 hour prior to his transfer	3%
An additional dose of 10mg morphine 1 hour prior to his transfer	22%
An additional dose of 15mg morphine 30 minutes prior to his transfer	4%

A breakthrough dose of morphine should be given 30 minutes prior to his transfer as indicated in the BNF. 1 hour is too long prior as it should last him the transfer back and 30 minutes prior is enough for the morphine to have the desired effect. A breakthrough dose should be 1/6th of his total daily dose which in this case is 10mg. Therefore, 15mg is too high a dose for this patient.

Although it is temporary pain, it should still be treated as the patient's comfort and quality of life are important aspects of his care.

Discuss (3) Improve

Next question

Palliative care prescribing: pain

NICE guidelines

In 2012 NICE published guidelines on the use of opioids in palliative care. Selected points are listed below. Please see the link for more details.

Starting treatment
- when starting treatment, offer patients with advanced and progressive disease regular oral modified-release (MR) or oral immediate-release morphine (depending on patient

preference), with oral immediate-release morphine for breakthrough pain

- if no comorbidities use 20-30mg of MR a day with 5mg morphine for breakthrough pain. For example, 15mg modified-release morphine tablets twice a day with 5mg of oral morphine solution as required
- oral modified-release morphine should be used in preference to transdermal patches
- laxatives should be prescribed for all patients initiating strong opioids
- patients should be advised that nausea is often transient. If it persists then an antiemetic should be offered
- drowsiness is usually transient - if it does not settle then adjustment of the dose should be considered

SIGN guidelines

SIGN issued guidance on the control of pain in adults with cancer in 2008. Selected points

- the breakthrough dose of morphine is one-sixth the daily dose of morphine
- all patients who receive opioids should be prescribed a laxative
- opioids should be used with caution in patients with chronic kidney disease
  - oxycodone is preferred to morphine in palliative patients with mild-moderate renal impairment
  - if renal impairment is more severe, alfentanil, buprenorphine and fentanyl are preferred
- metastatic bone pain may respond to strong opioids, bisphosphonates or radiotherapy. The assertion that NSAIDs are particularly effective for metastatic bone pain is not supported by studies. Strong opioids have the lowest number needed to treat for relieving the pain and can provide quick relief, in contrast to radiotherapy and bisphosphonates*. All patients, however, should be considered for referral to a clinical oncologist for consideration of further treatments such as radiotherapy

Other points

When increasing the dose of opioids the next dose should be increased by 30-50%.

In addition to strong opioids, bisphosphonates and radiotherapy, denosumab may be used to treat metastatic bone pain.

Opioid side-effects

Usually transient	**Usually persistent**
Nausea Drowsiness	Constipation

Conversion between opioids

From	To	Conversion factor
Oral codeine	Oral morphine	Divide by 10
Oral tramadol	Oral morphine	Divide by 10**

Oxycodone generally causes less sedation, vomiting and pruritis than morphine but more constipation.

From	To	Conversion factor
Oral morphine	Oral oxycodone	Divide by 1.5-2***

The current BNF gives the following conversion factors for transdermal perparations

- a transdermal fentanyl 12 microgram patch equates to approximately 30 mg oral morphine daily
- a transdermal buprenorphine 10 microgram patch equates to approximately 24 mg oral morphine daily.

From	To	Conversion factor
Oral morphine	Subcutaneous morphine	Divide by 2
Oral morphine	Subcutaneous diamorphine	Divide by 3
Oral oxycodone	Subcutaneous diamorphine	Divide by 1.5

*BMJ 2015;350:h315 Cancer induced bone pain

**this has previously been stated as 5 but the current version of the BNF states a conversion of 10

***historically a conversion factor of 2 has been used (i.e. oral oxycodone is twice as strong as oral morphine). The current BNF however uses a conversion rate of 1.5

123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

NICE 👍 3 👎 1

2012 Opioids in palliative care guidelines

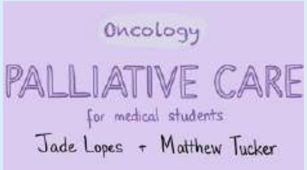
BNF 👍 3 👎 3

Prescribing opioids in palliative care

Suggest link

Report broken link

Media


Oncology
PALLIATIVE CARE
for medical students
Jade Lopes + Matthew Tucker

Complete Guide to Palliative Care for Medical Students

MedFlix - YouTube 👍 20 👎 2

HEART ATTACK
REFERRED PAIN
ACUTE PAIN
CHRONIC PAIN
PAIN
BRAIN
SPINAL CORD
C FIBRES
A-DELTA FIBRES
RECEPTORS
PERIPHERAL NERVES

Understanding The Causes of Pain and Pain Management

Zero To Finals - YouTube 👍 1 👎 1

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A 72-year-old man presents to the emergency department with shortness of breath. He describes his breathlessness has progressively become worse over the last two weeks and is especially troublesome when leaning forward. His wife has commented that his appearance has changed too, with his face becoming more swollen and different in colour. He has a past medical history of right-sided heart failure, myocardial infarction, type two diabetes, COPD and small cell lung cancer diagnosed three months ago. He is an ex-smoker of 20 pack-years and drinks moderate amounts of alcohol.

On examination, he appears settled at rest. His face is swollen and slightly purple. His neck veins are distended and his JVP is markedly elevated to 4cm. Auscultation reveals bibasal crepitations. There is pitting oedema at the ankles. A chest X-ray demonstrates a widened mediastinum. What investigation is most appropriate to establish the diagnosis?

- ○ ECG
- ○ Trans-thoracic echocardiogram
- ○ Trans-oesophageal echocardiogram
- ○ Bronchoscopy
- ○ CT chest

Submit answer

Reference ranges

Score: **0%**

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ECG	1%
Trans-thoracic echocardiogram	11%
Trans-oesophageal echocardiogram	7%
Bronchoscopy	3%
CT chest	78%

The correct answer is CT chest as the patient has a clinical history consistent with obstruction of the superior vena cava (SVC). There are several symptoms and signs consistent with SVC obstruction, including breathlessness worse, leaning forward, distended neck veins and a swollen face. This, in combination with known lung cancer, suggests progression of the primary malignancy or lymph nodes compressing the SVC. Whilst some of the finds could be suggestive of acute pulmonary oedema, the chest X-ray is consistent with SVC obstruction. As a cardiac cause is unlikely, an echocardiogram or ECG is unlikely to be diagnostic. A bronchoscopy is a possible approach for obtaining a biopsy if an SVC obstruction is diagnosed and the source of the obstruction is unclear, but as this patient is known to have lung cancer this is not going to be helpful for this patient.

Source:

Drews, Reed E., and Dmitry J. Rabkin. 'Malignancy-related Superior Vena Cava Syndrome.' UpToDate. N.p., 2 Mar. 2016.

Discuss (1) Improve

Next question

Superior vena cava obstruction

Superior vena cava (SVC) obstruction is an oncological emergency caused by compression of the SVC. It is most commonly associated with lung cancer.

Features
- dyspnoea is the most common symptom
- swelling of the face, neck and arms - conjunctival and periorbital oedema may be seen
- headache: often worse in the mornings
- visual disturbance
- pulseless jugular venous distension

Causes
- common malignancies: small cell lung cancer, lymphoma
- other malignancies: metastatic seminoma, Kaposi's sarcoma, breast cancer
- aortic aneurysm
- mediastinal fibrosis
- goitre
- SVC thrombosis

Management is dependant on the individual patient and malignancy and advice should be taken from the oncology team. Options include:
- endovascular stenting is often the treatment of choice to provide symptom relief
- certain malignancies such as lymphoma, small cell lung cancer may benefit from radical chemotherapy or chemo-radiotherapy rather than stenting
- the evidence base supporting the use of glucocorticoids is weak but they are often given

Next question

Textbooks

High-yield textbook

Extended textbook

Media

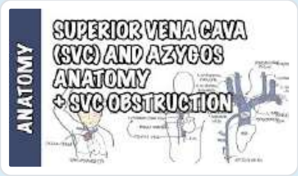
SVC SYNDROME

Superior Vena Cava Syndrome in 3 Minutes

Townsend Teaching - YouTube 👍 2 👎 1

Superior vena cava syndrome

Osmosis - YouTube 👍 2 👎 1


ANATOMY
SUPERIOR VENA CAVA (SVC) AND AZYGOS ANATOMY
+ SVC OBSTRUCTION

Superior vena cava and the azygos system clinical anatomy - SVC obstruction (oncology emergency)

Armando Hasudungan - YouTube 👍 1 👎 2

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Score: **9.3%**

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A 40-year-old man is seen in the Emergency Department with breathlessness.

He was diagnosed with non-Hodgkin lymphoma 6 months ago and has been receiving chemotherapy. He is awaiting an appointment in haematology outpatients to assess his response to chemotherapy.

He describes becoming more breathless over the last week and feeling that his arms and face are puffy. He has had no chest pain, cough or fevers.

On examination his oxygen saturations are 92% on air and his heart rare is 112 beats per minute. His chest is clear and he is unable to lay flat for his abdomen to be examined due to is breathlessness. His face appears flushed and he has oedema of the arms and hands. His neck veins appear engorged.

Chest x-ray shows a widened mediastinum with extensive lymphadenopathy.

A CT scan of the thorax confirms superior vena cava obstruction (SVCO). The scan is reviewed by the haematology team who explain to him that his lymphoma has progressed despite chemotherapy.

He is started on steroids and a proton pump inhibitor.

What is the next step in the management of this gentleman's SVCO?

- ○ Chemotherapy
- ○ Observation
- ○ Radiotherapy
- ○ Stent insertion
- ○ Surgery

Submit answer

Reference ranges

Score: **0%**

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He is started on steroids and a proton pump inhibitor.

What is the next step in the management of this gentleman's SVCO?

Chemotherapy	3%
Observation	1%
Radiotherapy	21%
Stent insertion	71%
Surgery	3%

SVCO is an oncological emergency and is most common in lung cancer and lymphomas. The immediate management is steroids with proton pump inhibitor cover for gastric protection.

Further options include stent insertion, radiotherapy and chemotherapy.

This gentleman is unable to lay flat so would not be able to tolerate radiotherapy. Radiotherapy also has the disadvantage of causing increased swelling acutely before it causes long term shrinkage.

Lymphomas are highly chemotherapy responsive tumours and this gentleman has only had first-line chemotherapy, therefore second-line chemotherapy should be planned in order to shrink his mediastinal disease.

Stent insertion is the most immediate management for relieving SVCO until chemotherapy can take effect. The patient's haemoglobin, platelets and clotting must be checked and optimised. In patients with end-stage disease, stents can still become blocked.

Surgery is not generally part of the management for SVCO.

Reference:
London Cancer Alliance. Acute Oncology Clinical Guidelines. 2013.

👍 👎 Discuss (3) Improve

Next question >

Superior vena cava obstruction ★

Superior vena cava (SVC) obstruction is an oncological emergency caused by compression of the SVC. It is most commonly associated with lung cancer.

Features
- dyspnoea is the most common symptom
- swelling of the face, neck and arms - conjunctival and periorbital oedema may be seen
- headache: often worse in the mornings
- visual disturbance
- pulseless jugular venous distension

Causes
- common malignancies: small cell lung cancer, lymphoma
- other malignancies: metastatic seminoma, Kaposi's sarcoma, breast cancer
- aortic aneurysm
- mediastinal fibrosis
- goitre
- SVC thrombosis

Management is dependant on the individual patient and malignancy and advice should be taken from the oncology team. Options include:

- endovascular stenting is often the treatment of choice to provide symptom relief
- certain malignancies such as lymphoma, small cell lung cancer may benefit from radical chemotherapy or chemo-radiotherapy rather than stenting
- the evidence base supporting the use of glucocorticoids is weak but they are often given

AI + Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Media

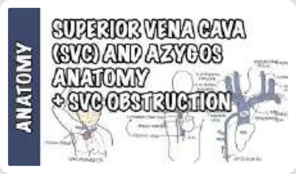
SVC SYNDROME

Superior Vena Cava Syndrome in 3 Minutes

Townsend Teaching - YouTube 👍 2 👎 1

Superior vena cava syndrome

Osmosis - YouTube 👍 2 👎 1


ANATOMY
SUPERIOR VENA CAVA (SVC) AND AZYGOS ANATOMY + SVC OBSTRUCTION

Superior vena cava and the azygos system clinical anatomy - SVC obstruction (oncology emergency)

Armando Hasudungan - YouTube 👍 1 👎 2

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Score: **9.3%**

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Question 35 of 43

A 71-year-old woman is referred by her GP to the rapid access chest clinic with progressive shortness of breath and reduced exercise tolerance over 3 months and a chronic productive cough. When asked about her past medical history she states that she has had some pain for the past month or so in her left shoulder, which has been present at a constant level. She has been taking paracetamol and codeine for the pain which helps slightly. Otherwise, the patient has moderate COPD, for which she taken ipratropium bromide and salmeterol and mechanical back pain. She has a 15 year pack-history and drinks 1-2 units of alcohol per day.

Examination reveals some wasting of the hypothenar eminence of the left hand, although the chest sounds clear bilaterally with no added sounds. Heart sounds 1 and 2 are present with no murmurs.

What would be the most useful investigation following a chest x-ray?

- ○ Electromyogram studies
- ○ Computed tomography of the thorax
- ○ Bronchoscopy
- ○ Myeloma screen
- ○ Sputum microscopy, culture and sensitivity

Submit answer

Reference ranges

Score: **0%**

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Question 35 of 43

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Examination reveals some wasting of the hypothenar eminence of the left hand, although the chest sounds clear bilaterally with no added sounds. Heart sounds 1 and 2 are present with no murmurs.

What would be the most useful investigation following a chest x-ray?

Electromyogram studies	6%
Computed tomography of the thorax	81%
Bronchoscopy	5%
Myeloma screen	6%
Sputum microscopy, culture and sensitivity	2%

The most likely diagnosis in this case is lung malignancy, in particular a Pancoast tumour, given this patient's wasting of the hypothenar eminence, shortness of breath and cough. The most useful investigation is a CT of the thorax - bronchoscopy is not as useful as the cancer is often too peripheral.

Discuss (3) Improve

Next question

Lung cancer: non-small cell

There are three main subtypes of non-small cell lung cancer:

Squamous cell cancer

- typically central
- associated with parathyroid hormone-related protein (PTHrP) secretion → hypercalcaemia
- strongly associated with finger clubbing
- cavitating lesions are more common than other types
- hypertrophic pulmonary osteoarthropathy (HPOA)

Adenocarcinoma

- typically peripheral
- most common type of lung cancer in non-smokers, although the majority of patients who develop lung adenocarcinoma are smokers

Large cell lung carcinoma

- typically peripheral
- anaplastic, poorly differentiated tumours with a poor prognosis
- may secrete β-hCG

+ Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

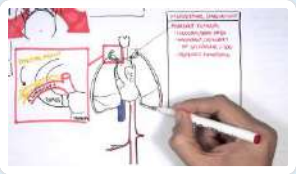
Links

Radiopaedia 👍 4 👎 3

Lung cancer review

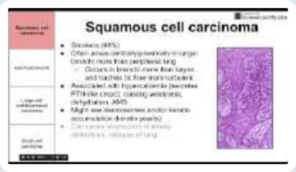
Suggest link Report broken link

Media



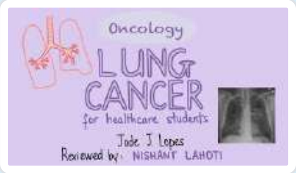
Lung cancer

Armando Hasudungan - YouTube 👍 1 👎 0


Squamous cell carcinoma

Cancers of the lung

MedLecturesMadeEasy - YouTube 👍 1 👎 0


Oncology
LUNG CANCER
for healthcare students
Jade J Lopes
Reviewed by: NISHANT LAHOTI

Lung cancer

MedFlix - YouTube 👍 0 👎 0

Oncology
For Med Students
LUNG CANCER:
An Overview

Lung Cancer: An Overview

Oncology for Medical Students - YouTube 👍 0 👎 0

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Score: **9.3%**

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You are asked to review a 67-year-old gentleman who was admitted to the medical emergency unit with pneumonia. He had been diagnosed with terminal small cell cancer of the lung which had extensively metastasised to his liver, thoracic vertebrae and femur bones. His past medical history was comprised of ischaemic heart disease, hypertension, hypercholesterolaemia and COPD.

His main complaint was of progressively worsening pain in his spine and legs. This was particularly bothersome at night; a trial of bisphosphonate therapy and radiotherapy did not provide any symptomatic relief. He was commenced initially on oral morphine sulphate solution which was converted to morphine sulphate tablets (MST); he had been on 60mg BD but this was titrated by the palliative care community nurse to 70mg BD a couple of days prior to admission with excellent analgesic effect. He was also using Oramorph solution 10mg on average 5-6 times a day for breakthrough pain.

He had been accordingly treated with intravenous antibiotics whilst on the ward making a full recovery. Prior to discharge, he was very keen on commencing transdermal treatment so as to minimise the number of oral medications taken.

What is the most appropriate starting dose to commence, with a view to completely discontinuing all oral opiate medication?

○ Commence Fentanyl 12 72 hr patch, paracetamol 1g QDS and oramorph solution PRN for breakthrough pain

○ Commence Fentanyl 25 72 hr patch, paracetamol 1g QDS and oramorph solution PRN for breakthrough pain

○ Commence Fentanyl 50 72 hr patch, paracetamol 1g QDS and oramorph solution PRN for breakthrough pain

○ Commence Fentanyl 75 72 hr patch, paracetamol 1g QDS and oramorph solution PRN for breakthrough pain

○ Commence Fentanyl 100 72 hr patch, paracetamol 1g QDS and oramorph solution PRN for breakthrough pain

Submit answer

Reference ranges

Score: **0%**

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What is the most appropriate starting dose to commence, with a view to completely discontinuing all oral opiate medication?

- Commence Fentanyl 12 72 hr patch, paracetamol 1g QDS and oramorph solution PRN for breakthrough pain 9%
- Commence Fentanyl 25 72 hr patch, paracetamol 1g QDS and oramorph solution PRN for breakthrough pain 17%
- Commence Fentanyl 50 72 hr patch, paracetamol 1g QDS and oramorph solution PRN for breakthrough pain 30%
- Commence Fentanyl 75 72 hr patch, paracetamol 1g QDS and oramorph solution PRN for breakthrough pain 30%
- Commence Fentanyl 100 72 hr patch, paracetamol 1g QDS and oramorph solution PRN for breakthrough pain 13%

This gentleman was on a rather complicated opiate analgesia regimen and the key to this question is calculate the total daily dose of morphine salt, to then convert it to the appropriate amount of fentanyl.

His total morphine salt intake was thus:

MST 70mg BD = 140mg of morphine
Oramorph 10mg PRN = 50-60mg of morphine

Total = 190-200mg daily morphine salt intake

The BNF states that a fentanyl 75 patch is equivalent to 180mg daily intake of morphine salt whilst a fentanyl 100patch is equivalent to 240mg daily morphine salt intake. So as to avoid opiate toxicity the correct answer is thus to commence a fentanyl 75 patch and to supplement this with PRN Oramorph solution.

👍 👎 Discuss (6) Improve

Next question >

Palliative care prescribing: pain ★

NICE guidelines

In 2012 NICE published guidelines on the use of opioids in palliative care. Selected points are listed below. Please see the link for more details.

Starting treatment
- when starting treatment, offer patients with advanced and progressive disease regular oral modified-release (MR) or oral immediate-release morphine (depending on patient preference), with oral immediate-release morphine for breakthrough pain
- if no comorbidities use 20-30mg of MR a day with 5mg morphine for breakthrough pain. For example, 15mg modified-release morphine tablets twice a day with 5mg of oral morphine solution as required
- oral modified-release morphine should be used in preference to transdermal patches
- laxatives should be prescribed for all patients initiating strong opioids
- patients should be advised that nausea is often transient. If it persists then an antiemetic should be offered
- drowsiness is usually transient - if it does not settle then adjustment of the dose should be considered

SIGN guidelines

SIGN issued guidance on the control of pain in adults with cancer in 2008. Selected points
- the breakthrough dose of morphine is one-sixth the daily dose of morphine
- all patients who receive opioids should be prescribed a laxative
- opioids should be used with caution in patients with chronic kidney disease

  - oxycodone is preferred to morphine in palliative patients with mild-moderate renal impairment
  - if renal impairment is more severe, alfentanil, buprenorphine and fentanyl are preferred
- metastatic bone pain may respond to strong opioids, bisphosphonates or radiotherapy. The assertion that NSAIDs are particularly effective for metastatic bone pain is not supported by studies. Strong opioids have the lowest number needed to treat for relieving the pain and can provide quick relief, in contrast to radiotherapy and bisphosphonates*. All patients, however, should be considered for referral to a clinical oncologist for consideration of further treatments such as radiotherapy

Other points

When increasing the dose of opioids the next dose should be increased by 30-50%.

In addition to strong opioids, bisphosphonates and radiotherapy, denosumab may be used to treat metastatic bone pain.

Opioid side-effects

Usually transient	Usually persistent
Nausea Drowsiness	Constipation

Conversion between opioids

From	To	Conversion factor
Oral codeine	Oral morphine	Divide by 10
Oral tramadol	Oral morphine	Divide by 10**

Oxycodone generally causes less sedation, vomiting and pruritis than morphine but more constipation.

From	To	Conversion factor
Oral morphine	Oral oxycodone	Divide by 1.5-2***

The current BNF gives the following conversion factors for transdermal perparations

- a transdermal fentanyl 12 microgram patch equates to approximately 30 mg oral morphine daily
- a transdermal buprenorphine 10 microgram patch equates to approximately 24 mg oral morphine daily.

From	**To**	**Conversion factor**
Oral morphine	Subcutaneous morphine	Divide by 2
Oral morphine	Subcutaneous diamorphine	Divide by 3
Oral oxycodone	Subcutaneous diamorphine	Divide by 1.5

*BMJ 2015;350:h315 Cancer induced bone pain

**this has previously been stated as 5 but the current version of the BNF states a conversion of 10

***historically a conversion factor of 2 has been used (i.e. oral oxycodone is twice as strong as oral morphine). The current BNF however uses a conversion rate of 1.5

+ Q 123

Next question >

B I A

Textbooks

High-yield textbook

Extended textbook

Links

NICE 👍 3 👎 1

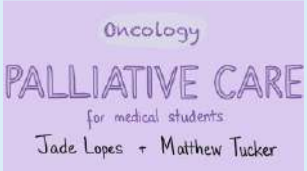
2012 Opioids in palliative care guidelines

BNF 👍 3 👎 3

Prescribing opioids in palliative care

Suggest link Report broken link

Media


Oncology
PALLIATIVE CARE
for medical students
Jade Lopes + Matthew Tucker

Complete Guide to Palliative Care for Medical Students

MedFlix - YouTube 👍 20 👎 2

HEART ATTACK
REFERRED PAIN
ACUTE PAIN
CHRONIC PAIN
PAIN
BRAIN
SPINAL CORD
C FIBRES
A-DELTA FIBRES
RECEPTORS
PERIPHERAL NERVES

Understanding The Causes of Pain and Pain Management

Zero To Finals - YouTube 👍 1 👎 1

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Score: **9.3%**

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A 68-year-old man presents with a five-week history of lower back pain that is worse with coughing. He has noticed that he has been requiring more painkillers to the point where he is struggling to manage, especially at night. The pain is worse when straining to pass stool. He lost three kg over the last four months as well. He has a past medical history of COPD, hypertension, type two diabetes mellitus, and hypercholesterolemia. He has been admitted three times in the last year for exacerbations of COPD requiring the recurrent use of oral prednisolone. His regular medications include Symbicort, tiotropium, atorvastatin, metformin, paracetamol, codeine and Movicol. He is an ex-smoker with a 50 pack-year history and recently opted for early retirement from his job as an accountant as he was struggling with tiredness. On examination, he appears tired and his clothes appear loose fitting. Examination of his chest is unremarkable, but there is a 1cm swelling above his left clavicle. His abdomen is soft, there is no tenderness over his spine, and a neurological examination is unremarkable.

Observations:

Saturations	95%
Respiratory rate	15/min
Blood pressure	148/87mmHg
Heart rate	74/min
Temperature	37.4°C

Blood tests:

Hb	128 g/l
MCV	72 fl
Platelets	327 * 10^9/l
WBC	12.4 * 10^9/l
Na^+	136 mmol/l
K^+	4.2 mmol/l
Urea	4.1 mmol/l
Creatinine	77 µmol/l

What is the most likely diagnosis that explains his symptoms?

○ Osteoporotic vertebral collapse ×

○ Spinal cord metastasis ×

○ Myeloma ×

○ Degenerative changes ×

○ Discitis ×

Submit answer

Reference ranges ∨

Score: **0%**

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Urea	4.1 mmol/l
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What is the most likely diagnosis that explains his symptoms?

Osteoporotic vertebral collapse 8%

Spinal cord metastasis 80%

Myeloma	7%
Degenerative changes	1%
Discitis	4%

The answer is spinal cord metastasis. This question highlights multiple subtle red flags in a history with back pain. He is over >55 and describes nocturnal and progressive pain worse with coughing and straining in the context of immunosuppression from repeated steroid use, weight loss, and increasing fatigue. Due to the high suspicion, the most diagnostic imaging investigation would be a whole spine MRI, which would differentiate between the above diagnoses. Osteoporotic fracture is suggested by the use of steroids but overall does not fit with the progressive nature and microcytic anaemia, nor do degenerative changes. There are very few symptoms to suggest infection such as fever and night sweats, but discitis cannot be fully excluded without imaging. Myeloma is a possible diagnosis, but so far there are no specific factors to suggest it, such as lytic bone lesions or deteriorating renal function.

Discuss (6) Improve

Next question ›

Spinal metastases ★

Patients may present with spinal metastases before developing metastatic spinal cord compression. It is, therefore, important to detect these patients early before any neurological compromise develops.

Symptoms and findings
- Unrelenting lumbar back pain
- Any thoracic or cervical back pain
- Worse with sneezing, coughing or straining
- Nocturnal
- Associated with tenderness

If any neurological features are present then spinal cord compression must be suspected and acted on promptly. Without neurological features, a whole spine MRI should be completed within one week. The whole spine should be imaged as patients commonly present with multi-level disease.

Sources:
Al-Qurainy, Rasha, and Emily Collis. 'Metastatic Spinal Cord Compression: Diagnosis and Management.' Bmj (2016): I2539.

Metastatic Spinal Cord Compression in Adults: Risk Assessment, Diagnosis and Management Clinical Guideline [CG75]. National Institute of Clinical Excellence. N.p., Nov. 2008.

AI + Q 123

Next question >

B I A

Textbooks

High-yield textbook

Extended textbook

Score: **9.3%**

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A 56-year-old male with non-hodgkin's lymphoma is on his third cycle of R-CHOP chemotherapy. He has struggled previous times with nausea but is two days following the most recent cycle and attended hospital due to intractable vomiting.

He is vomiting 20+ times a day with green to clear fluid. He does not feel hungry and struggles to swallow water. He is not having diarrhoea. He takes regular indapamide and as required ondansetron and this has not helped him despite the oncologist suggesting he double his dose of ondansetron.

On examination, he looks pale and has dry mucosa. He is alert, has a clear chest and soft abdomen but begins to retch on sitting forward. He last passed urine 6 hours ago and his observations show a heart rate of 115 beats per minute and blood pressure 90/65mmHg. He is started on intravenous fluids by the emergency department.

Na^+	135 mmol/l
K^+	3.5 mmol/l
Urea	6.5 mmol/l
Creatinine	98 µmol/l

What medical intervention would offer greatest benefit?

- ○ Cyclizine
- ○ Potassium salt replacement
- ○ Elective terminal ileostomy
- ○ Dexamethasone
- ○ Delay next course of chemotherapy

Submit answer

Reference ranges

Score: **0%**

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Na^+	135 mmol/l
K^+	3.5 mmol/l
Urea	6.5 mmol/l
Creatinine	98 µmol/l

What medical intervention would offer greatest benefit?

Cyclizine	17%
Potassium salt replacement	2%
Elective terminal ileostomy	1%
Dexamethasone	76%
Delay next course of chemotherapy	5%

Dexamethasone with ondansetron is effective in refractory chemotherapy-induced vomiting

Important for me Less important

This gentleman has refractory vomiting despite ondansetron. He need assistance to ensure he can maintain fluids. Cyclizine is an antiemetic but bind histamine receptors so is most useful in labyrinthitis and motion sickness. Potassium salt replacement will probably make him vomit more but does replace his lost salts. Delaying the course of chemotherapy would be an option if nothing else could be offered. Surgery is not the way forward here. Dexamethasone is proven as an effective second line antiemetic in chemotherapy related nausea and vomiting. it is often given in lymphoma to prevent tumour lysis syndrome.

Discuss (6) Improve

Next question ›

Chemotherapy side-effects: nausea and vomiting ★

Nausea and vomiting are common side-effects of chemotherapy. Risk factors for the development of symptoms include:

- anxiety
- age less than 50 years old
- concurrent use of opioids
- the type of chemotherapy used

For patients at low-risk of symptoms then drugs such as metoclopramide may be used first-line. For high-risk patients then 5HT3 receptor antagonists such as ondansetron are often effective, especially if combined with dexamethasone

AI + Q 123

Next question ›

B I A T↕

Textbooks

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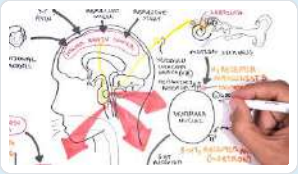
Media

Physiology of Vomiting - Vomiting reflex

Armando Hasudungan - YouTube 👍 3 👎 0



Pharmacology - Antiemetics

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Score: **9.3%**

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A 38-year-old man presents with increasing shortness of breath and ankle swelling. He reports he is more short of breath, especially on exertion, which is not being relieved by the use of his salbutamol inhaler. He has also noticed his ankles are swollen by the end of the day.

His past medical history includes asthma, which is usually well controlled, and lymphoma, which was treated with chemotherapy several years ago. His only medications are his beclometasone and salbutamol inhalers. The results of his blood tests are shown below:

Hb	140 g/L	(135-180)
Platelets	359 * 10^9/L	(150 - 400)
WBC	7.2 * 10^9/L	(4.0 - 11.0)
Na^+	137 mmol/L	(135 - 145)
K^+	4.3 mmol/L	(3.5 - 5.0)
Urea	5.9 mmol/L	(2.0 - 7.0)
Creatinine	119 µmol/L	(55 - 120)

His ECG showed sinus rhythm with left axis deviation. An echocardiogram was done which showed dilation of all four chambers of the heart and a left ventricular ejection fraction of 40%.

Which drug can be attributed to causing this man's presentation?

- ○ Cyclophosphamide
- ○ Vincristine
- ○ Doxorubicin
- ○ Docetaxel
- ○ Bleomycin

Submit answer

Reference ranges

Score: **0%**

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Urea	5.9 mmol/L	(2.0 - 7.0)
Creatinine	119 µmol/L	(55 - 120)

His ECG showed sinus rhythm with left axis deviation. An echocardiogram was done which showed dilation of all four chambers of the heart and a left ventricular ejection fraction of 40%.

Which drug can be attributed to causing this man's presentation?

Cyclophosphamide	1%
Vincristine	2%
Doxorubicin	88%
Docetaxel	4%
Bleomycin	5%

Anthracyclines (e.g. doxorubicin) may cause cardiomyopathy

Important for me Less important

This man is presenting with features of heart failure and the echocardiogram results would be in keeping with a diagnosis of dilated cardiomyopathy. Doxorubicin (an anthracycline) is used in the treatment of lymphoma and is known to be cardiotoxic.

Cyclophosphamide can also be used to treat lymphoma and other haematological conditions such as leukaemia. A well-known side effect is haemorrhagic cystitis.

Vincristine can be used with doxorubicin to treat lymphoma and is associated with peripheral neuropathy.

Docetaxel is often used in the management of breast and lung cancers and can cause neutropenia.

Bleomycin can be used as part of lymphoma management and is associated with the development of pulmonary fibrosis.

👍 👎 Discuss (2) Improve

Next question >

Cytotoxic agents ★

The tables below summarises the mechanism of action and major adverse effects of commonly used cytotoxic agents.

Alkylating agents

Cytotoxic	**Mechanism of action**	**Adverse effects**
Cyclophosphamide	Alkylating agent - causes cross-linking in DNA	Haemorrhagic cystitis, myelosuppression, transitional cell carcinoma

Cytotoxic antibiotics

Cytotoxic	**Mechanism of action**	**Adverse effects**
Bleomycin	Degrades preformed DNA	Lung fibrosis
Anthracyclines (e.g doxorubicin)	Stabilizes DNA-topoisomerase II complex inhibits DNA & RNA synthesis	Cardiomyopathy

Antimetabolites

Cytotoxic	Mechanism of action	Adverse effects
Methotrexate	Inhibits dihydrofolate reductase and thymidylate synthesis	Myelosuppression, mucositis, liver fibrosis, lung fibrosis
Fluorouracil (5-FU)	Pyrimidine analogue inducing cell cycle arrest and apoptosis by blocking thymidylate synthase (works during S phase)	Myelosuppression, mucositis, dermatitis
6-mercaptopurine	Purine analogue that is activated by HGPRTase, decreasing purine synthesis	Myelosuppression
Cytarabine	Pyrimidine antagonist. Interferes with DNA synthesis specifically at the S-phase of the cell cycle and inhibits DNA polymerase	Myelosuppression, ataxia

Acts on microtubules

Cytotoxic	Mechanism of action	Adverse effects
Vincristine, vinblastine	Inhibits formation of microtubules	Vincristine: Peripheral neuropathy (reversible) , paralytic ileus Vinblastine: myelosuppression
Docetaxel	Prevents microtubule depolymerisation & disassembly, decreasing free tubulin	Neutropaenia

Topoisomerase Inhibitors

Cytotoxic	Mechanism of action	Adverse effects
Irinotecan	Inhibits topoisomerase I which prevents relaxation of supercoiled DNA	Myelosuppression

Other cytotoxic drugs

Cytotoxic	Mechanism of action	Adverse effects
Cisplatin	Causes cross-linking in DNA	Nephrotoxicity (the primary dose-limiting side effect), Ototoxicity, peripheral neuropathy, hypomagnesaemia
Hydroxyurea (hydroxycarbamide)	Inhibits ribonucleotide reductase, decreasing DNA synthesis	Myelosuppression

123

Next question

Textbooks

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Links

Toxicity Bear's very own website

339

17

Toxicity Bear

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Media

DNA REPLICATION

Pharmacology - Chemotherapy agents

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CANCER
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Cancer
Drugs

Cancer Drugs - Learn with Visual Mnemonics!

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Score: **9.3%**

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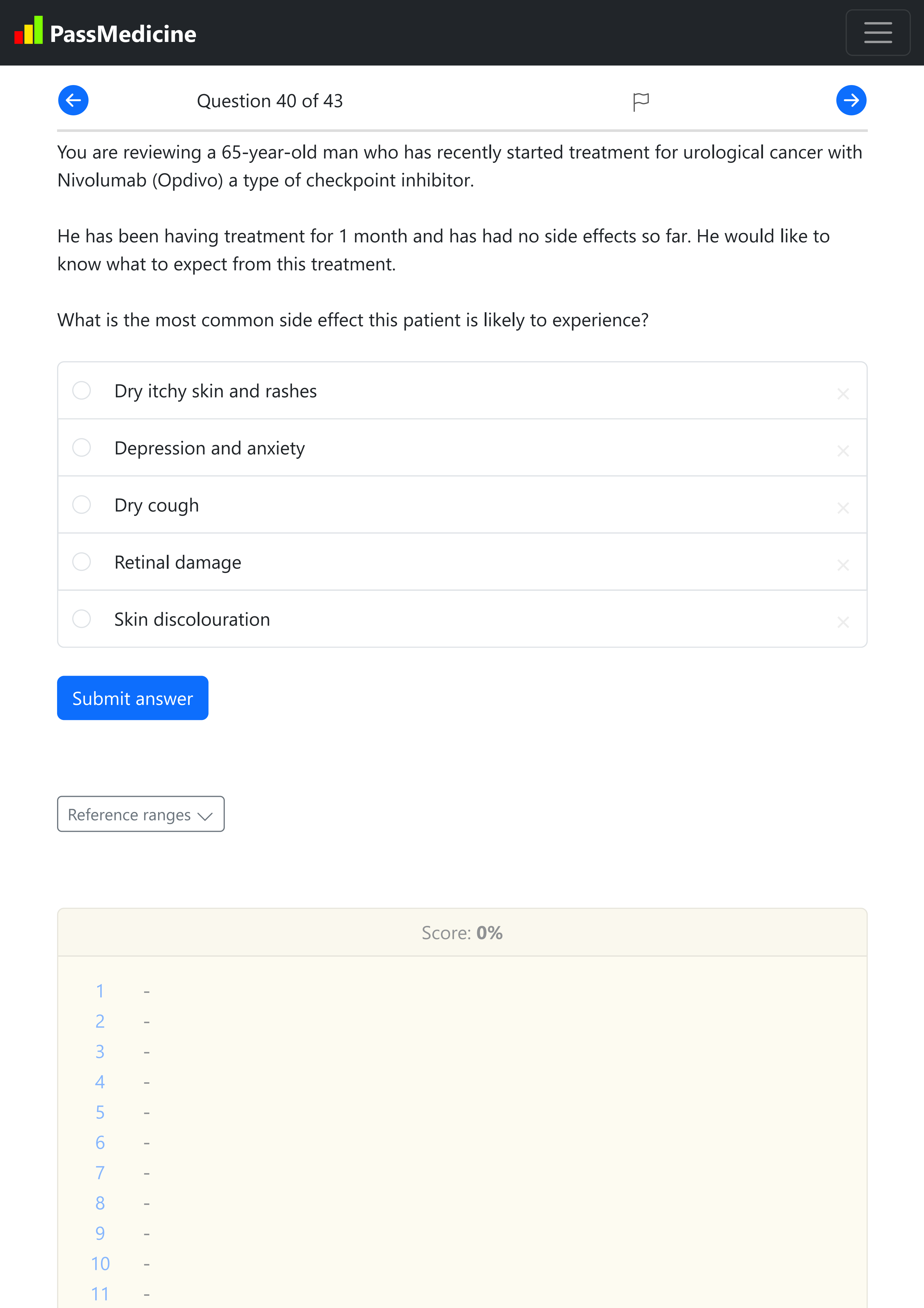

Question 40 of 43

You are reviewing a 65-year-old man who has recently started treatment for urological cancer with Nivolumab (Opdivo) a type of checkpoint inhibitor.

He has been having treatment for 1 month and has had no side effects so far. He would like to know what to expect from this treatment.

What is the most common side effect this patient is likely to experience?

- ○ Dry itchy skin and rashes
- ○ Depression and anxiety
- ○ Dry cough
- ○ Retinal damage
- ○ Skin discolouration

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Question 40 of 43

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He has been having treatment for 1 month and has had no side effects so far. He would like to know what to expect from this treatment.

What is the most common side effect this patient is likely to experience?

Dry itchy skin and rashes	57%
Depression and anxiety	14%
Dry cough	8%
Retinal damage	9%
Skin discolouration	11%

The most common side effect of immune checkpoint inhibitors is dry itchy skin and rashes

Important for me Less important

Immune checkpoint inhibitors are a type of immunotherapy that are increasingly being used to treat certain types of cancer, as an alternative to cytotoxic chemotherapy. They are currently only used to treat solid tumours. They work by enhancing the bodies own T-cell population to destroy the cancer cells.

Their side effect profile reflects their mechanism of action and are, therefore, inflammatory and autoimmune in nature. They include:

- Dry, itchy skin and rashes (most commonly)
- Nausea and vomiting
- Decreased appetite
- Diarrhoea
- Tiredness and fatigue
- Shortness of breath and a dry cough.

Therefore, option one is the only correct answer.

Immune checkpoint inhibitors ★

Immune checkpoint inhibitors are a type of immunotherapy that are increasingly being used to treat certain types of cancer, as an alternative to cytotoxic chemotherapy. In contrast to therapies such as chemotherapy and tumour-targeted drugs that directly affect the growth and proliferation of tumour cells, these targeted treatments harness the body's natural anti-cancer immune response. They boost its ability to attack and destroy the cancer cells.

T-cells are an important part of our immune system which help destroy cancer cells. Some cancer cells make high levels of proteins that turn T-cells off. Checkpoint inhibitors block this process and reactivate and increase the body's own T-cell population, enhancing the immune systems own ability to recognise and fight cancer cells.

Types:

- Ipilimumab (Yervoy) is a checkpoint inhibitor that blocks CTLA-4 (cytotoxic T lymphocyte-associated protein 4). It is a treatment for advanced melanoma.
- Nivolumab (Opdivo) and pembrolizumab (Keytruda) blocks PD-1 (programmed cell death protein 1). These are treatments for melanoma, Hodgkin's lymphoma, non-small cell lung cancer and urological cancers.
- Atezolizumab, Avelumab and Durvalumab block PD-L1 and is used to treat lung cancer and urothelial cancer. It is also undergoing trials as a treatment for breast cancer.

The proteins CTLA-4 and PD-1 are found on T-cells and PD-L1 are found on cancer cells.

Administration

All CTLA-4 inhibitors and PD-1/PD-L1 inhibitors currently available are monoclonal antibodies (laboratory-produced molecules engineered to serve as substitute antibodies that each bind to a specific protein). They are all administered by injection/intravenous infusion. Mostly, they are given as a single-agent treatment but sometimes, they can be combined with chemotherapy or each other. For example, ipilimumab and nivolumab can be used in combination.

Side effects

This mechanism of action of these drugs can result in side effects termed 'Immune-related adverse events' that are inflammatory and autoimmune in nature. This is because all immune cells are boosted by these drugs, not just the ones that target cancer. The over-active T-cells can produce side effects such as:

- Dry, itchy skin and rashes (most commonly)
- Nausea and vomiting
- Decreased appetite
- Diarrhoea
- Tiredness and fatigue
- Shortness of breath and a dry cough.

Management of such side effects reflects the inflammatory nature, often involving corticosteroids.

It is important to monitor liver, kidney and thyroid function as these drugs can affect these organs.

The future

The early success of immune checkpoint inhibitors in solid tumours has generated a tremendous interest in further developing and exploring these strategies across the oncology disease spectrum. Their ongoing testing in clinical trials creates new hope for patients affected by other types of disease.

+ Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Score: **9.3%**

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Question 41 of 43

A 54-year-old male presents to the emergency department with sudden onset palpitations and shortness of breath which began whilst he was watching television a few hours previously. He describes an odd fluttering sensation in his chest with mild dyspnoea but no chest pain. He denies having experienced this before. On systems review, he also complains of severe muscle cramps and a slight tremor in his hands over the last few days. His past medical history includes hypertension and gout for which he takes amlodipine and allopurinol. He denies any history of exertional dyspnoea but does occasionally get central chest pain on exertion for which he has not sought help. He has recently been diagnosed with small cell lung cancer and underwent his first round of chemotherapy last week; although he is unsure which drugs are being used. He used to smoke a pipe until his recent diagnosis of cancer and drinks a few measures of whisky most evenings.

On examination, his respiratory rate is 22 breaths/min and his oxygen saturations are 96% breathing two litres of oxygen. His heart rate is irregularly irregular and approximately 130 beats/min, his blood pressure is 152/78 mmHg. An ECG shows atrial fibrillation with no ischaemic changes.

What is the most likely cause for his new atrial fibrillation?

- ○ Ischaemic heart disease
- ○ Chemotherapy induced hypokalaemia
- ○ Chemotherapy induced hypomagnesaemia
- ○ Chemotherapy induced cardiomyopathy
- ○ Alcohol

Submit answer

Reference ranges

Score: **0%**

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What is the most likely cause for his new atrial fibrillation?

Ischaemic heart disease	2%
Chemotherapy induced hypokalaemia	14%
✓ Chemotherapy induced hypomagnesaemia	53%
Chemotherapy induced cardiomyopathy	25%
✗ Alcohol	6%

The most common chemotherapy regime for small cell lung cancer is etoposide and cisplatin. A common side effect of cisplatin is hypomagnesaemia which can result in muscle cramps, tremor, cardiac arrhythmias, confusion amongst other features. This gentleman appears to have angina and may well have some level of ischaemic heart disease in the background which would increase his risk of atrial fibrillation. However, it seems logical given the strong temporal relationship between the chemotherapy and the other symptoms of hypomagnesaemia that this is the cause.

Discuss (5) | Improve

Next question >

Cytotoxic agents ★

The tables below summarises the mechanism of action and major adverse effects of commonly used cytotoxic agents.

Alkylating agents

Cytotoxic	**Mechanism of action**	**Adverse effects**
Cyclophosphamide	Alkylating agent - causes cross-linking in DNA	Haemorrhagic cystitis, myelosuppression, transitional cell carcinoma

Cytotoxic antibiotics

Cytotoxic	**Mechanism of action**	**Adverse effects**
Bleomycin	Degrades preformed DNA	Lung fibrosis
Anthracyclines (e.g doxorubicin)	Stabilizes DNA-topoisomerase II complex inhibits DNA & RNA synthesis	Cardiomyopathy

Antimetabolites

Cytotoxic	**Mechanism of action**	**Adverse effects**
Methotrexate	Inhibits dihydrofolate reductase and thymidylate synthesis	Myelosuppression, mucositis, liver fibrosis, lung fibrosis
Fluorouracil (5-FU)	Pyrimidine analogue inducing cell cycle arrest and apoptosis by blocking thymidylate synthase (works during S phase)	Myelosuppression, mucositis, dermatitis
6-mercaptopurine	Purine analogue that is activated by HGPRTase, decreasing purine synthesis	Myelosuppression
Cytarabine	Pyrimidine antagonist. Interferes with DNA synthesis specifically at the S-phase of the cell cycle and inhibits DNA polymerase	Myelosuppression, ataxia

Acts on microtubules

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Cytotoxic	**Mechanism of action**	**Adverse effects**
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Hydroxyurea (hydroxycarbamide)	Inhibits ribonucleotide reductase, decreasing DNA synthesis	Myelosuppression

+ Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

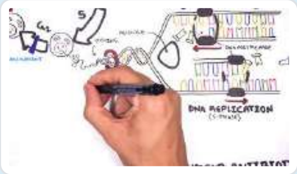
Links

Toxicity Bear's very own website 👍 339 👎 17

Toxicity Bear

Suggest link Report broken link

Media


DNA REPLICATION

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Cancer
Drugs

Cancer Drugs - Learn with Visual Mnemonics!

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Score: **9.3%**

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A 63-year-old man is diagnosed with colorectal cancer. Following successful resection he starts adjuvant capecitabine but after six doses develops severe diarrhoea, weakness and desquamation of the hands, feet and face. On admission, he is pancytopenic and has electrolyte abnormalities requiring IV replacement. His initial blood tests are shown bellow.

Na^+	140 mmol/l
K^+	3.6 mmol/l
Urea	9.2 mmol/l
Creatinine	125 µmol/l
Hb	75 g/l
Platelets	$35 * 10^9/l$
WBC	$0.4 * 10^9/l$
Neutrophils	$0.03 * 10^9/l$
Adjusted Ca^{2+}	1.5 mmol/l
Mg^{2+}	0.32 mmol/l
Glucose	4.0 mmol/l

What is the most likely cause?

○ DPD (dihydropyrimidine dehydrogenase) deficiency

○ Infective gastroenteritis

○ Cancer recurrence

○ Pityriasis alba

○ Neutropenic sepsis

Submit answer

Reference ranges

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Adjusted Ca^{2+}	1.5 mmol/l
Mg^{2+}	0.32 mmol/l
Glucose	4.0 mmol/l

What is the most likely cause?

DPD (dihydropyrimidine dehydrogenase) deficiency	63%
Infective gastroenteritis	3%
Cancer recurrence	1%
Pityriasis alba	2%
Neutropenic sepsis	31%

Capecitabine is the oral analog of 5-fluorouracil, a chemotherapeutic agent which is broken down, predominantly, by dihydropyrimidine dehydrogenase (DPD). Deficiency is autosomal recessive and will lead to a toxin buildup which in homozygous patients is usually fatal. The severe toxicity experienced by this patient are likely due to a heterozygous DPD deficiency.

Discuss (7) | Improve

Next question ›

Cytotoxic agents

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Acts on microtubules

Cytotoxic	Mechanism of action	Adverse effects
Vincristine, vinblastine	Inhibits formation of microtubules	Vincristine: Peripheral neuropathy (reversible) , paralytic ileus Vinblastine: myelosuppression
Docetaxel	Prevents microtubule depolymerisation & disassembly, decreasing free tubulin	Neutropaenia

Topoisomerase Inhibitors

Cytotoxic	Mechanism of action	Adverse effects
Irinotecan	Inhibits topoisomerase I which prevents relaxation of supercoiled DNA	Myelosuppression

Other cytotoxic drugs

Cytotoxic	Mechanism of action	Adverse effects
Cisplatin	Causes cross-linking in DNA	Nephrotoxicity (the primary dose-limiting side effect), Ototoxicity, peripheral neuropathy, hypomagnesaemia
Hydroxyurea (hydroxycarbamide)	Inhibits ribonucleotide reductase, decreasing DNA synthesis	Myelosuppression

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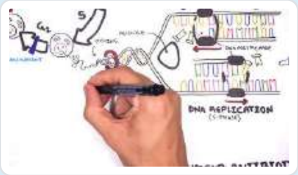
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A 53 year old patient has undergone six cycles of R-CHOP (rituximab, cyclophosphamide, doxorubicin, vincristine and prednisolone) chemotherapy and is now attending the lymphoma follow up clinic. He complains of an ongoing and unrelenting tingling in his feet and legs as well as his right hand. This is associated with pain in the extremities that is worsened by the cold weather.

Examination reveals normal motor strength throughout all 4 limbs but marked reduction in proprioception in both legs below the knee and also the right hand. The is a 'glove and stocking' distribution of loss when tested for pin-prick sensation and a reduced joint position sense to the ankle in both lower limbs. Plantar reflexes are normal. During the examination you it is noted that the patient has sustained an injury to the sole of foot which he was unaware of.

What is the most likely causative agent for these symptoms?

○ Rituximab

○ Cyclophosphamide

○ Doxorubicin

○ Vincristine

○ Prednisolone

Submit answer

Reference ranges

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What is the most likely causative agent for these symptoms?

Rituximab	1%
Cyclophosphamide	3%
Doxorubicin	3%
Vincristine	92%
Prednisolone	1%

The symptoms being experienced above are classical for chemotherapy induced peripheral neuropathy. This is a condition that can occur in up to 40% of patients being given chemotherapy with agents such as platinum based drugs (cisplatin and oxaliplatin), vinca alakloids (vincristine and vinblastine), thalidomide and taxanes (paclitaxel and docetaxel). The treatment of chemotherapy induced peripheral neuropathy is difficult with patients having severe problems long after the causative agent has been given. In most cases it is irreversible. The best medication for treatment is gabapentin or pregabalin which should be titrated up to therapeutic levels and used in conjunction with another analgesic agents.

There are ongoing trials aiming to find a prophylactic agent to help prevent this painful sequealae of treatment, but as yet there has been no convincing evidence to suggest any one treatment.

Discuss (2) Improve

Cytotoxic agents

The tables below summarises the mechanism of action and major adverse effects of commonly used cytotoxic agents.

Alkylating agents

Cytotoxic	**Mechanism of action**	**Adverse effects**
Cyclophosphamide	Alkylating agent - causes cross-linking in DNA	Haemorrhagic cystitis, myelosuppression, transitional cell carcinoma

Cytotoxic antibiotics

Cytotoxic	**Mechanism of action**	**Adverse effects**
Bleomycin	Degrades preformed DNA	Lung fibrosis
Anthracyclines (e.g doxorubicin)	Stabilizes DNA-topoisomerase II complex inhibits DNA & RNA synthesis	Cardiomyopathy

Antimetabolites

Cytotoxic	**Mechanism of action**	**Adverse effects**
Methotrexate	Inhibits dihydrofolate reductase and thymidylate synthesis	Myelosuppression, mucositis, liver fibrosis, lung fibrosis
Fluorouracil (5-FU)	Pyrimidine analogue inducing cell cycle arrest and apoptosis by blocking thymidylate synthase (works during S phase)	Myelosuppression, mucositis, dermatitis
6-mercaptopurine	Purine analogue that is activated by HGPRTase, decreasing purine synthesis	Myelosuppression
Cytarabine	Pyrimidine antagonist. Interferes with DNA synthesis specifically at the S-phase of the cell cycle and inhibits DNA polymerase	Myelosuppression, ataxia

Acts on microtubules

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Alrajhi Jad

PASSMEDICINE
DERMATOLOGY

Internal medicine

EDITED BY
DR. JAMIL ALRAJHI

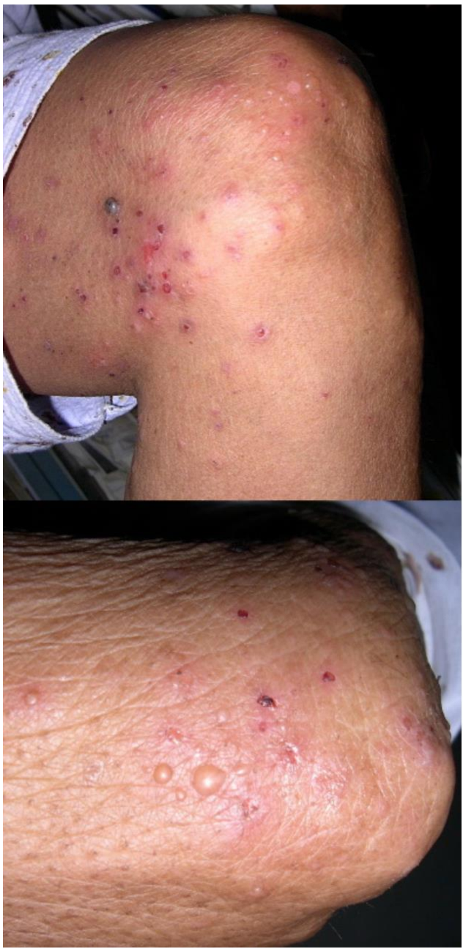
2024

773-555-493
jameelalrajhi@gmail.com

DERMATOLOGY
PASSMEDICINE
PART 2 2024

Question 1 of 182

A 32-year-old woman presents to her GP with a rash of several weeks, across the elbows and knees. She also mentions generalised tiredness. Upon examination, the following rash is noticed:



Blood tests reveal iron-deficiency anaemia.

Given the most likely diagnosis, which of the following is an appropriate first-line option?

A. Oral cetirizine
B. Oral dapsone
C. Oral prednisolone
D. Topical clobetasol
E. Topical tacrolimus

B. Oral dapsone

The diagnosis here is that of dermatitis herpetiformis. The photo above depicts a vesicular rash on an erythematous background, overlying an extensor surface (in this case, a knee). As in this photo, the lesions often appear somewhat excoriated due to the intense pruritus associated with the condition. The question also gives some additional clues by way of the tiredness and irondeficiency anaemia - commonly present in coeliac disease, which is very strongly associated with dermatitis herpetiformis. First-line treatment involves the use of oral dapsone.

Oral cetirizine is inappropriate. Whilst useful for the alleviation of itch, it does not help to resolve the condition. If the diagnosis were instead prickly heat rash or urticaria, cetirizine would be the sole appropriate option.

Oral prednisolone may be used as a second- or third-line option if initial measures fail. However, with regards to initial management, oral dapsone is considered most effective for dermatitis herpetiformis.

Topical clobetasol is a second-line option for dermatitis herpetiformis, not first-line. It is first-line for bullous pemphigoid, which would appear quite differently - tense blisters would be visible, rather than the bullae seen here.

Topical tacrolimus is a steroid-sparing agent sometimes used in the management of steroid-resistant eczema, especially for children and on sensitive areas such as the face. It also plays a role in the management of bullous pemphigoid. It is not routinely used in dermatitis herpetiformis.

Question 2 of 182

A 45-year-old man has been referred to the dermatology clinic for a long-standing facial rash that has not been evaluated before. You note that multiple erythematous papules and pustules are present over his nose and cheeks in a centrofacial distribution. The consultant diagnoses him with moderate to severe papulopustular rosacea.

What is the most appropriate management of this patient?

A. Oral isotretinoin
B. Topical azelaic acid and oral isotretinoin
C. Topical ivermectin
D. Topical ivermectin and oral doxycycline
E. Topical metronidazole and oral isotretinoin

D. Topical ivermectin and oral doxycycline

Rosacea: a combination of topical ivermectin + oral doxycycline is first-line for patients with severe papules and/or pustules

Topical ivermectin and oral doxycycline is the correct answer. Rosacea is a chronic skin disorder which primarily affects the central portion of the face. It is characterised by centrofacial erythema, papules, pustules flushing and telangiectasias. Ocular involvement may also be seen in the form of conjunctiva injection and lid telangiectasias. The management of rosacea is detailed in the notes linked to this question. This patient has been diagnosed with moderate-to-severe papulopustular disease, which is treated with a combination of topical ivermectin and oral doxycycline.

Oral isotretinoin is incorrect. Oral isotretinoin is not used as first-line therapy in rosacea. It is used only when patients fail to respond to topical therapies and oral antibiotics. Since this patient is treatment-naive, he should initially be prescribed the standard topical treatment along with an oral antibiotic.

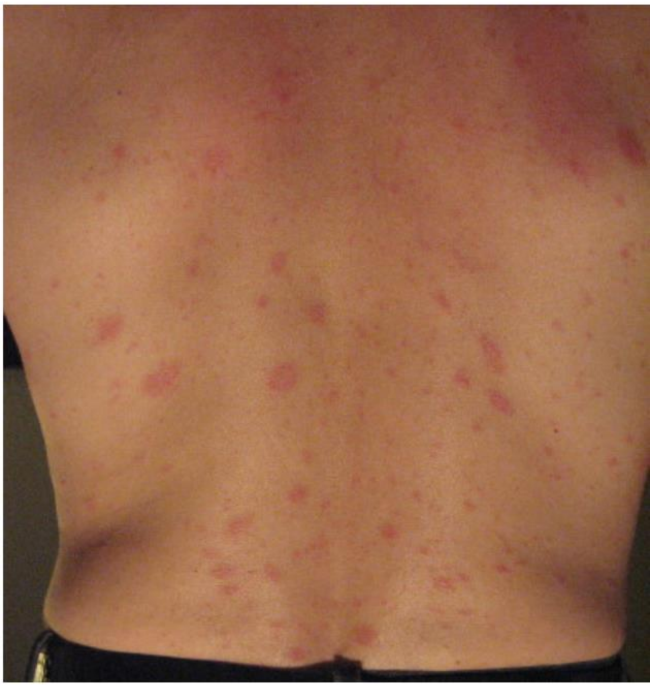
Topical azelaic acid and oral isotretinoin is incorrect. Topical azelaic acid is used to treat mild-tomoderate rosacea with papules and pustules. Hence, it will not apply to this patient with a greater disease severity. As mentioned previously, isotretinoin is not to be used as a first-line treatment, making this an unlikely prescription for this patient.

Topical ivermectin is incorrect. It is a first-line therapy for mild-to-moderate papulopustular rosacea. This patient, however, has a more severe rosacea phenotype and will require a combination of topical therapy and an oral antibiotic like doxycycline.

Topical metronidazole and oral isotretinoin is incorrect. Topical metronidazole is another topical treatment used as first-line therapy for mild-to-moderate papulopustular rosacea. Isotretinoin is not used as first-line therapy, as explained previously. Hence, a combination of topical metronidazole and oral isotretinoin will not be suitable for this patient who has severe disease and is treatment naïve.

Question 3 of 182

A 21-year-old man presents with an itchy rash around 2 weeks after a brief illness with mild fever and malaise. He first noticed a scaly patch on his chest around 2cm in diameter, and 2 days later developed the lesions as shown in the image. He is otherwise well.



What virus has been hypothesized to be a trigger for this condition?

A. Adenovirus
B. Cytomegalovirus
C. Enterovirus
D. Epstein Barr virus
E. Human herpes virus 7

E. Human herpes virus 7

This patient has pityriasis rosea. This is a self-limiting skin condition that presents numerous itchy, scaly oval lesions, mainly on the trunk and proximal aspects of the limbs. In over half the cases, the rash is preceded by the appearance of a 'herald patch' which is a larger lesion around 2-5cm in diameter and which appears around 2 days before the onset of the wider rash. Pityriasis rosea occurs mainly in older children and young adults.

The trigger for the rash is not definitively known but a viral cause is thought likely given that many cases are preceded by a viral-type illness with fever, malaise, headache, and sore throat and because cases can occur in clusters or outbreaks. The strongest evidence is for an association with **human herpes virus 7** (HHV7). This is a similar virus to human herpes virus 6 (HHV6), both of which cause exanthem subitum (also called roseola) in infants, as well as a non-specific febrile illness in older children.

Adenovirus is incorrect. This virus can cause a range of presentations, including conjunctivitis, pharyngitis, and gastroenteritis, but there is no known association with pityriasis rosea.

Cytomegalovirus is incorrect. This virus is also in the herpes virus family. It can cause an acute febrile illness similar to infectious mononucleosis and is associated with complications in immunesuppressed patients or pregnant women, but no association has been shown with pityriasis rosea.

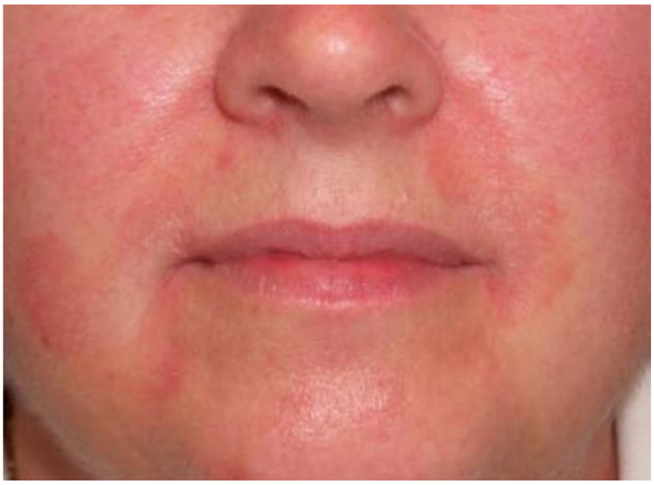
Enterovirus is incorrect. This is a group of viruses that can a range of conditions including gastroenteritis, meningitis/encephalitis, hand foot, and mouth disease, and non-specific rashes. They are not thought to be a trigger for pityriasis rosea.

Epstein Barr virus is incorrect. This is again a herpes virus, but it is not known to trigger pityriasis rosea.

Question 4 of 182

A 34-year-old male sex worker presents with a sudden onset of a flaky, erythematous rash affecting his nasolabial folds and upper chest. He has also noticed dandruff, which has not previously been an issue.

On examination, there is fissuring behind his ears and yellow scaling between his eyelashes.



What investigation is most likely to diagnose the underlying condition predisposing to this patient's skin changes?

A. Anti-dsDNA antibodies
B. HIV serology
C. Hepatitis B serology
D. Oral glucose tolerance test
E. Treponemal EIA

B. HIV serology

The patient in this scenario presents with seborrhoeic dermatitis. While seborrhoeic dermatitis is commonly seen in individuals without underlying medical conditions, it occurs more frequently in immunocompromised patients, particularly those with HIV. Therefore, **HIV serology** testing is recommended to exclude HIV as a predisposing factor, especially given the patient's increased risk as a male sex worker.

Anti-dsDNA antibodies are highly specific to systemic lupus erythematosus (SLE). Although SLE can present with various skin manifestations, such as a malar or discoid rash, it does not cause seborrhoeic dermatitis. Therefore, testing for anti-dsDNA antibodies would be inappropriate in this case.

While it would be prudent to test for hepatitis B given the patient's risk of blood-borne viruses, hepatitis B does not cause seborrhoeic dermatitis. Thus, **hepatitis B serology** is less appropriate than HIV serology. Hepatitis B can present with skin manifestations like erythema nodosum, erythema multiforme, and urticaria, but not seborrhoeic dermatitis.

Diabetes mellitus can present with skin conditions such as diabetic dermopathy, acanthosis nigricans, and necrobiosis lipoidica; however, seborrhoeic dermatitis is not associated with diabetes. An **oral glucose tolerance test** would therefore be unnecessary, as it would not help diagnose the skin condition and there are no indications of diabetes in the patient's history.

While testing for syphilis may be advisable for a sex worker, syphilis does not cause seborrhoeic dermatitis. **Treponema EIA** testing is therefore also incorrect, as seborrhoeic dermatitis is more likely to be related to HIV. Syphilis has characteristic skin manifestations, including the primary chancre and the rash found on the soles of the feet and palms of the hands in secondary syphilis.

Question 5 of 182

A 82-year-old man is reviewed in dermatology clinic as part of ongoing treatment for metastatic melanoma. He had been diagnosed with melanoma three years previously after resection of a lesion on his left shoulder. Surgical margins had been satisfactory, however a metastatic lesion had been identified in the patient's liver four months prior to the present day. Following oncology MDT discussion, patient had been treated with vemurafenib. Initial follow-up with repeat abdominal CT scan had demonstrated an excellent response with dramatic reduction in size of metastatic deposit.

The patient reported continuing to feel well in himself. However, he was concerned about a new skin lesion he had noticed on his right thigh. While he could not recall exactly when this lesion had first appeared, he was confident that it had been present for less than two months. It had not been present during a full dermatological examination at the previous clinic appointment three months previously. The patient reported that the lesion was a bit uncomfortable when he caught it on his trousers but denied any other symptoms.

Examination of the lesion on the patient's thigh revealed a 11 mm diameter erythematous papule with minimally everted edges. A slight scale was noted on the surface of the lesion. There was no evidence of telangiectasia associated with the lesion. Examination of the inguinal lymph nodes was unremarkable.

Following assessment in clinic, a biopsy of the lesion was taken.

What is the most likely diagnosis following histological examination of the biopsy specimen?

A. Melanoma
B. Merkel cell carcinoma
C. Squamous cell carcinoma
D. Atypical fibroxanthoma
E. Keratocanthoma

C. Squamous cell carcinoma

Vemurafenib and dabrafenib are selective BRAF inhibitors that increase overall survival in metastatic melanoma. These drugs paradoxically activates the MAPK pathway in keratinocytes and so can cause squamous cell carcinomas, often within the first three months of therapy. The risk of squamous cell carcinoma with vemurafenib and dabrafenib is higher in older patients and they can occur in sun-protected sites.

Given the typical appearance of the above lesion and the recent treatment with vemurafenib, the most likely histology of the lesion in this case is squamous cell carcinoma. Melanoma is less likely due to the atypical appearance and apparent good treatment response to vemurafenib.

The description of the lesion is not consistent with the typical appearance of the other possible answers.

Question 6 of 182

A 35-year-old man is reviewed in the medical clinic. He has severe eczema and over the last two weeks has had a flare of eczema which the GP has been unable to manage. He normally manages his condition with emollients and hydrocortisone 1%, but has noted worsening flexural irritation and pruritus. He denies any systemic symptoms. He also has a past medical history of asthma for which he rarely needs his salbutamol inhaler, and hay-fever. He takes no oral medications. He has a previous dermatology appointment letter which advises that if his eczema remains poorly controlled he should have a short course of a moderate topical steroid.

Which prescription is most appropriate?

On examination, he has macular erythema of his flexural surfaces, especially elbow and knee creases.

A. Clobetasone butyrate 0.05% (Eumovate)
B. Betamethasone valerate 0.1% (Betnovate)
C. Fluticasone propionate 0.05% (Cutivate)
D. Clobetasol propionate 0.05% (Dermovate)
E. Hydrocortisone 2.5%

A. Clobetasone butyrate 0.05% (Eumovate)

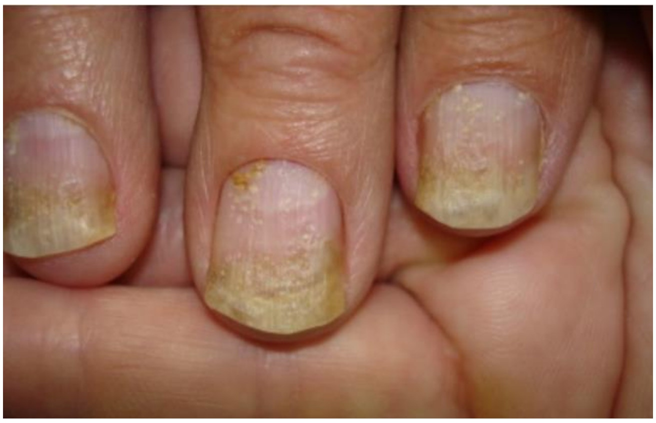
Clobetasol is the most potent topical steroid compared to clobetasone, betamethasone valarate and hydrocortisone

This patient has poorly controlled eczema and needs a moderate strength topical steroid. This question tests knowledge of the strength of different topical steroids. This can be important as the level of systemic absorption can become significant with higher dose topical steroid creams and also stronger topical treatments should not be applied to sensitive skin due to the potential of skin thinning.

Question 7 of 182

A 45-year-old man presents to his GP with a 4-week history of worsening nail changes. He works as a banker and is finding that the appearance of his nails is affecting his confidence. He also describes occasional pain in his wrists and fingers but says this is managed well with over-thecounter analgesia. His past medical history includes coeliac disease and hypothyroidism for which he takes levothyroxine.

On examination, his observations are within normal limits. There are no visible skin or mucosal changes. His nails are examined, as shown below.



What is the most likely diagnosis?

A. Onychomycosis
B. Parakeratosis pustulosa
C. Paronychia
D. Psoriasis
E. Yellow nail syndrome

D. Psoriasis

This patient's nails show signs of onycholysis (lifting of the nail bed) and pitting (indentations to the surface of the nails) which are suggestive of nail psoriasis. Nail psoriasis is commonly associated with chronic plaque psoriasis and is a risk factor for the development of psoriatic arthritis. The clinical history of wrist and finger pain further supports the diagnosis of developing psoriatic arthropathy. Other nail features of psoriasis (although not seen here) include splinter haemorrhages, leukonychia, and nail crumbling.

Onychomycosis is otherwise known as a fungal nail infection. Common organisms include dermatophytes such as *Trichophyton rubrum* and *Candida albicans*. Signs of onychomycosis include distal and lateral onycholysis, subungual hyperkeratosis (scaling under the nail), and nail destruction. Not all nails are necessarily affected by onychomycosis and involvement of all nails should raise suspicion of an alternative diagnosis. This patient also complains of wrist and finger pain and has evidence of nail pitting, making psoriasis the more likely diagnosis.

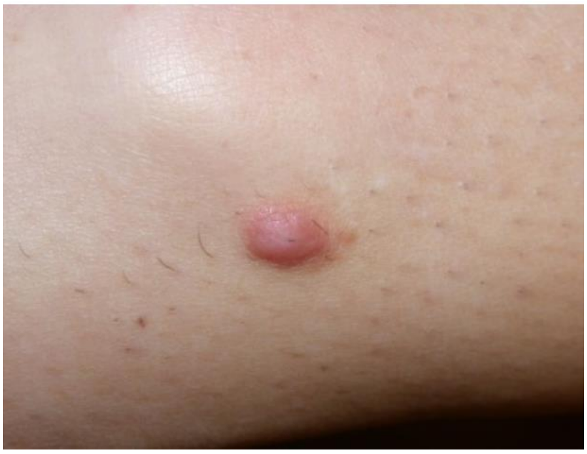
Parakeratosis pustulosa is most commonly seen in younger children and involves the destruction or crumbling of a finger or toenail along with erythema and scaling of the surrounding skin. Similarly to this patient, onycholysis and nail pitting are possible nail features. However, the involvement of all nails with no surrounding skin changes and the older age of the patient makes parakeratosis pustulosa unlikely.

Paronychia is the inflammation of the skin surrounding a finger or toenail secondary to infection with *Staphylococcus aureus* or *Streptococcus pyogenes*. In acute paronychia, usually, only one finger or toe is affected and the nail is less commonly involved compared to chronic disease (i.e. > 6 weeks).

Yellow nail syndrome refers to the thickening and yellow discolouration of nails secondary to halted nail growth. It is associated with multiple conditions including chronic sinus infections, bronchiectasis, and pleural effusions.

Question 8 of 182

A 35-year-old man asks you to review a lesion on his right arm. This has been present for the past six months but has not changed significantly over that period.



What is the most likely diagnosis?

A. Spitz naevus
B. Dermatofibroma
C. Nodular basal cell carcinoma
D. Viral wart
E. Dermoid cyst

B. Dermatofibroma

The correct answer is **Dermatofibroma**. Dermatofibromas are common benign cutaneous nodules of unknown aetiology. This lesion, as seen in the image, is a firm papule or nodule that appears dermal yet is firmly adherent to the epidermis. The colour can vary from skin-coloured to redbrown. It is often asymptomatic but can be tender or itchy, particularly if traumatised. The characteristic 'dimple sign' (a central depression when the lesion is laterally compressed) is usually present.

The first incorrect option is a **Spitz naevus**. Although Spitz naevi may appear similar to dermatofibromas on visual inspection, they are typically seen in children and teenagers rather than adults. They often have a more rapid onset (weeks to months) compared with the six-month history provided in this case. Furthermore, Spitz naevi tend to change significantly over time, unlike the stable nature of the lesion described here.

The second incorrect option is a **Nodular basal cell carcinoma**. While these lesions may also appear similar to dermatofibromas visually, they are typically characterised by more aggressive growth patterns and features such as telangiectasia (tiny blood vessels), ulceration or bleeding which were not described in this case.

The third incorrect option is a **Viral wart**. Viral warts caused by human papillomavirus (*HPV*) are typically keratotic and rough-surfaced lesions that grow rapidly over weeks to months. They may also have black dots representing thrombosed capillaries ('wart seeds'). None of these features were mentioned in this case.

Lastly, a **Dermoid cyst** was an incorrect choice because dermoid cysts are congenital lesions that present early in life and grow slowly over years rather than months. They can be fluctuant or doughy to touch unlike the firm lesion described here.

Question 9 of 182

A 58-year-old man is referred to the clinic with chronic diarrhoea and episodes of facial flushing. A computed tomography scan of his chest and abdomen reveals that he has a neuroendocrine carcinoma of the appendix, causing carcinoid syndrome.

He refuses treatment and presents six months later having been referred by a neurologist as having early onset dementia and a rapidly evolving photosensitive rash on his trunk and upper arms.

Which of the following is the most likely diagnosis?

A. Systemic lupus erythematosus
B. Herpes zoster infection
C. Pellagra
D. Wernicke-Korsakoff syndrome
E. Porphyria

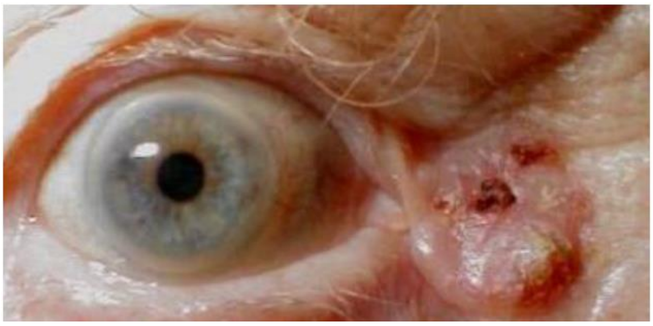
C. Pellagra

Niacin (B3) deficiency is characterised by dermatitis, diarrhoea and dementia, a condition known as pellagra

The most likely diagnosis, in this case, is pellagra, a vitamin deficiency disease most frequently caused by a chronic lack of niacin (vitamin B3). In carcinoid syndrome, neuroendocrine tumours along the GI tract use tryptophan as the source for serotonin production, which limits the available tryptophan for niacin synthesis.

Question 10 of 182

An 80-year-old woman presents due a 'sore' on the medial aspect of her right eye:



What is the most likely diagnosis?

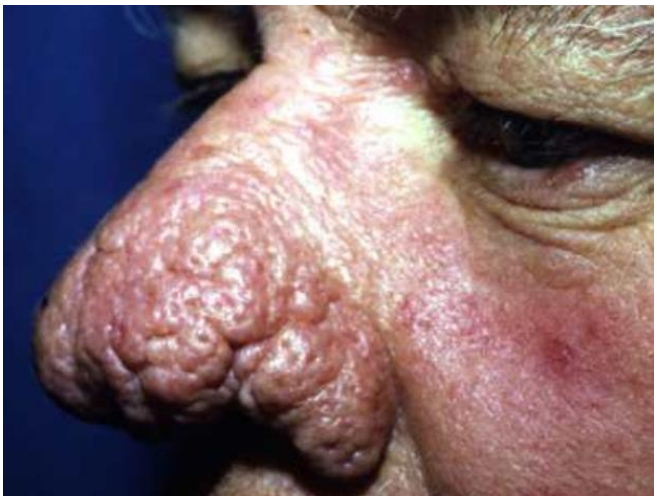
A. Basal cell carcinoma
B. Squamous cell carcinoma
C. Chalazion
D. Stye
E. Indurated dacryocystitis

A. Basal cell carcinoma

The rolled, pearly edges with telangiectasia on the inferior border of the lesion make basal cell carcinoma the most likely diagnosis.

Question 11 of 182

An elderly man complains about the appearance of his nose:



What is the most likely diagnosis?

A. Sarcoidosis
B. Basal cell carcinoma
C. Systemic lupus erythematosus
D. Alcohol excess
E. Acne rosacea

E. Acne rosacea

Rosacea features:

- nose, cheeks and forehead
- flushing, erythema, telangiectasia → papules and pustules

This man has a rhinophyma, a complication of acne rosacea.

Question 12 of 182

A 72-year-old woman is diagnosed with a number of erythematous, rough lesions on the back of her hands. A diagnosis of actinic keratoses is made.

What is the most appropriate management?

A. Reassurance
B. Urgent referral to a dermatologist
C. Topical fluorouracil cream
D. Review in 3 months
E. Topical betnovate

C. Topical fluorouracil cream

The correct answer is **Topical fluorouracil cream**. Actinic keratoses (AKs) are rough, scaly patches on the skin that are caused by excessive exposure to the sun. They can potentially develop into squamous cell carcinoma, a type of skin cancer. Therefore, treatment is recommended to prevent this progression. In the UK, according to NICE guidelines, topical fluorouracil cream is considered first-line therapy for multiple AKs. This medication works by interfering with the DNA in skin cells, preventing their growth and causing them to die.

The first option, **Reassurance**, is incorrect because although actinic keratoses are not malignant themselves, they have the potential to progress into squamous cell carcinoma. Hence it's important to treat them rather than simply reassuring the patient.

The second option, **Urgent referral to a dermatologist**, isn't necessary unless there's suspicion of malignant transformation or if initial treatments fail - neither of which we're told is applicable in this case scenario.

Review in 3 months without any treatment intervention may allow for unnecessary progression of the lesions and hence is not an appropriate management strategy.

Finally, **Topical betnovate**, a corticosteroid cream, is also not a suitable treatment for actinic keratoses as it doesn't target the abnormal proliferation of keratinocytes seen in these lesions. Instead, it's primarily used for conditions involving inflammation and itching such as eczema and dermatitis.

Question 13 of 182

A 68-year-old man presents with blistering lesions on his skin. He reports he has suffered from mouth ulcers recently for which his GP prescribed chlorhexidine mouthwash. However, this has not been of any benefit and the ulcers are now affecting his ability to eat and drink. He reports over the last few days, large blisters have appeared across his torso and back.

His past medical history includes hypertension and angina. He currently takes ramipril, simvastatin and GTN spray when needed.

On examination, you find his mouth is erythematous with multiple superficial blisters; some have ulcerated and the patient reports they have been slow to heal. He has flaccid bullae across his torso and upper back, which appear to be filled with clear fluid. The bullae are fragile, with a thin outer layer. There are some areas where the blisters have ruptured, leaving exposed eroded skin, with some crusting around the edges. Dermatology comes to review and they perform a punch biopsy, but advise you to start treatment whilst the results are awaited.

Given the likely diagnosis, what is the most appropriate first-line treatment?

A. Azathioprine
B. Mycophenolate mofetil
C. Topical emollients
D. Topical potent corticosteroids
E. Oral corticosteroids

E. Oral corticosteroids

Systemic glucocorticoids are the first-line treatment in pemphigus vulgaris

This patient has presented with bullous skin lesions with mucosal involvement therefore the diagnosis is pemphigus vulgaris. The first-line treatment for this is oral corticosteroids.

Azathioprine and mycophenolate mofetil are steroid-sparing agents that can be used longer-term to control the disease, but would not be initiated in the acute phase.

Topical emollients are useful for dry skin, eczema, psoriasis and lots of other skin conditions. However, they will not be useful in the management of bullous skin disease.

Topical potent corticosteroids are not appropriate treatment in pemphigus vulgaris due to the widespread nature of the disease, including mucosal involvement. Oral steroids are of greater benefit.

Question 14 of 182

A 38-year-old patient is referred to the dermatology clinic due to persistent pruritus. She has a long-standing history of eczema and is using topical corticosteroids and emollients as prescribed. On examination, you note erythematous, excoriated patches of eczema on the flexural surfaces of the legs. The patient complains of intense itching and seeks additional relief.

What non-pharmacological method is most appropriate for controlling pruritus in this patient?

A. Bandaging lesions
B. Low humidity environment
C. Multiple baths a day
D. Oral antihistamines
E. Use of hard water

A. Bandaging lesions

Bandaging can help control itching in eczema to prevent further scratching

Bandaging lesions is the correct answer. Bandaging eczematous lesions can provide significant relief from itching as it protects the skin from further scratching, thereby interrupting the itchscratch cycle. It also helps to maintain moisture and enhance the penetration of topical medications. Dressings can also be worn overnight to prevent involuntary scratching during sleep.

Low humidity environment is incorrect. Low-humidity environments have been recognised to aggravate eczema as they dry the skin, which can lead to itching. Maintaining skin hydration is an essential cornerstone of managing eczema, and this is done with the help of emollients and moisturisers applied multiple times during the day.

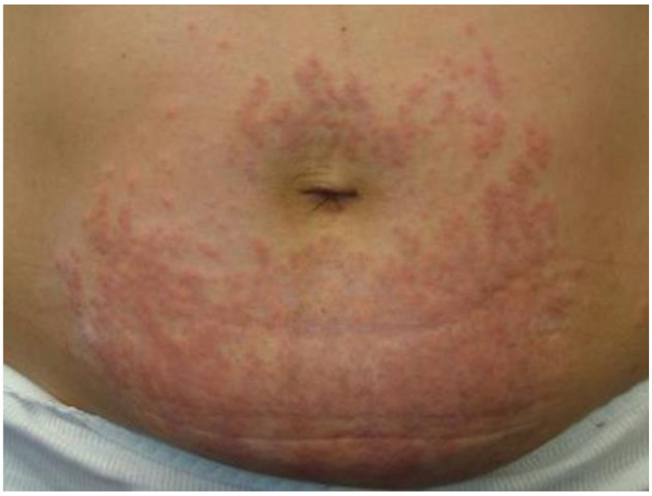
Multiple baths a day is incorrect. Excessive bathing or washing can cause flares of eczema and increase pruritus, as it can dry the skin and expose it to chemicals in the water that can irritate it. Daily bathing/showering should be accompanied by using a mild cleanser during the bath, followed immediately by applying an emollient to prevent the skin from drying.

Oral antihistamines is incorrect. Oral antihistamines, especially the first-generation antihistamines, which have an additional sedative effect, are commonly used to relieve pruritis in patients with eczema. However, they are a pharmacological method to control pruritus, which makes them the wrong answer in the given context.

Use of hard water is incorrect. It has been reported that areas with hard water have a higher prevalence of eczema in the population. It is postulated that the minerals in the hard water cause the deposition of soap compounds like sodium lauryl sulfate on the skin, which can cause skin irritation and itching.

Question 15 of 182

A woman who is 30 weeks pregnant asks you about an itchy rash on her abdomen:



What is the most likely diagnosis?

A. Pre-eclampsia
B. Polymorphic eruption of pregnancy
C. Primary herpes simplex infection
D. Pemphigoid gestationis
E. Pompholyx

B. Polymorphic eruption of pregnancy

The correct answer is **Polymorphic eruption of pregnancy**. This condition, also known as PEP or pruritic urticarial papules and plaques of pregnancy (PUPPP), typically presents in the third trimester. It is characterised by an itchy rash that usually starts on the abdomen, particularly around stretch marks, and can spread to the rest of the body except for the face. The rash consists of small red bumps and hives, and sometimes larger patches of raised skin. The exact cause is unknown, but it may be related to stretching of the skin.

Pre-eclampsia is incorrect because this condition does not typically present with a rash. Preeclampsia is a serious disorder of pregnancy characterised by high blood pressure and signs of damage to another organ system, most often the liver and kidneys. Symptoms often include severe headaches, visual disturbances, upper abdominal pain, swelling in your face or hands, and shortness of breath.

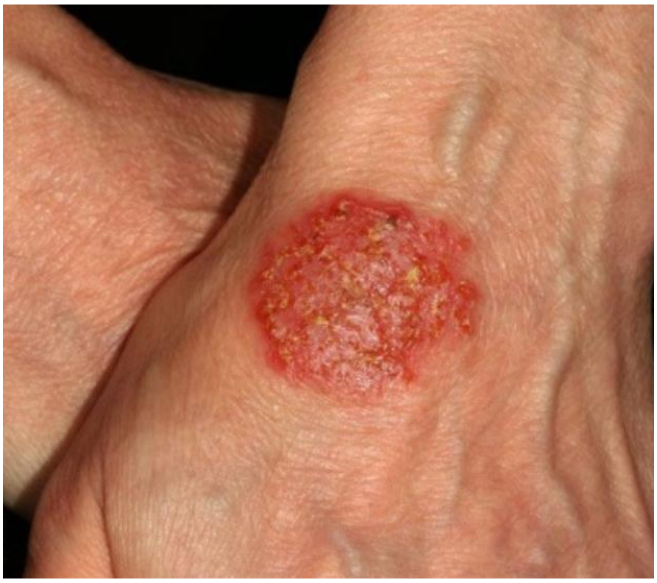
Primary herpes simplex infection during pregnancy could potentially occur but would be unusual to only affect the abdomen and would likely present with painful vesicles rather than an itchy rash. Herpes simplex virus (HSV) infections during pregnancy can lead to neonatal herpes, which can be deadly.

Pemphigoid gestationis, also known as gestational pemphigoid, is an autoimmune blistering disorder that occurs during pregnancy. This option is incorrect because this condition typically presents with blisters rather than a rash. It usually begins with itchy bumps or hives that quickly develop into large blisters over large areas of your body.

Pompholyx, also known as dyshidrotic eczema, causes tiny blisters to erupt across the fingers, palms of the hands and sometimes the soles of your feet. It's extremely itchy but doesn't tend to affect other parts of the body so wouldn't explain a rash on a pregnant woman's abdomen.

Question 16 of 182

A 65-year-old man presents after developing an intensely itchy 'rash' or the dorsum of his left hand. This has now been present for around 6 weeks. He also points out a similar lesion on the dorsum of the right wrist which has appeared in the past week. His past medical history includes type 2 diabetes mellitus and depression. From reviewing his medication history it seems that citalopram has recently been changed to sertraline around 8 weeks ago due to a poor response. The lesion is shown below:



What is the most likely diagnosis?

A. Discoid eczema
B. Contact dermatitis
C. Psoriasis
D. Allergic reaction to sertaline
E. Lichen planus

A. Discoid eczema

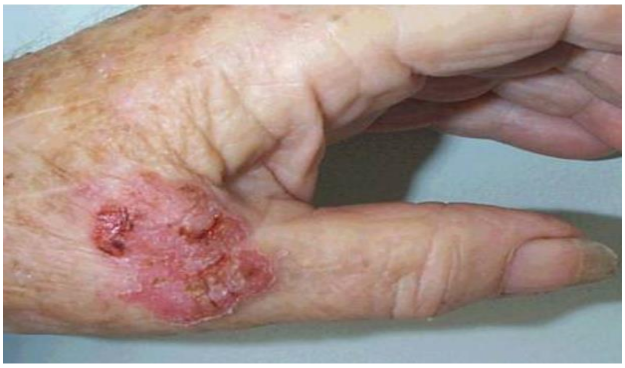
This lesion is typical of discoid eczema. Lesions are typically well defined, intensely pruritic and located on the peripheries. Discoid, or nummular, eczema shows a bimodal distribution, being more common in the 20's (predominately females) and the 60's (predominately males).

These lesions are not consistent with an allergic reaction. If the option of a fixed drug eruption had been given the differential would have been much harder as the appearance is much more similar. Fixed drug eruptions however tend to occur within 24 hours of starting the drug and disappear after 10 days.

The location of the lesions is not consistent with contact dermatitis.

Question 17 of 182

Please look at the image below:



The lesion has been getting bigger for the past few weeks. There is no history of trauma.

What is the most likely diagnosis?

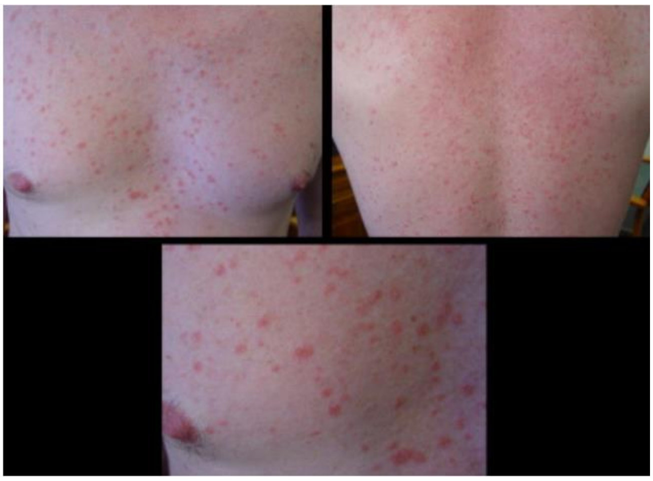
A. Basal cell carcinoma
B. Seborrhoeic keratosis
C. Bowen's disease
D. Tinea corporis
E. Nummular eczema

C. Bowen's disease

Question 18 of 182

A 23-year-old man presents to his General Practitioner with a 3-day history of a widespread rash across his body. He denies fever, pain or itch but is very concerned as the rash is unsightly and is affecting his confidence. Other than recovering from a sore throat and coryzal symptoms over the past week, there is no other past medical history of note and he has no known allergies. He is a teaching assistant in a nursery school.

His rash is examined, as shown below:



What is the most likely diagnosis?

A. Disseminated varicella zoster
B. Guttate psoriasis
C. Pityriasis rosea
D. Pityriasis versicolor
E. Tinea corporis

B. Guttate psoriasis

Guttate psoriasis is correct. This patient has multiple pinky-red scaly lesions predominantly over his trunk. They are numerous in number and relatively small in size (<1cm) in keeping with guttate psoriasis. These lesions can typically have a tear-drop shape to them. Guttate psoriasis is most common in children and young adults and often follows an upper respiratory tract infection with *Streptococcus* species (which may be more likely in an occupational setting with young children).

Disseminated varicella zoster is incorrect. Chickenpox causes a macular, papular and vesicular rash with crusting of lesions over time. In cases of disseminated chickenpox, there is often cerebral involvement and thrombocytopenia, causing purpura. This is not seen in this scenario.

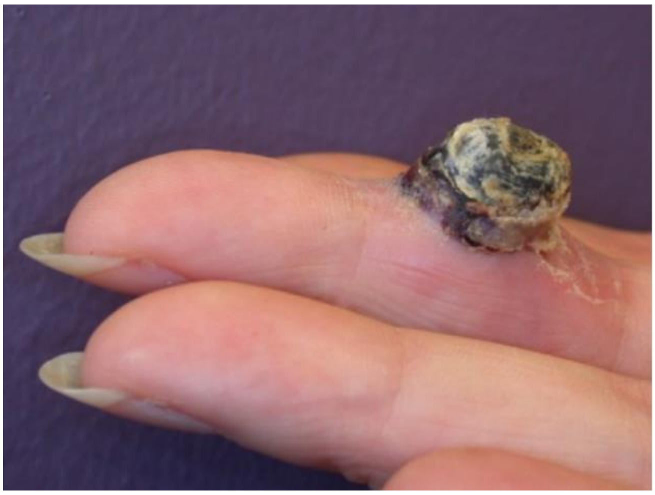
Pityriasis rosea is incorrect. This is a self-limiting rash with no clear cause. The onset of the rash typically begins with a 'herald patch' found on the torso, followed by a widespread salmon-pink or red rash consisting of small (2-5cm) plaques with a peripheral scale. There is no history of a herald patch in this scenario making the diagnosis less likely.

Pityriasis versicolor is incorrect. This is a skin infection caused by the *Malassezia* species of yeast. Features of pityriasis versicolor include asymptomatic hyper- or hypopigmentation or pinkcoloured skin lesions. It is more common in hot climates where perspiration is frequent.

Tinea corporis is incorrect. This is a superficial fungal infection that causes a solitary erythematous and scaly circular lesion on the skin surface. In contrast, this patient has multiple lesions, making tinea corporis incorrect.

Question 19 of 182

A 23-year-old woman who is 10 weeks pregnant presents with a rapidly growing lesion on her finger. This has grown from the size of a 'pin-prick' when it first appeared 4 weeks ago.



What is the most likely diagnosis?

- A. Viral wart
- B. Orf
- C. Pyogenic granuloma
- D. Capillary haemangioma
- E. Squamous cell carcinoma

C. Pyogenic granuloma

The most likely diagnosis in this case is a **Pyogenic granuloma**. Pyogenic granulomas are benign, vascular skin lesions that grow rapidly over weeks or months. They can occur at any age but are more common in children and young adults, particularly pregnant women due to hormonal changes. The lesion often appears as a small, red, raised bump that may bleed easily. In this case, the rapid growth of the lesion and the patient's pregnancy status make pyogenic granuloma the most likely diagnosis.

A **Viral wart** is less likely because these typically grow slowly over time rather than rapidly.

Moreover, they usually have a rough surface whereas the lesion in question appears smooth.

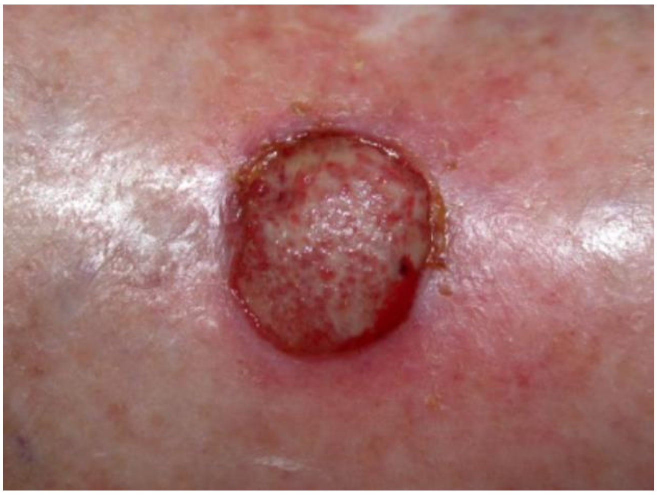
Orf is an infectious disease caused by a parapoxvirus affecting sheep and goats. Humans can contract it through direct contact with infected animals or contaminated objects. However, it usually presents as one or few lesions on hands or fingers with surrounding erythema and regional lymphadenopathy which does not seem to be the case here.

A **Capillary haemangioma**, also known as strawberry naevus, commonly occurs in infants and usually regresses spontaneously by childhood. It would be rare for it to first appear in an adult woman.

Finally, **Squamous cell carcinoma** (SCC) is unlikely given the patient's age - SCC typically occurs in older individuals who have had significant sun exposure over their lifetime. Furthermore, SCCs generally grow slowly over time rather than rapidly.

Question 20 of 182

A 54-year-old man presents with a lesion on his forearm. This has been slowly developing over the past 2-3 months and has bled on one occasion. His past medical history includes Alport syndrome for which he had a renal transplant ten years ago. He currently takes ciclosporin as immunosuppression. He also takes ramipril and amlodipine for hypertension. The lesion is shown below:



What is the most likely diagnosis?

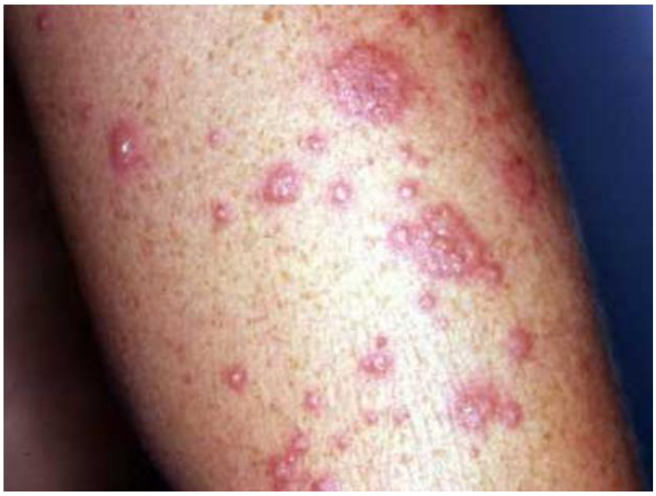
- A. Squamous cell carcinoma
- B. Basal cell carcinoma
- C. Pyogenic granuloma
- D. Haemangioma
- E. Pyoderma gangrenosum

A. Squamous cell carcinoma

The clue here is immunosuppression. Patients on ciclosporin and other immunosuppressants have a much higher risk of squamous cell carcinoma.

Question 21 of 182

A 50-year-old man who has had a cough for the past week develops a rash. It initially appeared on his arms but has now spread to the torso:



What is the most likely underlying diagnosis?

A. Goodpasture's syndrome
B. Adenovirus
C. Mycoplasma pneumoniae
D. Legionella pneumophilia
E. Rhinovirus

C. Mycoplasma pneumoniae

This patient has developed erythema multiforme (EM), a known complication of Mycoplasma infection. *Mycoplasma pneumoniae* is the second most common trigger of EM after the herpes simplex virus.

Question 22 of 182

A 67-year-old gentleman is reviewed in the medical clinic. He was diagnosed by his GP with scabies two weeks ago, but despite treatment he has ongoing itching. He was diagnosed following a GP visit to his house which was found to be unkempt. He was treated with permethrin 5% with the aid of his daughter, who has confirmed that his whole body was treated in two sessions, one week apart, and that his clothes were washed at high temperature at both occasions, as well as his bed sheets.

He has a past medical history of heart failure, ischaemic heart disease and dementia. He lives on his own with the assistance of his daughters. He normally takes bisoprolol, aspirin, ramipril and simvastatin. On examination, there are burrows with track marks on his fingers.

What is the most appropriate plan?

A. Investigate for immunosuppression
B. Repeat treatment with permethrin 5%
C. Treat with malathion 0.5%
D. No further treatment needed
E. Skin biopsy

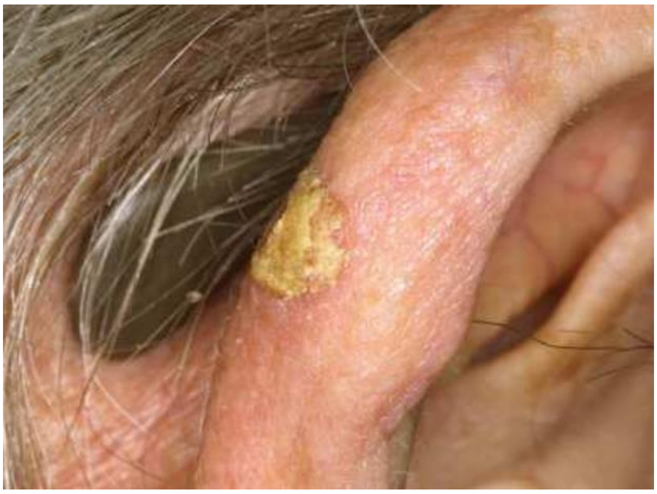
C. Treat with malathion 0.5%

In scabies treatment failure, if application was applied appropriately, then treat with difference insecticide, e.g. malathion 0.5% or permethrin 5%

This is a patient with scabies who has failed treatment despite correct treatment. Whilst patients with may have pruritus until six weeks following treatment completion, there should not be any burrows. For such patients they should be treated with malathion 0.5% if they have been treated correctly with permethrin 5%. If they were treated with malathion 0.9% then they should be treated with permethrin 5%. Skin biopsy is not needed, and immunosuppression is unlikely; only if evidence crusted scabies should this be done, or if extremely wide-spread.

Question 23 of 182

A 60-year-old man asks you to have a look at a 'sore' on his right ear.



It has been present for around 6 months and is not painful.

What is the most likely diagnosis?

A. Fungal otitis externa
B. Actinic keratosis
C. Pyogenic granuloma
D. Basal cell carcinoma
E. Chondrodermatitis nodularis helicis

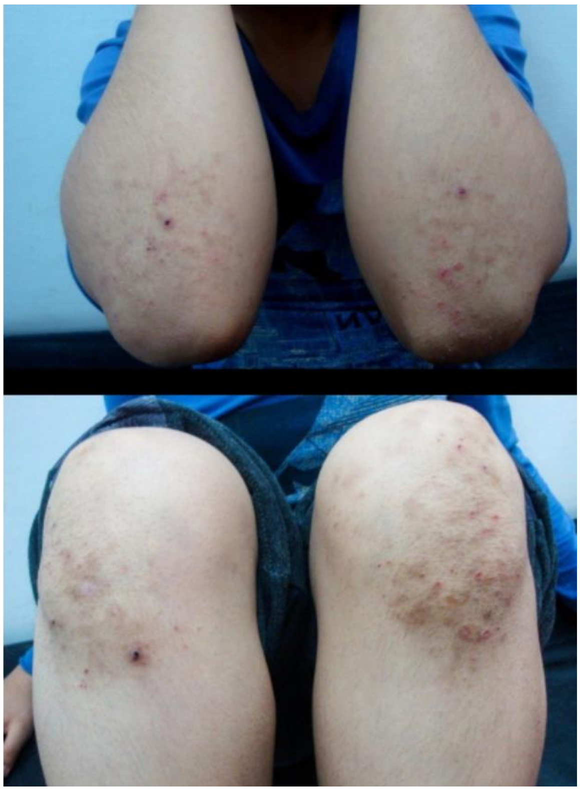
B. Actinic keratosis

Chondrodermatitis nodularis helicis is usually painful.

Question 24 of 182

A 31-year-old man presents with an itchy rash that has been present for several weeks, across both the elbows and knees. He mentions general fatigue but is otherwise well in himself.

On examination, the rash appears as shown below:



Blood tests are also performed:

Hb	118 g/L	Male: (135-180)Female: (115 - 160)
Platelets	267 * 10 /L 9	(150 - 400)
WBC	5.6 * 10 /L 9	(4.0 - 11.0)
Na +	137 mmol/L	(135 - 145)
K +	3.8 mmol/L	(3.5 - 5.0)
Urea	5.8 mmol/L	(2.0 - 7.0)
Creatinine	62 µmol/L	(55 - 120)
CRP	4 mg/L	(< 5)

Given the likely diagnosis, which of the following is the most appropriate first-line treatment?

A. Oral dapsone
B. Oral prednisolone
C. Topical calcipotriol
D. Topical clobetasol
E. Topical tacrolimus

A. Oral dapsone

The diagnosis is that of dermatitis herpetiformis. The photo shows a bilateral, symmetrical, papulovesicular rash across the elbows and knees. This is consistent with dermatitis herpetiformis, which usually overlies extensor surfaces. The lesions appear excoriated due to pruritus. Dermatitis herpetiformis is strongly associated with coeliac disease, which is likely in this case, particularly given the concomitant anaemia and fatigue. Initial management involves the use of oral dapsone.

If initial measures were to fail, oral prednisolone may be used. However, for first-line management, oral dapsone is usually used for dermatitis herpetiformis.

Topical calcipotriol is a vitamin D analogue commonly used in the management of psoriasis. The images here do not clearly demonstrate psoriatic plaques, which would have a scaly appearance - rather, the papulovesicular rash is more suggestive of dermatitis herpetiformis.

Topical clobetasol is a second-line option for dermatitis herpetiformis. First-line, oral dapsone remains the most appropriate option.

Topical tacrolimus is a steroid-sparing agent sometimes used for steroid-resistant eczema, especially for children and on sensitive areas such as the face. It also plays a role in the management of other conditions such as bullous pemphigoid. It is not routinely used in dermatitis herpetiformis.

Question 25 of 182

A 60-year-old man with a history of renal transplant presents to the dermatology clinic.

He has a 7mm scaly nodular lesion on his left left forearm with an inflamed red base. This appears similar to two squamous cell skin carcinomas he has had excised in the past. The lesion is not painful and he has no lymphadenopathy.

He continues to take tacrolimus, though his dose was lowered following his last squamous cell carcinoma excision. He also suffers from hypertension which is well controlled on ramipril.

His blood tests are as follows:

Hb	120 g/l	Na +	136 mmol/l
Platelets	308 * 10 /l 9	K +	4.9 mmol/l
WBC	6 * 10 /l 9	Urea	5 mmol/l
Neuts	4 * 10 /l 9	Creatinine	98 µmol/l

What is the most appropriate management?

A. Curettage and acitretin
B. Radiotherapy and acitretin
C. Surgery alone
D. Surgery and acitretin
E. Systemic chemotherapy

D. Surgery and acitretin

This man has a high risk squamous cell carcinoma (SCC) of the skin given that he is on immunosuppression. He also has a history of previous SCC. Given these factors, surgery is indicated and systemic retinoids, such as acitretin, should be considered to prevent further SCCs.

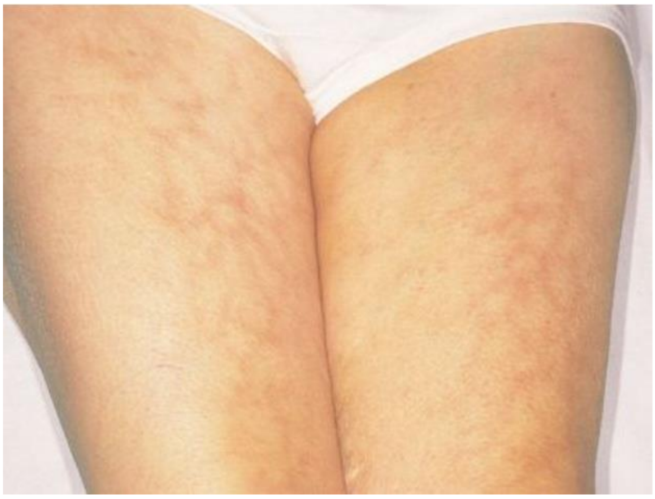
Curettage is acceptable management for a low risk SCCs. Radiotherapy would be considered postoperatively if there were inadequate tumour margins or evidence of invasion at surgery but is not an appropriate a primary curative therapy in this case. Studies to date have not shown sufficient evidence for systemic chemotherapy for SCC.

In patients on immunosuppression with recurrent SCC, reduction of immunosuppression should be considered and discussion at a multidisciplinary team meeting is warranted.

Reference: Scottish Intercollegiate Guidelines Network. 2014. SIGN 140. Management of primary cutaneous squamous cell carcinoma.

Question 26 of 182

A 41-year-old woman shows you a rash on her legs:



What is the most likely cause of such a rash?

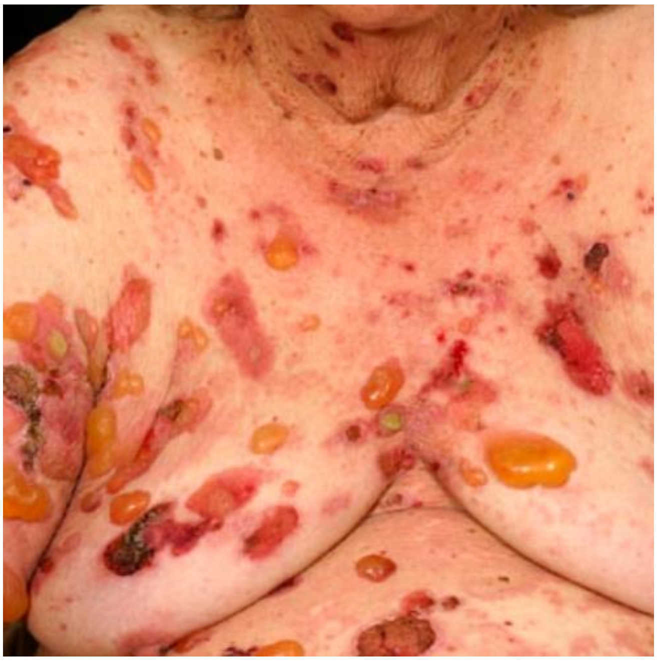
A. Domestic abuse
B. Excessive ultraviolet light
C. Drug reaction
D. Infrared radiation
E. Syphilis

D. Infrared radiation

This patient has erythema ab igne, a skin reaction caused by excessive infrared radiation.

Question 27 of 182

A 79-year-old woman presents with an itchy, blistering rash. Examination of her mouth is unremarkable.



What is the most likely diagnosis?

A. Dermatitis herpetiformis
B. Drug reaction to lisinopril
C. Bullous pemphigoid
D. Pemphigus vulgaris
E. Epidermolysis bullosa

C. Bullous pemphigoid

Blisters/bullae

- no mucosal involvement (in exams at least*): bullous pemphigoid
- mucosal involvement: pemphigus vulgaris

The correct answer to this question is **Bullous pemphigoid**. Bullous pemphigoid is a blistering skin condition that typically occurs in elderly patients, and the presentation of an itchy, blistering rash in a 79-year-old woman is highly suggestive of this diagnosis. The blisters in bullous pemphigoid are usually large and filled with clear fluid, appearing on normal or red skin. They do not typically affect mucosal surfaces, which aligns with the unremarkable examination of the patient's mouth.

The first incorrect option is **dermatitis herpetiformis**. Dermatitis herpetiformis also presents with an itchy, blistering rash, but it is most commonly associated with coeliac disease and tends to present at a younger age than bullous pemphigoid. Additionally, dermatitis herpetiformis often affects extensor surfaces like elbows and knees, whereas the image provided does not suggest this distribution.

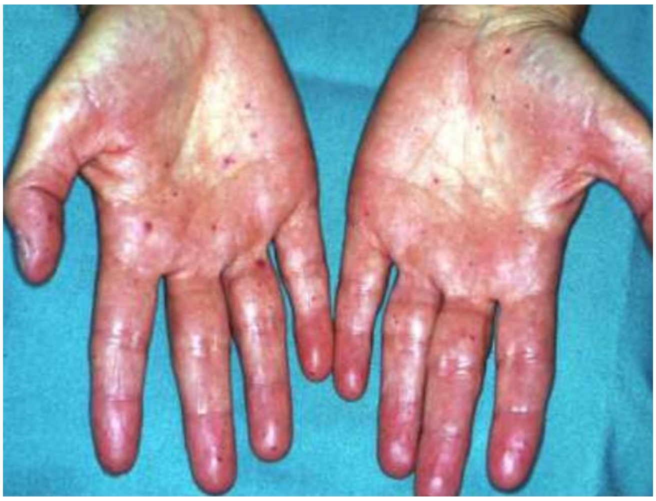
The second incorrect option is a **drug reaction to lisinopril**. While drug reactions can certainly cause rashes and even blisters in some cases, they would typically be more widespread and may be accompanied by other symptoms such as fever or involvement of other organ systems. There is no information given suggesting that this patient has recently started taking lisinopril or any other new medications.

Pemphigus vulgaris, the third incorrect option, is another blistering skin condition which could potentially present similarly to bullous pemphigoid. However, pemphigus vulgaris typically involves mucosal surfaces initially or exclusively - often starting in the mouth - which does not fit with this patient's unremarkable oral examination.

Finally, **epidermolysis bullosa** is incorrect because it's a group of rare inherited conditions causing fragile skin that blisters easily from minor injuries or friction. This condition usually becomes apparent at birth or soon afterwards.

Question 28 of 182

A 30-year-old man is investigated for recurrent nose bleeds and iron deficiency anaemia. You notice a number of erythematous lesions on his skin:



What is the most likely underlying diagnosis?

A. Peutz-Jeghers syndrome
B. Alcohol excess
C. Hereditary haemorrhagic telangiectasia
D. Haemophilia A
E. Idiopathic thrombocytopenic purpura

C. Hereditary haemorrhagic telangiectasia

The correct answer is **Hereditary haemorrhagic telangiectasia**. This condition, also known as Osler-Weber-Rendu syndrome, is a genetic disorder characterised by abnormal blood vessel formation in the skin, mucous membranes, and often in organs such as the lungs, liver, and brain. The erythematous lesions seen on the skin of this patient are likely telangiectasias - small dilated blood vessels near the surface of the skin. Recurrent nosebleeds (epistaxis) and iron deficiency anaemia due to chronic bleeding are common manifestations of this condition.

Peutz-Jeghers syndrome is an autosomal dominant genetic disorder characterised by the development of non-cancerous growths called hamartomatous polyps in the gastrointestinal tract and a characteristic pigmentation around the mouth, lips, eyes and genitalia. It does not typically present with recurrent nosebleeds or erythematous skin lesions.

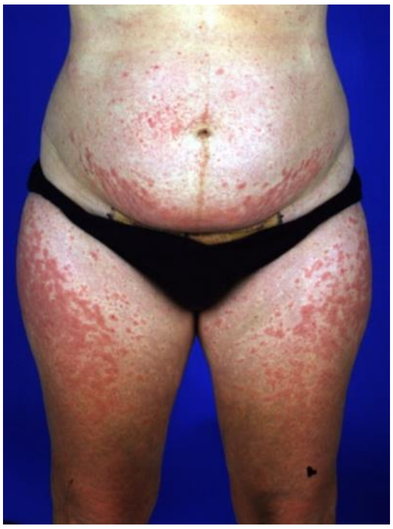
Alcohol excess can cause numerous health issues including liver disease and coagulopathy which might lead to easy bruising and bleeding. However, it would not cause erythematous skin lesions as seen in this case. Furthermore, alcohol-related nosebleeds would usually be associated with other signs of alcohol misuse or its complications.

Haemophilia A is an X-linked recessive disorder characterised by a deficiency in factor VIII leading to prolonged bleeding times. Although patients with haemophilia may experience frequent nosebleeds due to impaired coagulation, they do not develop telangiectasias or similar skin lesions.

Lastly, **Idiopathic thrombocytopenic purpura (ITP)** is an autoimmune disorder causing platelet destruction which leads to a low platelet count (thrombocytopenia). Patients with ITP typically present with petechiae and purpura but not telangiectasia as seen in this case. Nosebleeds could occur due to thrombocytopenia but ITP wouldn't explain the specific type of skin lesion observed here.

Question 29 of 182

A woman who is 31 weeks pregnant presents with a rash on her abdomen and thighs:



The rash is very itchy and she is having difficulty sleeping at night.

What is the most likely diagnosis?

A. Primary herpes simplex infection
B. Pityriasis rosea
C. Polymorphic eruption of pregnancy
D. Pemphigoid gestationis
E. Pompholyx

C. Polymorphic eruption of pregnancy

The correct answer is **Polymorphic eruption of pregnancy**. Polymorphic eruption of pregnancy (PEP), also known as pruritic urticarial papules and plaques of pregnancy (PUPPP), is a benign dermatological condition that occurs in late pregnancy. It typically presents with intensely itchy, polymorphic lesions including erythematous papules, vesicles or plaques. The rash often starts on the abdomen, particularly within stretch marks (striae), and can spread to the thighs and buttocks but rarely involves the face or mucous membranes. This presentation matches the patient's symptoms and her stage of pregnancy.

Primary herpes simplex infection would typically present with painful grouped vesicles on an erythematous base, which may then ulcerate. Primary infections can be associated with systemic symptoms such as fever and malaise. Herpes simplex is not confined to late stages of pregnancy and does not specifically target areas with striae.

Pityriasis rosea usually starts with a single 'herald patch', followed by a 'Christmas-tree' distribution of smaller patches along skin cleavage lines, predominantly on the trunk. It is not related to pregnancy nor does it preferentially affect areas with striae.

Pemphigoid gestationis, previously known as herpes gestationis, is a rare autoimmune blistering disorder associated with pregnancy. It usually begins in the second or third trimester or immediately postpartum. Lesions often start around the umbilicus before spreading to other parts of the body but unlike PEP, pemphigoid gestationis frequently involves the palms, soles and mucous membranes.

Lastly, **pompholyx**, also known as dyshidrotic eczema, primarily affects hands and feet causing itchy vesicles; therefore it wouldn't explain this patient's rash location.

Question 30 of 182

A 52-year-old male was referred to the outpatients dermatology clinic with a four-month history of a persistent rash. He described the rash as a very itchy blistering rash occurring on the popliteal fossa as well as his buttocks. He had been prescribed levocetirizine 5mg OD as well as a four-week course of Eumovate BD (moderately-potent corticosteroid) and Diprobase (emollient) QDS but despite the treatment his symptoms had worsened. He had a past medical history comprising refractory eczema, gout, angina, hypertension and diabetes mellitus type 2 for which he was prescribed allopurinol 100mg OD, aspirin 75mg OD, simvastatin 40mg OD, ramipril 5mg OD and metformin M/R 1g BD. Regarding his eczema, this is presently in a state of remission, having been treated with azathioprine six months ago. Other than the rash he was otherwise well though he mentioned he had a history of abdominal bloating and diarrhoea which was diagnosed as irritable bowel syndrome several years ago.

On examination, he was systemically well with a blood pressure of 142/76 mmHg. Examination of the cardiovascular, respiratory and gastrointestinal systems was unremarkable. Examination of his skin revealed the presence of multiple bullae and papules on the extensor surface of his knees and buttocks, with excoriation.

Which of the following is the most likely diagnosis?

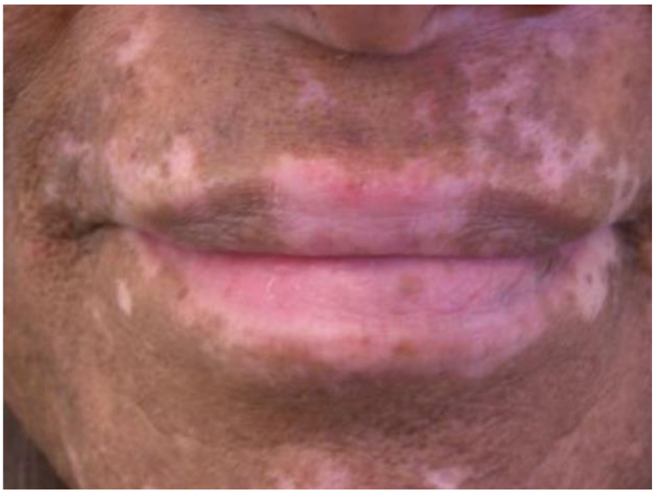
A. Bullous pemphigoid disease
B. Linear IgA bullous dermatosis
C. Dermatitis herpetiformis
D. Transient acantholytic dermatosis
E. Eczema

C. Dermatitis herpetiformis

This rash tends to present as an intensely itchy bullous rash comprising of blisters and papules on extensor surfaces, though can also occur on any other part of the body. This disease is strongly associated with uncontrolled coeliac disease, and the clue in this question is the presence of abdominal bloating and diarrhoea (red herring being diagnosed with irritable bowel syndrome, a common mistake to make in clinical practice!).

Question 31 of 182

You review a 27-year-old man who is under the care of the dermatology department.



Which one of the following conditions is most associated with this skin disorder?

A. Addison's disease
B. Asthma
C. Iron-deficiency anaemia
D. Toxic multinodular goitre
E. Type 2 diabetes mellitus

A. Addison's disease

Vitiligo is associated with other autoimmune conditions such as Addison's disease, type 1 diabetes mellitus and autoimmune thyroid disorders.

Question 32 of 182

A 47-year-old lady presented with shortness of breath and a nosebleed. She had been experiencing heavy nosebleeds over the past fifteen years and had twice required cauterization. On this admission she felt very tired, with shortness of breath and chest pain on minimal exertion.

Aside from nosebleeds she had a past medical history of hypertension and hypothyroidism. She had recently consulted a cosmetic surgeon privately as she had spider veins over her lips which she wished to have removed.

She lived with her husband who had been diagnosed with multiple sclerosis and her 18-year-old son who had learning difficulties and epilepsy. She reported that her mother and sister also experienced frequent nosebleeds.

On examination there was conjunctival pallor. The lung fields were clear on auscultation and both heart sounds were present with a systolic murmur audible over the aortic region. The abdomen was soft and non-tender with no palpable masses or organomegaly. On inspection of the skin there was a bruise in the left antecubital fossa and no rashes.

What is the most likely unifying diagnosis?

A. Von Willebrand disease
B. Wegeners granulomatosis
C. Von Hippel Lindau disease
D. Idiopathic Thrombocytopaenic Purpura
E. Hereditary haemorrhagic telangiectasia

E. Hereditary haemorrhagic telangiectasia

Hereditary haemorrhagic telangiectasia (HHT), also known as Osler Weber Rendu syndrome, is an autosomal dominant disorder leading to abnormal blood vessel formation in the skin, mucous membranes, liver, lungs and brain. In this case, both the patients mother, sister and son are also affected indicating a likely autosomal dominant inheritance. Telangiectasia, such as the ones on this patients lips, are found on mucosal surfaces and in the gastrointestinal tract. These can cause nosebleeds and this is the most common presentation of HHT. Nosebleeds and bleeding from telangiectasia in the gastrointestinal tract can lead to an iron deficiency anaemia which explains the shortness of breath, pallor, chest pain and flow murmur in this case. Other manifestations of HHT occur secondary to the formation of arteriovenous malformations (AVMs) and include haemoptysis, portal hypertension and oesophageal varices, congestive cardiac failure, headache, intracerebral haemorrhage and seizures. Treatment is largely symptomatic and anaemia is treated with transfusions and iron replacement.

Von Willebrand disease can present with nosebleeds and may be inherited in an autosomal dominant fashion. However, this diagnosis would not account for the severe symptomatic anaemia, telangiectasia or the neurological features in this patients son. Other features of von Willebrands disease include bleeding gums and purpura.

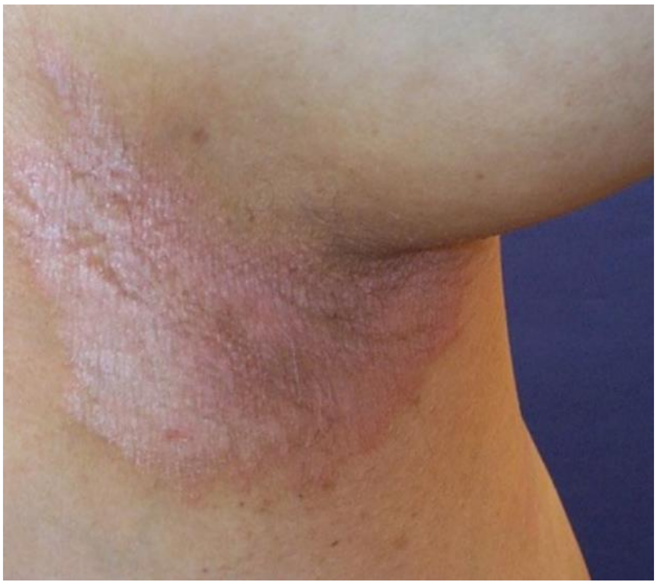
Wegeners granulomatosis is a form of vasculitis that can cause nosebleeds. It is not a genetic condition and other clinical manifestations include haemoptysis, rapidly progressive glomerulonephritis, arthritis and a purpuric rash.

Von Hippel Lindau disease is a rare autosomal dominant condition which is associated with many tumour types including haemangioblastomas, neuroendocrine tumours and renal cell carcinoma. It can present with walking difficulties, headaches and hypertension in addition to other symptoms and signs however nosebleeds are not a commonly recognised feature of this disease.

Idiopathic Thrombocytopaenic Purpura (ITP) is an autoimmune condition which can present with easy bruising, purpura and nosebleeds. In patients with ITP there is usually no family history and the condition does not cause telangiectasia.

Question 33 of 182

A 33-year-old woman with a history of psoriasis presents with a 'rash' in both axillae.



What is the most appropriate first-line treatment?

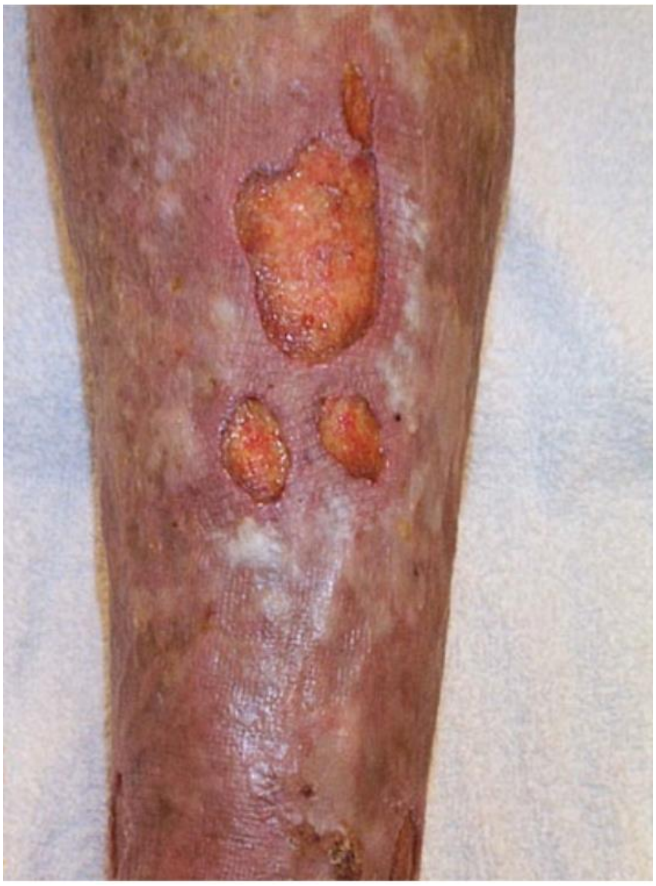
A. Miconazole twice a day
B. Dithranol once a day
C. Calcipotriol twice a day
D. Eumovate (clobetasone butyrate) once a day
E. Calcipotriol + beclomethasone once a day

D. Eumovate (clobetasone butyrate) once a day

This patient has flexural psoriasis - a mild or moderately potent steroid should be used.

Question 34 of 182

You review an 82-year-old woman who has developed 'sores' on her legs. For the past two years she has had dry, itchy skin around her ankles but over the past few weeks the skin has started to break down.



What is the most likely diagnosis?

A. Necrobiosis lipoidica diabeticorum
B. Pyoderma gangrenosum
C. Arterial ulcers
D. Venous ulcers
E. Pretibial myxoedema

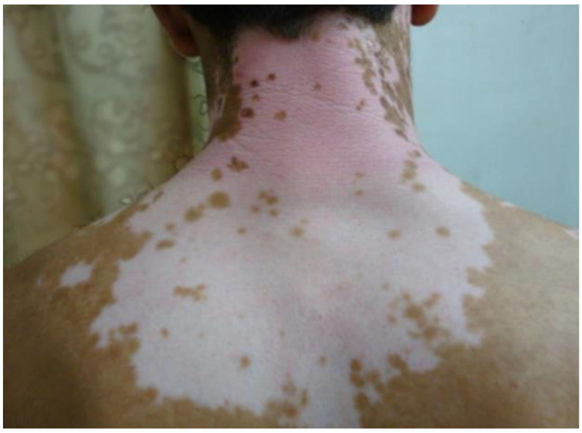
D. Venous ulcers

The dry, skin represents varicose eczema. Arterial ulcers tend to have a more 'punched-out' appearance.

Question 35 of 182

A 45-year-old man has been referred to the dermatology clinic to seek treatment for a long-term skin condition. He is an asylum seeker who has recently arrived in the UK and has never previously been able to access healthcare regarding his skin. He has not yet been started on any treatment. Approximately 20% of his total body surface area is affected by the condition.

His skin is examined, as shown below:



What other condition is this patient most likely to have in addition to his skin disorder?

A. Grave's disease
B. Inflammatory bowel disease
C. Melanoma
D. Microcytic anaemia
E. Systemic lupus erythematosus

A. Grave's disease

The image shows a clear area of skin depigmentation with irregular borders, affecting the patient's back, shoulders and neck. This is a typical presentation of vitiligo, which is an autoimmune condition resulting from the destruction of melanocytes. Vitiligo can be challenging to treat, but treatment options are available that can help to restore pigment in depigmented skin and prevent further depigmentation from occurring in other areas.

The correct answer is **Grave's disease**. As vitiligo is an autoimmune disease, it has a number of associations with other autoimmune conditions. The most common association is with autoimmune thyroid disease, which is found in 15% of adults with vitiligo.

Microcytic anaemia is incorrect. Vitiligo can be associated with pernicious anaemia, but this would cause B12 deficiency which typically causes a macrocytosis rather than microcytosis.

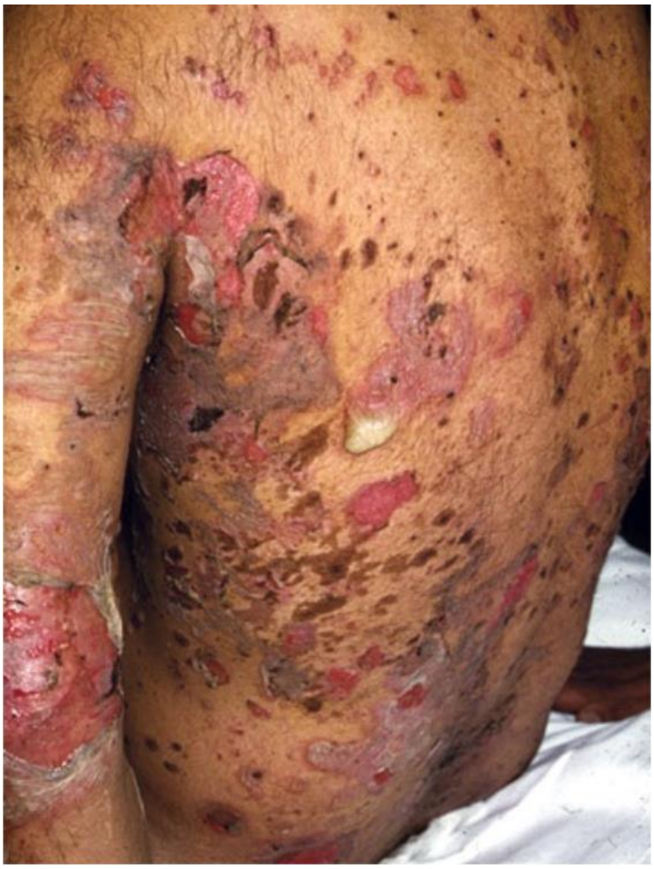
Systemic lupus erythematosus is incorrect. There is an association between systemic lupus erythematosus (SLE) and vitiligo, but this is much less common than thyroid disorders. Other conditions which can be associated with vitiligo include rheumatoid arthritis, Addison's disease, pernicious anaemia, alopecia areata, psoriasis and lichen sclerosis.

Inflammatory bowel disease is incorrect. There is a possible link between vitiligo and inflammatory bowel disease, although this requires further research and is less well established than the association between vitiligo and the conditions above.

Melanoma is incorrect. Surprisingly patients with vitiligo are at lower risk than the general population from skin malignancies, including squamous cell carcinoma, basal cell carcinoma and melanoma, even though the depigmented areas of skin are prone to sun damage. The mechanism for this lower risk is not completely understood and is an ongoing area of research.

Question 36 of 182

A 78-year-old woman presents with a number of blistering lesions on her torso. Around one week prior to the skin lesions developing she noticed widespread mouth ulceration:



What is the most likely diagnosis?

A. Epidermolysis bullosa
B. Pemphigus vulgaris
C. Bullous pemphigoid
D. Stevens-Johnson Syndrome
E. Staphylococcal scalded skin syndrome

B. Pemphigus vulgaris

The correct answer is **Pemphigus vulgaris**. This condition is an autoimmune blistering disorder that often initially presents with oral lesions, which may precede the onset of skin blisters by weeks to months. The image demonstrates flaccid bullae and erosions, which are characteristic of pemphigus vulgaris. In this condition, autoantibodies target desmoglein 3, a protein in the desmosomes that connect keratinocytes in the epidermis. This results in intraepidermal blistering.

Epidermolysis bullosa is incorrect because it typically begins in infancy or early childhood with blistering that occurs following minor trauma to the skin. It's a group of rare inherited skin disorders characterised by blister formation in response to mechanical trauma.

The diagnosis of **Bullous pemphigoid** is also incorrect. Bullous pemphigoid generally affects patients over the age of 60 and usually starts with pruritus, followed by the development of tense bullae. Oral involvement is less common than in pemphigus vulgaris and when present, usually follows rather than precedes cutaneous involvement.

Stevens-Johnson Syndrome (SJS) can be ruled out as well. SJS is usually drug-induced and involves not only the skin but also the mucous membranes such as eyes and genitals along with systemic symptoms like fever and malaise. The rash usually starts as erythematous macules which progress to form blisters and then slough off leading to painful erosions.

Lastly, **Staphylococcal scalded skin syndrome** (SSSS) is unlikely due to the patient's age and presentation. SSSS typically affects neonates and children under 5 years old, presenting with widespread erythema, exfoliation, and purulent conjunctivitis without mucosal involvement. It's caused by exfoliative toxins produced by certain strains of *Staphylococcus aureus*.

Question 37 of 182

A 19-year-old man develops multiple tear-drop papules on his trunk and limbs. He is otherwise well. A diagnosis of guttate psoriasis is suspected.

What is the most appropriate management?

A. Oral penicillin for 14 days
B. Reassurance + topical treatment if lesions are symptomatic
C. Oral penicillin for 14 days + topical treatment if lesions are symptomatic
D. Referral to secondary care
E. Oral corticosteroids

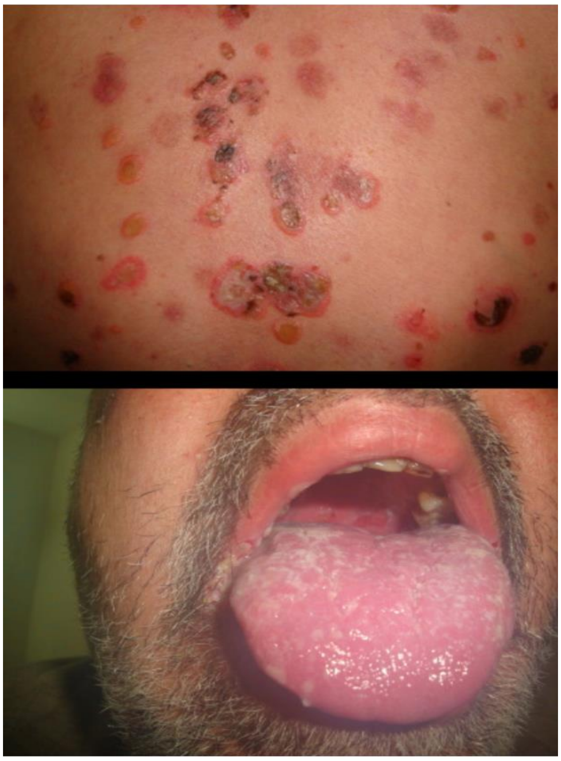
B. Reassurance + topical treatment if lesions are symptomatic

The British Association of Dermatologists state in their psoriasis guidelines that 'evidence does not support a therapeutic benefit from antibiotic therapy'.

Question 38 of 182

A 40-year-old man presents to the dermatology clinic with a 2-week history of an evolving, painful rash. He reported that the lesions initially began inside his mouth before spreading to the rest of his body. Other than a brief coryzal illness before the onset of the rash, he has no past medical history of note and takes no regular medications.

On examination, he has a temperature of 37.8ºC with a heart rate of 96bpm and blood pressure of 117/61mmHg. His rash is examined, as shown below:



What is the most likely diagnosis?

A. Bullous pemphigoid
B. Pemphigus vulgaris
C. Steven Johnson syndrome
D. Toxic epidermal necrolysis
E. Toxic shock syndrome

B. Pemphigus vulgaris

Pemphigus vulgaris is correct. This patient has multiple flaccid blistering lesions, a portion of which have burst. These blisters are filled with a clear fluid that, once ruptured, causes itchy, erythematous and painful erosions. Pemphigus vulgaris is an autoimmune disease that causes skin blistering that often ruptures. Unlike bullous pemphigoid, mucosal ulceration is a common presenting feature in pemphigus vulgaris and can precede the spread of other lesions on the body. Approximately 50-70% of patients have oro-mucosal involvement in pemphigus vulgaris with widespread superficial blistering and erosions and ulceration.

Bullous pemphigoid is incorrect. Unlike pemphigus vulgaris, bullous pemphigoid causes tense blisters that are less likely to rupture. The condition is also more common in the elderly population, particularly elderly women.

Steven Johnson syndrome (SJS) is classically a drug reaction secondary to anti-epileptics, antibiotics and anti-inflammatory medications. Symptoms include flu-like symptoms, widespread blistering and mucosal involvement. Although there is some overlap with this patient's symptoms, there is no history of recent drug administration.

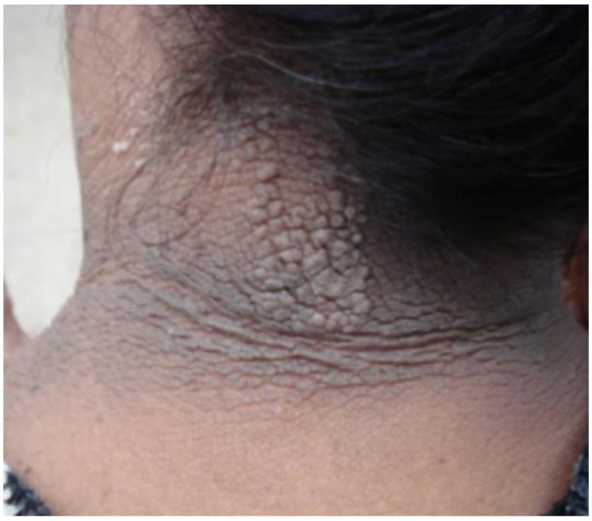
Toxic epidermal necrolysis (TEN) is incorrect. This is a severe form of SJS where > 30% of the total body surface area of the skin is affected. Similarly to SJS, TEN is caused by a drug reaction, which is not described in this scenario.

Toxic shock syndrome is incorrect. This is a severe illness secondary to exotoxin-producing bacteria such as *Staphylococcus aureus*. It causes systemic upset and a diffuse macular erythematous rash with skin-shedding. However, blistering is uncommon.

Question 39 of 182

A 44-year-old woman presents to the dermatology clinic with a 5-week history of skin irritation. She reports that the skin on the back of her neck feels thick and rough to the touch and is occasionally itchy. She used a hand mirror to visualise the skin and was concerned that the colour seems to have changed compared to normal.

On examination, her observations are recorded within normal limits. Her neck is examined, as shown below:



Given the likely diagnosis, what is the most appropriate investigation to consider?

A. ANA panel
B. IgA tissue transglutaminases
C. Random cortisol
D. Random glucose level
E. Skin biopsy

D. Random glucose level

Random glucose level is correct. This patient has a diagnosis of acanthosis nigricans characterised by hyperkeratosis and hyperpigmentation of the skin, predominantly affecting the flexural skin folds such as the axillae, back of the neck and groin. The skin may have a velvety texture secondary to papillomatous overgrowth of the epidermis (which can be visualised in this patient). Common underlying causes include metabolic diseases such as obesity, type 2 diabetes mellitus and polycystic ovarian syndrome. Therefore, an important initial investigation includes checking random or fasting glucose levels to check for the presence of diabetes mellitus.

ANA panel is incorrect. This is used in the diagnosis of autoimmune diseases such as rheumatic disease and vasculitis. However, it has no role in the diagnosis of acanthosis nigricans as it is not associated with underlying rheumatological disease.

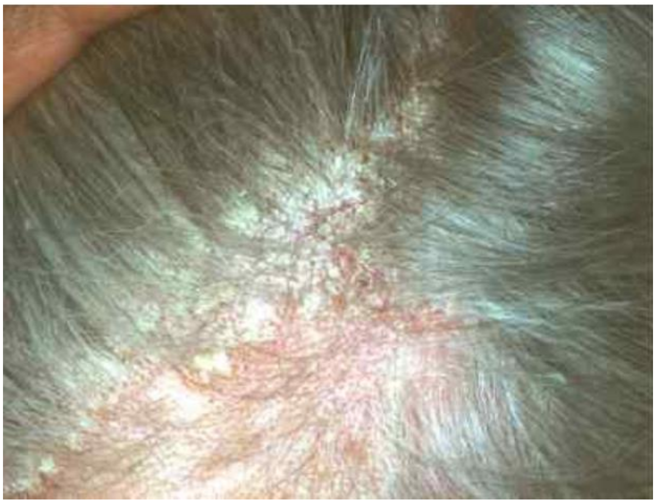
IgA tissue transglutaminase is incorrect. This is used to diagnose coeliac disease that is associated with a vesicular, erythematous and itchy rash seen on the extensor surfaces of the skin.

Random cortisol is incorrect. This is used in the diagnosis of primary hypoadrenalism (e.g. Addison's disease). Addison's disease is associated with the appearance of tanned skin due to excess ACTH production causing stimulation of melanocytes. However, it is not used to diagnose acanthosis nigricans.

Skin biopsy is incorrect. It is not used in the diagnosis of acanthosis nigricans.

Question 40 of 182

A 53-year-old man presents complaining of an itchy scalp and dandruff. On examination he is noted to have the following lesions on his scalp, along with similar lesions behind his ears and around his nose.



He has tried 'Head and Shoulders' and 'Neutrogen T-gel' but with poor results.

Which one of the following is the most appropriate treatment for his scalp?

A. Topical hydrocortisone
B. Topical dermovate
C. Topical selenium sulphide
D. Oral terbinafine
E. Topical ketoconazole

E. Topical ketoconazole

Seborrhoeic dermatitis - first-line treatment is topical ketoconazole

The most appropriate treatment for this patient's scalp is **topical ketoconazole**. The clinical presentation of an itchy scalp, dandruff, and eczema on the scalp, behind the ears, and around the nose suggests a diagnosis of seborrhoeic dermatitis. Seborrhoeic dermatitis is a common inflammatory skin condition that mainly affects areas rich in sebaceous glands such as the scalp, face, and upper trunk. It is thought to be related to an overgrowth of yeast-like fungi from the genus *Malassezia*, which are part of the normal skin flora.

Topical ketoconazole is an antifungal medication that targets *Malassezia* species and helps reduce inflammation in seborrhoeic dermatitis. It can be applied as a shampoo or cream depending on the affected area. In this case, since the patient has already tried other shampoos without success and his symptoms involve both his scalp and face, topical ketoconazole would be suitable to manage his condition.

Topical hydrocortisone may provide some relief from itching and inflammation associated with seborrhoeic dermatitis; however, it does not target the underlying fungal overgrowth contributing to the condition. Additionally, long-term use of topical corticosteroids on facial skin can cause thinning of the skin and other side effects.

Topical dermovate, which contains clobetasol propionate (a potent corticosteroid), may also help reduce inflammation but carries similar risks as hydrocortisone regarding long-term use on facial skin. Moreover, it does not address the fungal component of seborrhoeic dermatitis.

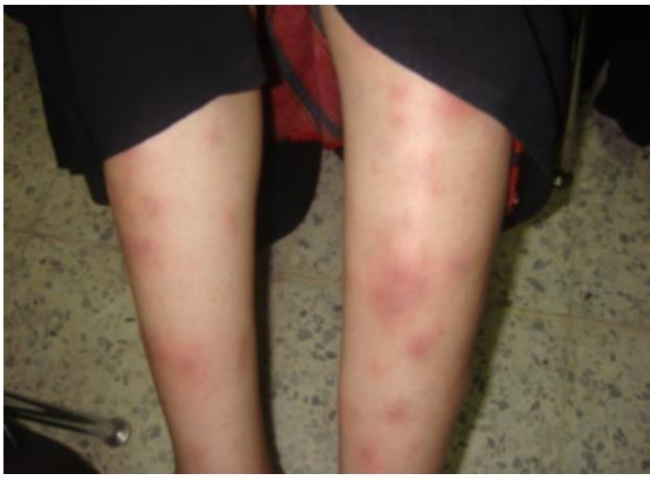
Topical selenium sulphide is another antifungal agent that may be used in treating seborrhoeic dermatitis. However, it is not as effective as ketoconazole and can cause skin irritation, making it a less suitable option for this patient who has already tried other shampoos without success.

Oral terbinafine is an antifungal medication that is primarily used to treat dermatophyte infections such as tinea pedis (athlete's foot), tinea corporis (ringworm), and tinea capitis (scalp ringworm). It is not typically prescribed for seborrhoeic dermatitis, as its efficacy against *Malassezia* species is limited compared to topical ketoconazole. Additionally, oral antifungal therapy carries a risk of systemic side effects and potential drug interactions, making it a less desirable option in this case.

Question 41 of 182

A 29-year-old woman presents with a rash over the shins for the past week, as shown below:

A 29-year-old woman presents with a rash over the shins for the past week, as shown below:



She describes the lesions as tender. She is otherwise well. She has a past medical history of asthma, recurrent acne vulgaris, chronic knee pain following a sports injury and epilepsy. Her medications include salbutamol and beclometasone inhalers, montelukast, oxytetracycline, a combined oral contraceptive pill, naproxen and lamotrigine.

Given the likely diagnosis, which of the following is the most likely cause?

A. Combined oral contraceptive pill
B. Lamotrigine
C. Montelukast
D. Naproxen
E. Oxytetracycline

A. Combined oral contraceptive pill

The diagnosis here is that of erythema nodosum - tender, nodular inflammation of subcutaneous fat, typically over the shins. The image above depicts this well - multiple red lesions are visible, appearing slightly raised and obviously inflamed, across both shins. They are generally oval or round in shape and no ulcers can be seen in the photo, in keeping with erythema nodosum, which does not ulcerate. It normally resolves within 6 weeks and heals without scarring. There are a number of associations, including infections, sarcoidosis, inflammatory bowel disease, Behcet's, and various medications. Of the drugs listed above, the most likely cause is the combined oral contraceptive pill, which is well documented as causing erythema nodosum.

Very rarely, lamotrigine is associated with erythema multiforme and Stevens-Johnson syndrome, which are much more severe, widespread cutaneous disorders than erythema nodosum and can be life-threatening if they progress. It is not associated with erythema nodosum.

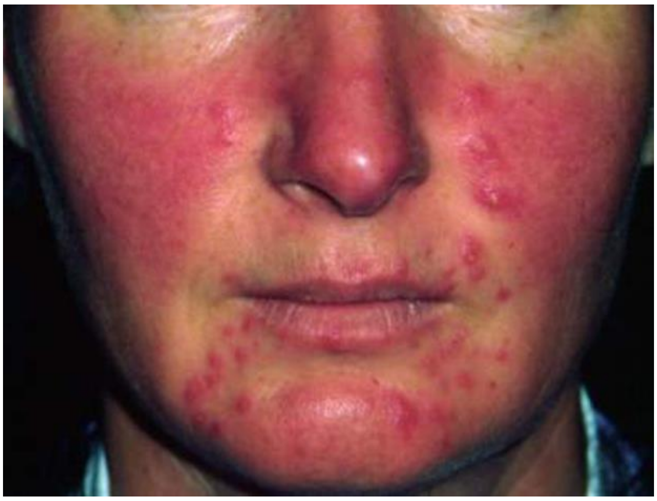
Montelukast is used for control of this patient's asthma, as the next step-up after a low-dose steroid inhaler; it is not linked to erythema nodosum.

Naproxen is presumably being used for her knee pain. It is not linked to the development of erythema nodosum and is, in fact, mentioned in various case reports as being effective in the management of pain arising from the condition.

Oxytetracycline is an antibiotic, being used here for acne vulgaris. Whilst penicillins and sulphonamides are linked to erythema nodosum, tetracyclines are not.

Question 42 of 182

A 43-year-old woman comes for review. A few months ago she developed redness around her nose and cheeks. This is worse after drinking alcohol. She is concerned as one of her work colleagues asked her if she had a drink problem despite her drinking 10 units per week.



What is the most likely diagnosis?

A. Mitral stenosis
B. Seborrhoeic dermatitis
C. Alcohol-related skin changes
D. Acne rosacea
E. Systemic lupus erythematosus

D. Acne rosacea

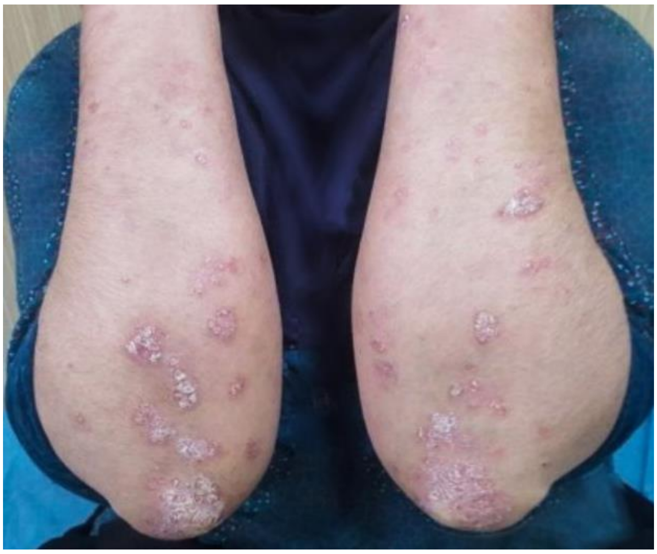
Rosacea features:

- nose, cheeks and forehead
- flushing, erythema, telangiectasia → papules and pustules

This is a typical history of acne rosacea.

Question 43 of 182

A 34-year-old man attends a dermatology clinic with a rash that isn't improving, the rash is pictured below:



He has recently moved to the area but, for the past 3 months, has been using a clobetasol propionate cream in the morning and calcipotriol in the evening, as recommended by his previous dermatologist.

What would be the most appropriate next treatment step?

A. Coal tar applications
B. Increase calcipotriol to twice a day
C. Increase clobetasol propionate cream to twice daily
D. Oral prednisolone
E. Switch to potent topical steroids

B. Increase calcipotriol to twice a day

The picture shows the classic extensor surface plaques of psoriasis.

This patient has been using once-a-day calcipotriol for over 8 weeks without resolution of his psoriasis. It is therefore appropriate to escalate his treatment further.

Increase calcipotriol to twice a day is the correct answer. It would be appropriate to increase the frequency of applications and re-review in 8 weeks.

Increase clobetasol propionate cream to twice daily is incorrect. This is the third-line option and should be considered if the psoriasis rash is not improving after increasing calcipotriol to twice a day.

Coal tar applications is incorrect. This could be considered if twice daily calcipotriol has failed, however, many patients prefer topical steroids as coal tar applications can be messy.

Oral prednisolone is incorrect. This is not the next step in treatment escalation according to NICE guidelines.

Switch to potent topical steroids is incorrect. Clobetasol propionate is already a potent steroid. There is no evidence to suggest that switching to an alternative potent topical steroid is an appropriate option as a second-line treatment for psoriasis.

Question 44 of 182

A 27-year-old man is referred to clinic with an intensely pruritic rash on both his forearms and buttocks.

The rash started around one month ago as papules and vesicles that were very itchy. Once scratched they would quickly crust over. Older lesions would leave pale and dark patches after the crusting had healed over. He had tried multiple emollients and creams including over the counter hydrocortisone cream which had not helped.

He has a history of intermittent abdominal bloating, nausea and diarrhoea which he diagnosed as irritable bowel syndrome (IBS) after reading about it on the internet. Despite trying to adjust his diet, his symptoms are ongoing. He is otherwise fit and well.

Of note in his family history, his mother has a history of hypothyroidism and diabetes. He currently manages his IBS with diet and takes no regular medication.

On examination he is thin but well. Physical observations are all within normal ranges. There are multiple crops of vesicles and papules over the extensor aspects of both his forearms and over his buttocks with evidence of excoriation, erythema and crusting.

Cardiorespiratory, abdominal and neurological examinations are all unremarkable.

Bloods come back as:

Na+	140 mmol/L
K+	3.7 mmol/L
Urea	4.0 mmol/L
Creatinine	68 µmol/L
Hb	98.0 g/dL
MCV	94 fL
WBC	6.5x10^9/L
Platelets	200x10^9/L
Serum folate	1.1 mcg/L
Serum B12	160 ng/L
Ferritin	10 g/L

In view of the likely diagnosis, what is the most appropriate initial treatment?

A. Reassurance
B. Oral steroids
C. Potent topical steroids
D. Oral acyclovir
E. Dapsone

E. Dapsone

Dermatitis herpetiformis is typically treated with a gluten-free diet and topical dapsone

This patient has a rash suggestive of dermatitis herpetiformis characterised by highly pruritic vesicles and papules on extensor surfaces and buttocks. The self-diagnosis of IBS is likely to be incorrect. In view of the low folate and low ferritin (suggestive of malabsorption) an underlying diagnosis of coeliac disease is most likely here.

Dermatitis herpetiformis responds very well to dapsone with relief of itch within a few days. Oral and potent steroids are used second line in patient intolerant or with contraindications to dapsone.

Aciclovir can be used in severe forms of herpes zoster which doesn't fit with this clinical picture. Reassurance is wrong as this condition is unlikely to get better without treatment.

In the long term this patient would need further investigation to confirm the diagnosis of coeliac disease and start a gluten-free diet.

Question 45 of 182

A 25-year-old Caucasian male presents with his first ever episode of witnessed generalised seizure, with witnessed jerking of his right arm and leg, lasting for 4 minutes terminated by benzodiazepines administered by paramedics. There is no other past medical history and documentation of head injuries. His past medical history includes recurrent epistaxes since childhood and current treatment for a left calf deep vein thrombosis provoked following two recent long-haul flights from Australia. A thrombophilia screen has subsequently been negative. Family history is unavailable.

On examination, the patient is confused postictally and is uncooperative with the examination. You note that his pupils are equal and reactive, he is moving all 4 limbs with no obvious focal neurology. There appears to be no visual or sensory neglect. A urinary toxicology screen is negative.

His blood tests and arterial blood gas are as follows:

Hb	175 g/l
MCV	87 fl
Platelets	223 * 10 /l9
WBC	9.2 * 10 /l9
Urea	6.6 mmol/l
Creatinine	64 µmol/l
CRP	24 mg/l
pH	7.32
PaO2	9.1 kPa
PaCO2	4.2 kPa
Lactate	4.2 mmol/l
Bicarbonate	16 mmol/l

With sedation, the patient undergoes a CT head, which demonstrates an arterio-venous fistula in his right parietal lobe.

The patient remains an inpatient while discussed for management with neurosurgeons and returns to baseline with no focal neurological deficits. At 48 hours after admission, he develops sudden onset left face, arm and leg weakness and loss of sensation, with flaccid tone, downgoing plantars.

A repeat CT head confirms a right middle cerebral artery ischaemic stroke.

What is the unifying diagnosis?

A. Malignancy of unclear primary
B. Haemorrhagic hereditary telangiectasia
C. Saddle pulmonary embolus
D. Polycythaemia rubra vera
E. Paroxysmal nocturnal haemoglobinuria

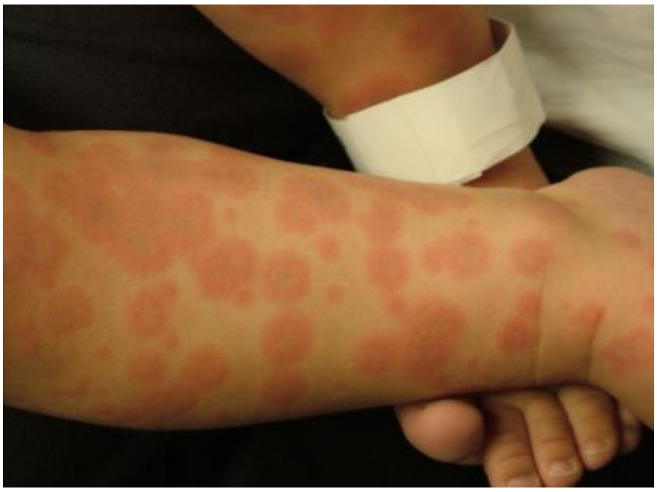
B. Haemorrhagic hereditary telangiectasia

This is a difficult question: the patient has an intracerebral arterio-venous fistula and likely at least another in his pulmonary vasculature, resulting in a chronic hypoxaemia (note normal bicarbonate and secondary polycythaemia), ruling out an acute pulmonary embolus and likely resulting in a paradoxical stroke. Primary polycythaemia rubra vera should not result in hypoxaemia. There does not appear to be an underlying prothrombotic element: his current DVT is uncommon but appears provoked with a negative thrombophilia. There is little to suggest malignancy.

A previous history of epistaxis is also suggestive of haemorrhagic hereditary telangiectasia (HHT), which appears to be the most common and earliest clinical feature. Diagnosis is clinched by the consensus Curacao criteria, requiring the following 4 features to be investigated in this case: spontaneous or recurrent epistaxis, multiple cutaneous telangiectasia (a full dermatological examination is essential), visceral involvement (in this case, pulmonary and cerebral AVMs) and a first-degree relative with HHT (family history is required). Meeting at least 3 features produces a definite diagnosis while 2 of 4 features suggests 'suspected' diagnosis.

Question 46 of 182

A 2-year-old girl develops a rash on her legs. The following day she is brought to the Emergency Department by which time the rash has spread to the rest of her body.



What is the most likely diagnosis?

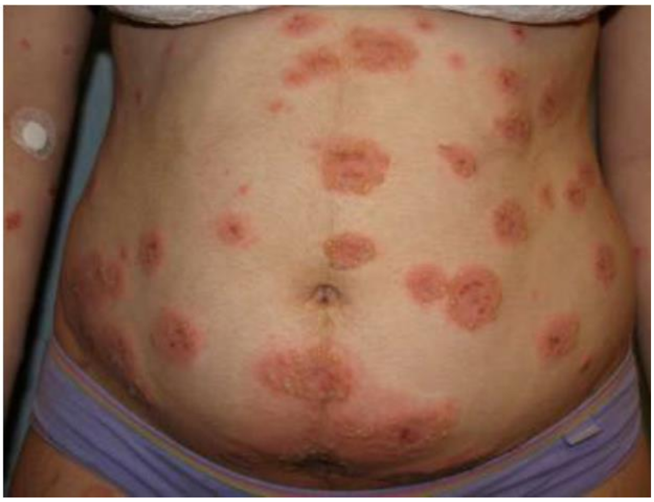
A. Erythema multiforme
B. Erythema chronica migrans
C. Erythema nodosum
D. Urticaria
E. Dermatitis artefacta

A. Erythema multiforme

The classic 'target' lesions of erythema multiforme can be seen clearly on this image.

Question 47 of 182

A woman who is 24 weeks pregnant presents with a rash:



What is the most likely diagnosis?

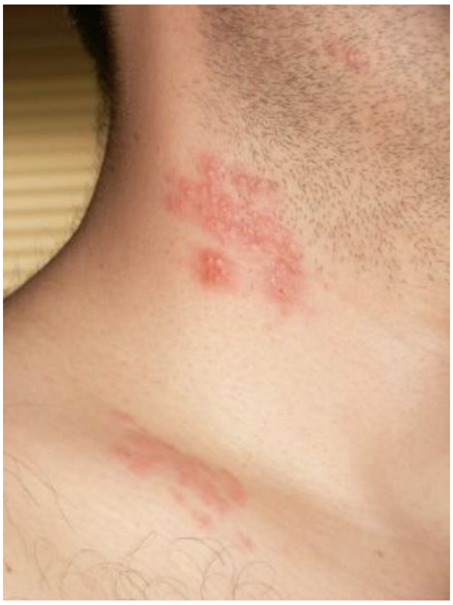
A. Pityriasis rosea
B. Pompholyx
C. Primary herpes simplex infection
D. Polymorphic eruption of pregnancy
E. Pemphigoid gestationis

E. Pemphigoid gestationis

The blistering lesions are clearly visible on this image.

Question 48 of 182

A 34-year-old who has recently returned from a business trip to New York presents with a one-day history of a painful rash on his neck:



What is the most appropriate management?

A. Topical fusidic acid
B. Topical clotrimazole + hydrocortisone
C. Oral aciclovir + prednisolone
D. Oral aciclovir
E. Send blood for antibodies to Borrelia burgdorferi

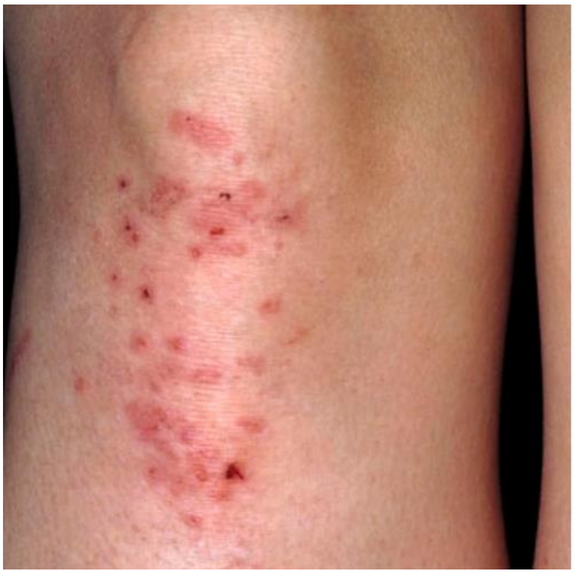
D. Oral aciclovir

One of the main clues in the question is the combination of a rash with pain. Other than shingles, there are not many conditions which cause both.

Whilst there is some evidence that systemic steroids speed up the healing of shingles, consensus guidelines do not advocate their use as adverse effects probably outweigh potential benefits.

Question 49 of 182

A 34-year-old man presents with a three week history of an intensely itchy rash just below his knees. On examination he has a symmetrical vesicular rash as shown below and also some early lesions on the back of his arms.



Which one of the following antibodies is most likely to be positive?

A. Anti-mitochondrial antibody
B. Anti-tissue transglutaminase antibody
C. Anti-nuclear antibody
D. Anti-neutrophil cytoplasmic antibody
E. Anti-Jo-1 antibody

B. Anti-tissue transglutaminase antibody

The correct answer in this case is **Anti-tissue transglutaminase antibody**. This patient's presentation of an intensely itchy, symmetrical vesicular rash on the knees and back of arms is highly suggestive of dermatitis herpetiformis. Dermatitis herpetiformis is an autoimmune skin condition associated with coeliac disease, and it often presents as a pruritic, blistering rash on extensor surfaces such as the elbows, knees, and buttocks. The presence of anti-tissue transglutaminase antibodies confirms the diagnosis as these antibodies are commonly found in patients with coeliac disease.

Anti-mitochondrial antibody is incorrect because these antibodies are typically associated with primary biliary cholangitis (PBC), a chronic liver disease characterised by destruction of intrahepatic bile ducts. PBC usually presents with fatigue, pruritus, and jaundice rather than a vesicular rash.

Anti-nuclear antibody (ANA) is also incorrect because ANAs are generally associated with systemic autoimmune diseases such as systemic lupus erythematosus (SLE) or Sjogren's syndrome. Although these conditions can present with skin manifestations like rashes, they do not typically present with the characteristic vesicular rash seen in dermatitis herpetiformis.

Anti-neutrophil cytoplasmic antibody (ANCA) is not correct either because ANCAs are primarily associated with vasculitides like granulomatosis with polyangiitis (GPA), microscopic polyangiitis (MPA), and eosinophilic granulomatosis with polyangiitis (EGPA). These conditions can have cutaneous manifestations like purpura or nodules but not the specific vesicular rash seen in this patient.

Lastly, **Anti-Jo-1 antibody** is incorrect because Anti-Jo-1 antibodies are associated with idiopathic inflammatory myopathies, particularly polymyositis and dermatomyositis. While dermatomyositis does present with skin manifestations like a heliotrope rash or Gottron's papules, these are distinct from the vesicular rash seen in dermatitis herpetiformis.

In summary, the presence of **Anti-tissue transglutaminase antibody** is most likely in this patient due to the characteristic presentation of dermatitis herpetiformis, which is associated with coeliac disease.

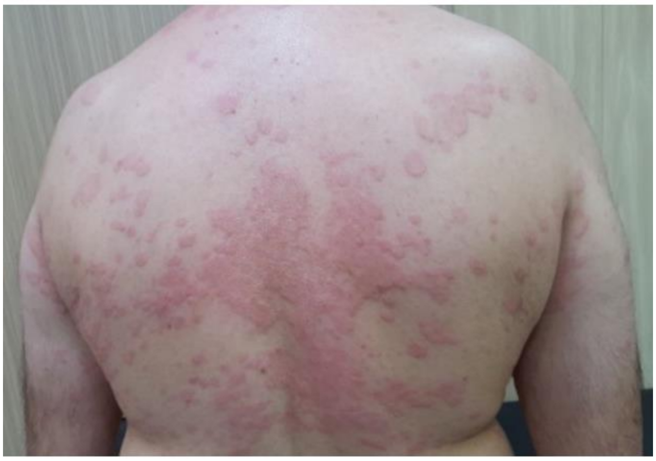
Question 50 of 182

A 25-year-old man presents with a rash predominantly affecting his back. The rest of his skin is unaffected.

The rash is not painful, although is very itchy. He had a similar occurrence last week but the rash resolved spontaneously within the same day. He denies any shortness of breath, and there is no swelling surrounding his mouth. He has not tried any new foods or started any new medicines. There is no past medical history of note, no regular medicines, and no recent illness.

His observations are as follows:

- Respiratory rate: 12 /min
- Saturations: 98% on room air
- Heart rate: 64bpm
- Blood pressure: 122/74mmHg
- Temperature: 37.2 deg C



Given the likely diagnosis, which is the most appropriate next step in management?

A. Diphenhydramine
B. Epinephrine
C. Fexofenadine
D. Omalizumab
E. Prednisolone

C. Fexofenadine

The picture here shows the superficial irregular raised swellings/plaques surrounded by erythema that are characteristic of urticaria.

Urticaria can be classed based on its duration as acute (≤6 weeks) or chronic (>6 weeks). It can also be further subdivided as either spontaneous (with no definite trigger involved) or inducible (specific definite trigger involved). This man is presenting with acute spontaneous urticaria without signs of anaphylaxis or angioedema.

Acute urticaria is generally self-limiting. As such, it usually does not require a diagnostic workup apart from enquiring about possible trigger factors. Acute urticarias that may require further diagnostic workup would include those likely due to a type I food allergy or drug hypersensitivity. In these instances, allergy tests and patient education would play a role, enabling patients to avoid re-exposure to causative factors.

Here, there is no suggestion this is the case and so initial management is symptom-based. The predominant symptom is pruritus, so the focus is on short-term management of this.

Fexofenadine is the most appropriate of the given options as it is a second-generation antihistamine and so is non-sedating.

Diphenhydramine is a first-generation anti-histamine, and so is sedative and to be avoided in the first instance. It could be considered in addition to the above if there is an inadequate response to the first-line antihistamine treatment, and itch is interfering with sleep.

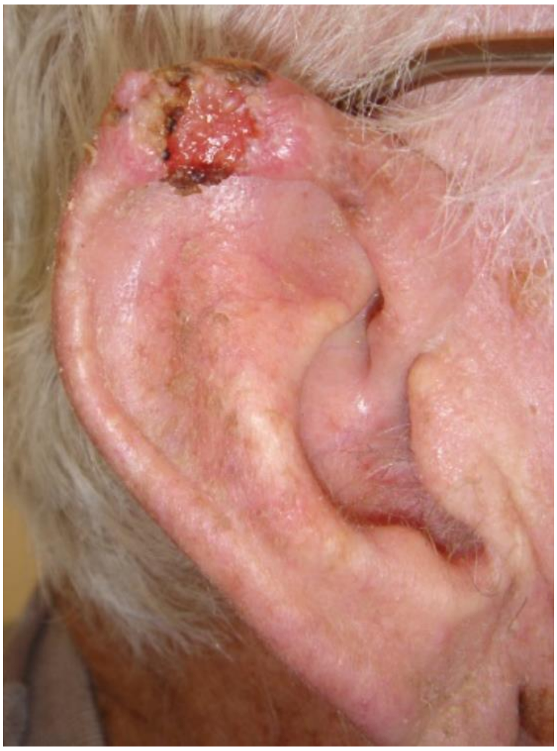
Omalizumab is a monoclonal anti-immunoglobulin E antibody that is used in the treatment of chronic idiopathic urticaria, however would not be the first line in this instance.

Prednisolone as a short course could be given if symptoms were considered particularly severe, in addition to the non-sedating oral antihistamine. This patient has only a relatively small area of skin involved.

Finally, whilst anaphylaxis is something that should always be ruled out with urticaria, the above patient does not have any other features to suggest this and so **epinephrine** is not indicated here.

Question 51 of 182

An 85-year-old man complains of a persistent 'scab' on his right ear. This has been present for the past year and causes some discomfort if he lays on his right side at night. It is mildly pruritic. His past medical history includes having an actinic keratosis removed from his left temple two years ago. A number of seborrhoeic keratoses are also noted across his body.

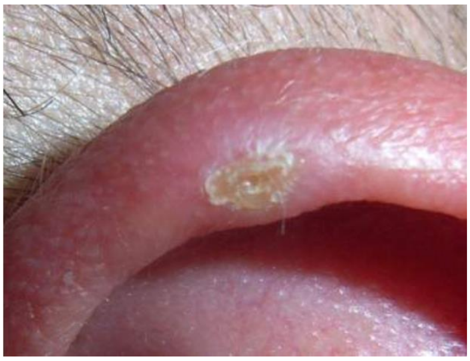


What is the most likely diagnosis?

A. Marjorin's ulcer
B. Squamous cell carcinoma
C. Basal cell carcinoma
D. Chondrodermatitis nodularis helicis

B. Squamous cell carcinoma

The main differential diagnosis here is chondrodermatitis nodularis helicis, however, this has a very different typical appearance - much more well defined with regular features. A typical lesion is shown below:

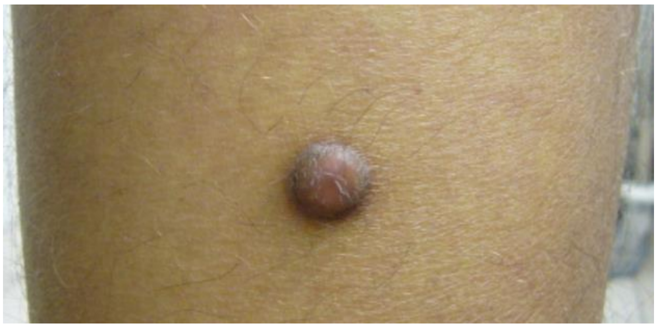


Typical appearance of chondrodermatitis nodularis helicis

Question 52 of 182

A 48-year-old male presents with a solitary skin nodule on his forearm which he first noticed a few weeks ago. He reports he was bitten by some form of insect prior to noticing the skin nodule.

On questioning, he describes the site as original very itchy with some tenderness after the bite but by the time he noticed the skin lesion, the area was and has remained, non-painful with only a mild itch.



He is otherwise well with no systemic symptoms.

What is the most likely diagnosis?

A. Basal-cell carcinoma
B. Dermatofibroma
C. Prurigo nodularis
D. Leiomyoma
E. Keratoacanthoma

B. Dermatofibroma

This patient has presented with a dermatofibroma, also called a histiocytoma. These are fibrous solitary, slow-growing papules caused by abnormal growth of dermal dendritic histiocyte cells. As seen in the picture they are raised and normal brown in colour. Although they can be itchy they are rarely painful and are associated with 'dimple sign', where applying lateral pressure produces a central depression. They can occur anywhere but are commonly seen on the arms and leg as they are often precipitated by an injury such as an insect bite (as with this patient) or a thorn prick.

Basal-cell carcinomas (BCC) are the most common form of skin cancer and often appear over several months, in sun-exposed areas. They are normally painless, slightly raised/macular areas of skin with a shiny, vascular appearance. BCC are rarely symmetrical and are unrelated to previous skin trauma.

Prurigo nodularis, also known as nodular prurigo, is a dermatological condition characterised by multiple, pruritic nodules commonly affecting the legs and arms. Unlike in dermatofibroma, itching only develops after the nodules have formed and increases in intensity as the condition progresses. Due to their pruritic nature patients commonly present with multiple excoriated lesions due to scratching and symptoms may only be alleviated by steroid use.

A leiomyoma, also known as a fibroid, is a smooth muscle tumour that is most commonly benign, only progressing to cancer in extremely rare cases (0.1%). As they arise in the smooth muscle they are seen within organs, commonly the uterus, small bowel, or oesophagus. Cutaneous leiomyomas are rare but when they do occur they are seen as multiple, freely movable nodules seen with overlying reddish skin.

A keratoacanthoma is a common, normally benign skin lesion that is thought to originate from the hair follicle. They are often dome-shaped and symmetrical, similar to a dermatofibroma however keratoacanthomas are surrounded by a smooth wall of inflamed skin and have a characteristic cap made of keratin scales and debris. The exact cause of keratoacanthomas is unclear but they are believed to be linked to ultraviolet light, chemical carcinogens and immunosuppression.

Question 53 of 182

A 19-year-old man presents to the dermatology clinic with his parents. He has a background of learning difficulties and eczema. His parents have noticed that he has had difficulty in controlling his eczema with topical emollients. He has previously had several episodes of infections of his eczema, but it is free of infection currently. On examination, his flexural folds are erythematous with clear discharge. There are no pungent odours. He has been using E45 cream three times a day with the assistance of his parents.

His parents wonder what else could be done to help control his eczema?

A. Change E45 to a urea containing emollient
B. Long-term prophylactic antibiotics
C. Oral steroids
D. Bandaging after emollient use
E. Topical immunosuppressant therapy

D. Bandaging after emollient use

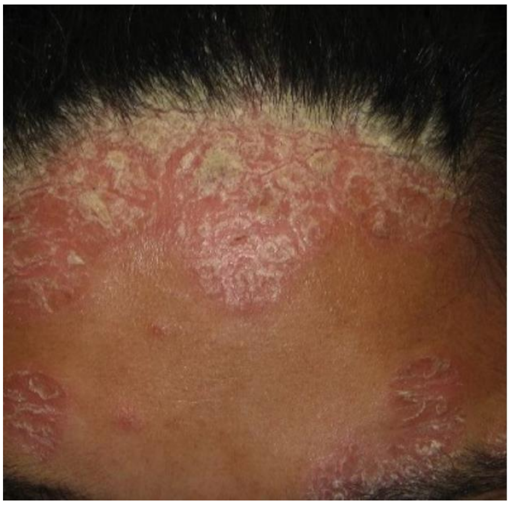
Bandaging can help control itching in eczema to prevent further scratching

The most appropriate action is the use of bandaging. Bandaging in eczema can be used in noninfected patients for weeping or lichenified skin in combination with emollients and, if needed, topical corticosteroids. This patient's weeping skin could be helped by bandaging to help absorb the discharge and enhance the emollient effect.

Oral steroids and topical immunosuppressant therapy are only used in severe cases or when topical treatment has failed. Using a urea-containing topical agent can help with itching and moisturising effect, but can provoke further inflammation and would, therefore, be inappropriate for this patient. Long-term antibiotics are not used for eczema.

Question 54 of 182

A 28-year-old woman with a history of psoriasis presents with dandruff and an itchy scalp. She has tried using 'Head & Shoulders' with limited effect. A photograph of her scalp problems is shown below:



What is the most appropriate first-line treatment?

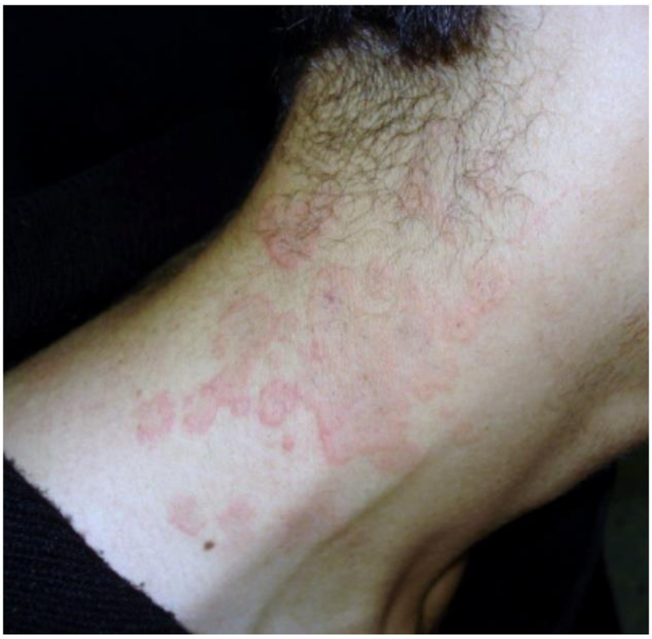
A. A coal tar based shampoo
B. A ketoconazole based shampoo
C. Topical beclomethasone
D. Topical calcipotriol
E. Topical calcipotriol + beclomethasone

C. Topical beclomethasone

Scalp psoriasis - first-line treatment is topical potent corticosteroids

Question 55 of 182

A 44-year-old woman visits her general practitioner with a 48-hour history of a new, erythematous rash on her neck. She described initially noticing lesions on the backs of her hands the day before. The rash is mildly itchy but not painful. There is no past medical history of note other than taking a course of antibiotics for a lower respiratory tract infection, diagnosed 4 days earlier. Her rash is examined, as pictured below:



What is the most likely diagnosis?

A. Dermatitis herpetiformis
B. Erythema multiforme
C. Guttate psoriasis
D. Pityriasis rosea
E. Tinea corporis

B. Erythema multiforme

This patient has multiple, round erythematous lesions on her neck. On closer inspection, the lesions appear as the classical target lesions with an outermost erythematous ring and paler centre. This is in keeping with a diagnosis of erythema multiforme. Erythema multiforme is an acute, self-limiting skin eruption most commonly secondary to an infective trigger (e.g. herpes simplex virus or *Mycoplasma pneumoniae*). The rash appears abruptly and affects the upper limbs most often before spreading down towards the trunk. The ongoing lower respiratory tract infection points toward an infection with *Mycoplasma pneumoniae*, the second most common infective cause of erythema multiforme.

Dermatitis herpetiformis is an itchy vesicular rash commonly associated with coeliac disease. It typically has a symmetrical distribution appearing on the elbows, knees, shoulders and buttocks. The rash seen in the above image is not symmetrical, nor does it appear vesicular. Furthermore, the absence of a history of autoimmune disease or coeliac disease makes dermatitis herpetiformis unlikely.

Guttate psoriasis is a subtype of psoriasis that commonly occurs after a streptococcal infection and is characterised by multiple small scaly plaques on the trunk and limbs. The onset of guttate psoriasis can be rapid and occur within days of initial infection. Although this patient has had a recent bacterial infection, the appearance of this rash is not scaly, but instead, shows target lesions in keeping with erythema multiforme.

Pityriasis rosea is a viral rash that is characterised by an initial herald patch followed by multiple red plaques developing over the trunk. The rash is usually asymptomatic with the only symptoms present being of an underlying viral infection (e.g. cough and coryza). There is no history of a herald patch in this question and the location and appearance of the lesions are not in keeping with pityriasis rosea.

Tinea corporis or ringworm is a fungal skin infection capable of affecting most parts of the body. The rash initially presents as a single well-demarcated, erythematous circular patch with a raised edge and central hypopigmentation. Itching is a prominent clinical feature. it does not classically cause multiple defined lesions as shown in this picture and tends to have a slower onset of development.

Question 56 of 182

A 45-year-old woman presents to her GP with an ulcer on her left shin. It has been present for the past 4 months and gradually got bigger. It is tender and yellow-brown in colour and there appears to be another smaller lesion that is growing next to the bigger one. She has a past medical history of multiple sclerosis, of which she is currently in remission phase and type 1 diabetes mellitus. Her only regular medication includes insulin.

What is the most appropriate treatment?

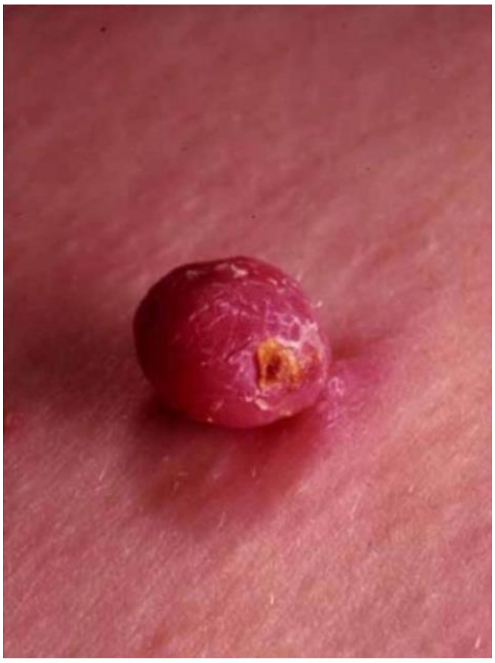
A. Flucloxacillin
B. Topical corticosteroids
C. TED stockings
D. Oral corticosteroids
E. Topical tacrolimus

B. Topical corticosteroids

The most likely diagnosis is necrobiosis lipoidica, a rare granulomatous skin disorder which can affect the shin of insulin dependent diabetics. Skin biopsy can confirm the diagnosis, although it is often done clinically and topical steroids are a useful treatment, along with injectable corticosteroids and camouflage creams.

Question 57 of 182

A 19-year-old woman asks you to have a look at a 'tag' on her neck. It developed around two months ago and has bled on a number of occasions after catching it.



What is the most likely diagnosis?

A. Pyogenic granuloma
B. Capillary haemangioma
C. Vital wart
D. Malignant melanoma
E. Molluscum contagiosum

A. Pyogenic granuloma

The most likely diagnosis for this patient is a **Pyogenic granuloma**. Pyogenic granulomas are benign skin lesions that often appear as small, reddish bumps on the skin. They can develop rapidly over a few weeks and tend to bleed easily because they contain many blood vessels. The history of this lesion developing over two months and bleeding after minor trauma is consistent with a pyogenic granuloma.

A **Capillary haemangioma**, also known as a strawberry naevus, is an incorrect choice. These usually present at birth or shortly thereafter and grow rapidly before starting to regress by 1-2 years of age. They are rarely seen in adults and do not typically bleed unless traumatised.

The option of **Viral wart** is also incorrect. Viral warts caused by human papillomavirus (HPV) are usually rough, hard, and non-bleeding unless injured.

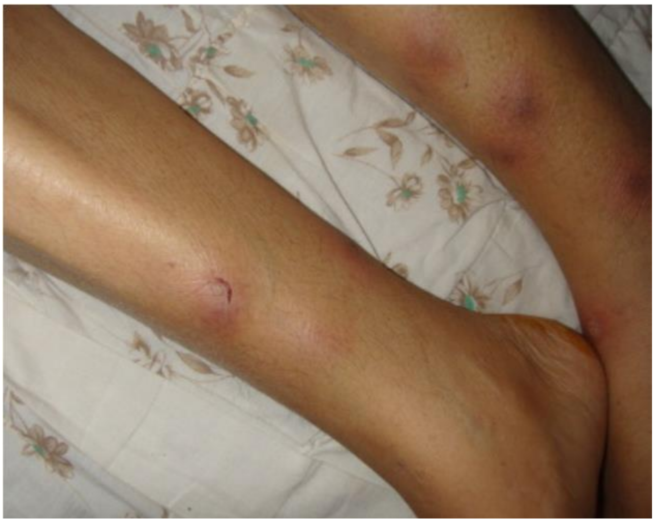
Malignant melanoma is unlikely given the patient's history and clinical presentation. Melanomas usually have irregular borders, vary in colour from one area to another, have a diameter larger than 6mm and evolve over time (ABCDE criteria). Furthermore, malignant melanomas don't typically bleed unless they ulcerate or are traumatised.

Finally, **Molluscum contagiosum** is also an incorrect choice. Molluscum contagiosum presents as multiple small, dome-shaped papules with a central umbilication caused by a poxvirus. It does not generally cause bleeding but may become inflamed or itchy. The solitary nature of this lesion makes molluscum contagiosum less likely.

Question 58 of 182

A 35-year-old woman who was born in Eastern Europe presents with a 5-day history of skin changes on her legs, as shown in the image below. She has a past medical history of asthma and type I diabetes. She has had a recent illness with persisting sore throat, myalgia and fatigue for 10 days.

There are no skin changes elsewhere in the body. She has some mild arthralgia affecting the knees and ankles on both sides, but no effusion or erythema affecting the joints.



What is the most likely precipitating cause?

A. Haemophilus influenzae infection
B. Epstein Barr virus
C. Sarcoidosis
D. Streptococcal throat infection
E. Tuberculosis

D. Streptococcal throat infection

This patient has erythema nodosum (EN) - as shown by the several raised erythematous tender lesions on her shins. This is a classical appearance of erythema nodosum, which is a delayed-typehypersensitivity reaction, causing inflammation of the subcutaneous fat. Lesions are most common on the shins, but may also be found on the thighs, buttocks, or forearms. Erythema nodosum is more common in women than in men. There are numerous potential triggers for erythema nodosum, which can be broadly grouped into infectious, inflammatory, drug, or malignancy. In many cases, a specific trigger is never found.

The correct answer is **Streptococcal throat infection**. The most common trigger for erythema nodosum is a preceding infection with a beta-haemolytic Streptococcus, which is usually pharyngitis caused by *Streptococcus pyogenes* (Group A Strep). It is thought that 25-45% of all cases of erythema nodosum are due to Streptococcal throat infections.

Epstein Barr virus is incorrect. This virus is the causative agent of infectious mononucleosis. It can trigger EN but it is a less common cause than Streptococcal infections and accounts for <1% of cases.

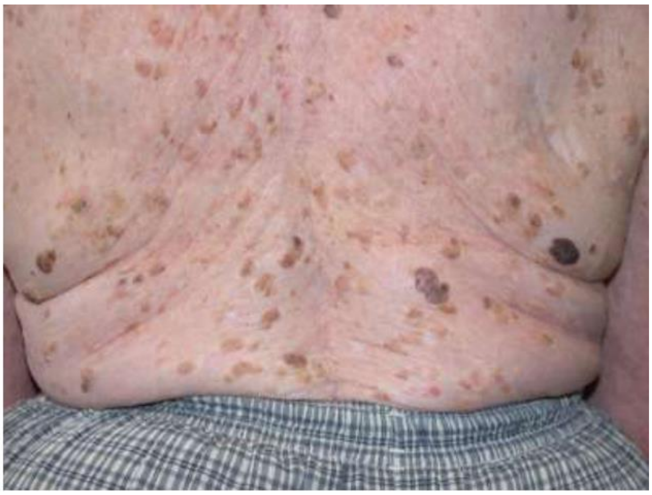
Haemophilus influenzae is incorrect. This is a common cause of upper respiratory tract infections but has no significant association with erythema nodosum.

Sarcoidosis is incorrect. Sarcoidosis is associated with around 10-25% of cases of EN, which is less than Streptococcal throat infections.

Tuberculosis is incorrect. TB and other mycobacteria can be associated with EN but the association is much less frequent than with Streptococcal infection.

Question 59 of 182

A 74-year-old man is admitted with an infective exacerbation of chronic obstructive pulmonary disease. Whilst auscultating his chest you notice a number of lesions on his back:



What is the most likely diagnosis?

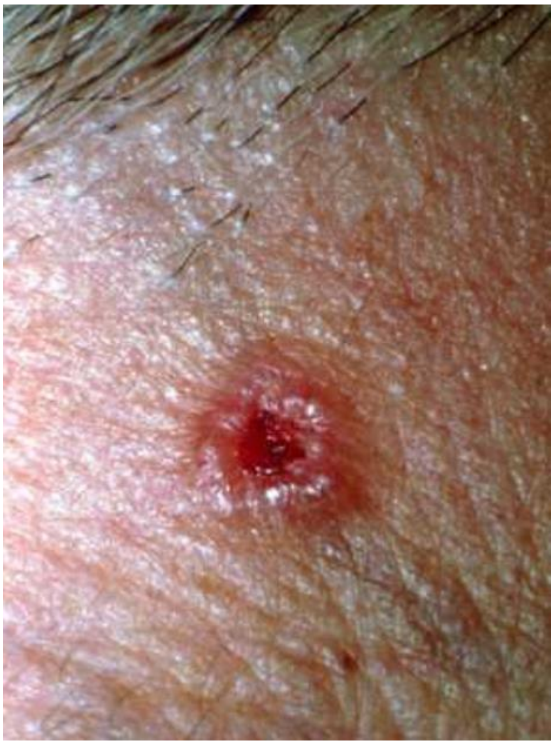
A. Bowen's disease
B. Multiple benign moles
C. Metastatic malignant melanoma
D. Seborrhoeic keratoses
E. Dysplastic naevus syndrome

D. Seborrhoeic keratoses

This man has multiple seborrhoeic keratoses, also known as basal cell papillomas.

Question 60 of 182

Please look at the skin lesion shown below:



Which one of the following statements regarding this type of skin lesion is true?

A. Curettage is an acceptable treatment option
B. They exhibit the Koebner phenomenon
C. They typically grow rapidly
D. Bleeding is unusual
E. Metastases are present in 10% of patients at the time of diagnosis

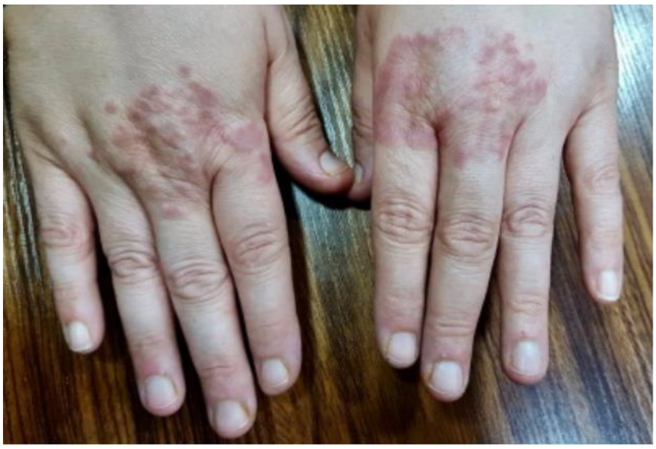
A. Curettage is an acceptable treatment option

Question 61 of 182

A 60-year-old homeless female is brought to the emergency department intoxicated. You note from her medical records she had diabetes mellitus only. You also note that her blood glucose levels are frequently high on attendances.

Her examination, other than the intoxication, is normal and observations are all within normal range except a slightly raised blood glucose.

After a few hours she beginnings to sober up and is preparing to leave the department. Prior to leaving, she asks you to have a look at the rash on the back of her hands (see below). She reports she has similar, mildly itchy rashes on her elbows and the dorsal of her feet.



What is the most likely diagnosis?

A. Dermatitis herpetiformis
B. Erythema migrans
C. Granuloma annulare
D. Necrobiosis lipoidica
E. Scabies

C. Granuloma annulare

Granuloma annulare is a papular rash often with slightly hyperpigmented lesions which have depressed centres. They are commonly found on the dorsal surfaces of the hands and feet as well as the elbows and knees. The lesions are sometimes mildly itchy and are through to be due to clustering of T cells below the skin. Granuloma annulare is 4x more prevalent in females and is thought to be linked to diabetes but there is limited evidence to confirm this.

Dermatitis herpetiformis is a cutaneous manifestation of coeliac disease, characterised by an intensely itchy, papulovesicular like rash, with blisters filled with watery fluid. The rash is similar in appears to a herpes rash (hence the name) and is normal found on the trunk.

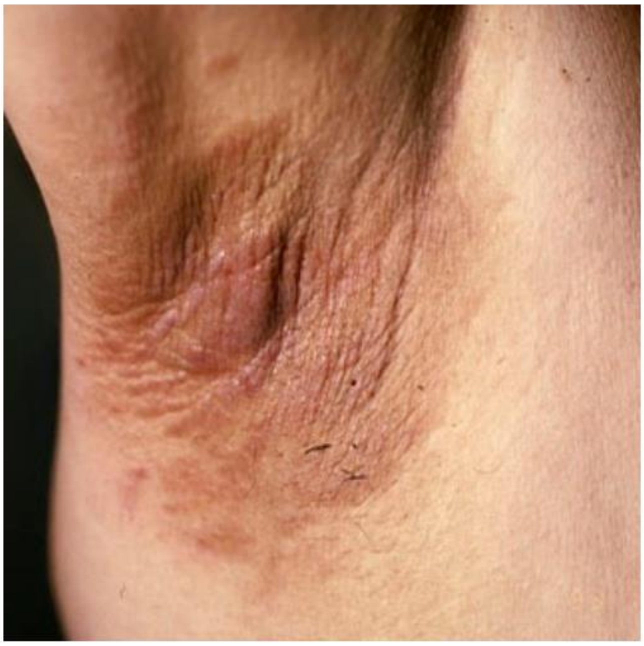
Erythema migrans is an expanding erythematous plaque with central clearing, creating a 'bull's eye/target' like rash, often associated with the early stages of Lyme disease. The skin lesions occur at the site of a tick bite, normal around skin folds or on the trunk, and as such patient's common present following outdoor activities in a woodland area.

Necrobiosis lipoidica is a dermatological feature of a few conditions but is most commonly associated with diabetes. It presents as an initially asymptomatic, necrotising rash normally found on the shins which can become tender and ulcerate when injured.

Scabies is an intensely itchy, rash with associated small burrow lesions, caused by the mite *Sarcoptes scabiei*. The rash is normally found in the finger webs but also around the feet, ventral wrists, elbows, trunk, and external genitals.

Question 62 of 182

A 56-year-old woman develops a rash in both axilla:



What is the most likely diagnosis?

A. Pellagra
B. Erythema gyratum repens
C. Hidradenitis suppurativa
D. Tinea corporis
E. Acanthosis nigricans

E. Acanthosis nigricans

The correct answer is **Acanthosis nigricans**. Acanthosis nigricans is a skin condition characterised by dark, thickened patches of skin that can appear in various parts of the body including the axilla. The image shows a velvety, hyperpigmented rash which is typical of acanthosis nigricans. It's often associated with insulin resistance and can be an early sign of type 2 diabetes.

Pellagra is incorrect because it typically presents with the 'three Ds': dermatitis, dementia, and diarrhoea. The dermatitis caused by pellagra usually occurs on areas exposed to sunlight and takes on a symmetrical, butterfly-shaped appearance on the face. It does not typically present as darkened patches in the axilla.

Erythema gyratum repens is also incorrect. This condition produces concentric circles or waves of erythema that migrate across the skin surface, giving it a wood-grain appearance. These patterns are not visible in the provided image.

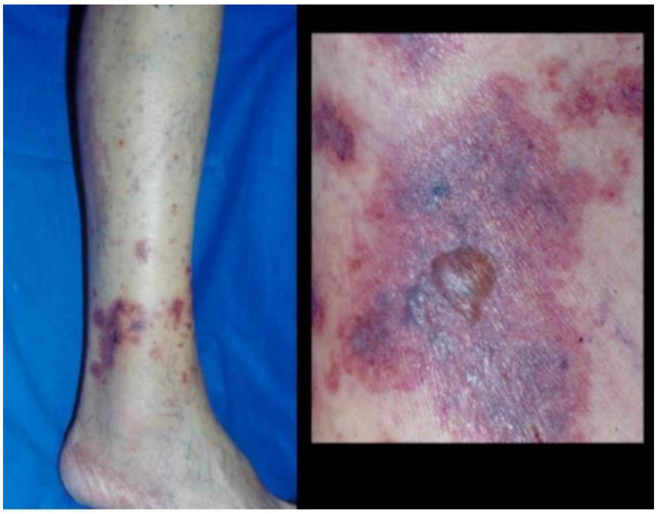
Hidradenitis suppurativa would be an incorrect choice as well because this chronic inflammatory skin condition causes painful lumps under the skin. These lumps or abscesses occur near hair follicles where many oil and sweat glands are found such as in the armpits or groin area. However, they do not resemble the darkened patches seen in this patient's axillae.

Lastly, **Tinea corporis** (ringworm) is also an incorrect option. Tinea corporis presents with circular rashes with raised borders that expand over time - often leaving clear or normal-looking skin in the centre which gives it a ring-like appearance hence its common name 'ringworm'. This pattern cannot be seen in this image either.

Question 63 of 182

A 60-year-old man has been an inpatient in a general medical ward for 2 weeks. He presented with a 10-day history of fever and malaise. He developed lesions on his lower legs the day before his admission. Despite the administration of broad-spectrum antibiotics since admission, he is continuing to have temperatures greater than 38ºC on a daily basis. The rest of his observations remain stable.

On examination, his skin appears as shown below:



Blood tests are taken with the following results:

Hb	108 g/L	Male: (135-180)Female: (115 - 160)
Platelets	114 * 10 /L 9	(150 - 400)
WBC	14.5 * 10 /L 9	(4.0 - 11.0)
Na +	138 mmol/L	(135 - 145)
K +	4.2 mmol/L	(3.5 - 5.0)
Urea	9.5 mmol/L	(2.0 - 7.0)
Creatinine	145 µmol/L	(55 - 120)
CRP	120 mg/L	(< 5)

What is the most likely cause for this patient's skin changes?

A. Drug reaction
B. Haemolytic uraemic syndrome
C. Kaposi's sarcoma
D. Small vessel vasculitis
E. Thrombocytopenia

D. Small vessel vasculitis

The image shows a number of petechial and purpuric lesions of different sizes on the lower legs. This is a classic cutaneous presentation of the small vessel vasculitides, which can cause inflammation of the blood vessels in the skin, as well as elsewhere in the body.

The correct answer is **small vessel vasculitis** . This is a group of conditions, including ANCAassociated vasculitides, cryoglobulinaemia and Henoch-Schonlein purpura. They may often present with non-specific symptoms, including as pyrexia of unknown origin as in this scenario. The raised creatinine also points to a systemic condition.

Drug reaction is incorrect. Skin manifestations of drug reactions are varied, and drug-induced small vessel vasculitis can occasionally occur but this is rare. The patient also developed the lesions before his admission to the hospital, making a drug reaction less likely. This would also not explain the rest of his symptoms.

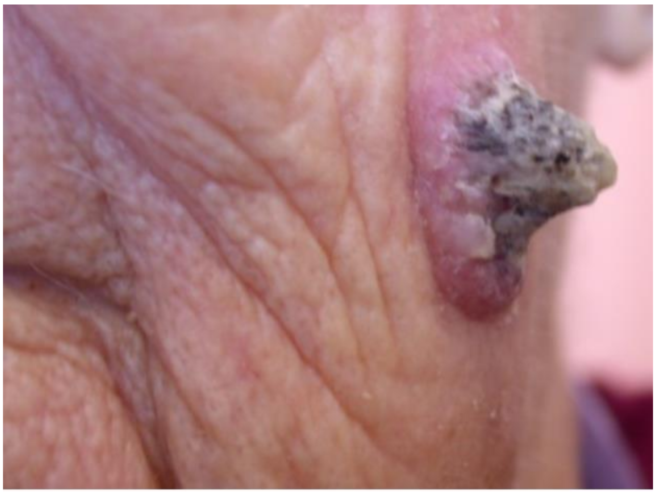
Kaposi's sarcoma is incorrect. This condition causes red to purplish lesions on the skin, but they tend to be solitary or few in number in the early stages, and would not be on a background of numerous small lesions as in this image. The progression of lesions also tends to be much slower than in this patient.

Haemolytic uraemic syndrome (HUS) is incorrect. This condition can cause a purpuric rash due to thrombocytopenia. It is unlikely in this patient as the renal function is only mildly abnormal, thrombocytopenia is mild and there is no clear evidence of haemolytic anaemia. HUS is also much more common in children than in adults. There is also no history of diarrhoea given in this patient, and HUS is usually triggered by infection with a verotoxin-producing strain of *E.coli* causing diarrhoea before the onset of HUS.

Thrombocytopenia is incorrect. Low platelets can cause petechiae and purpura due to bleeding into the skin, but this is unlikely with relatively mild thrombocytopenia as in this patient.

Question 64 of 182

An elderly man presents with a lesion on his cheek that has got progressively worse over the past year. He asks for it to be removed as it is catching:



What is the most likely diagnosis?

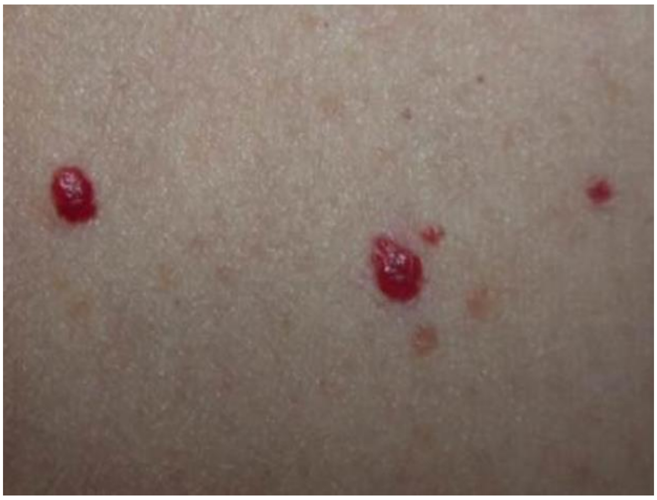
A. Nodular malignant melanoma
B. Viral wart
C. Solar keratosis
D. Seborrhoeic keratosis
E. Squamous cell carcinoma

E. Squamous cell carcinoma

The main differential diagnosis here is keratoacanthoma which are often described as 'minivolcanos' with a central core filled with keratin.

Question 65 of 182

A 55-year-old man asks you to have a look at some 'red spots' on his torso. They have been present for about the past six months.



What is the most likely diagnosis?

A. Kaposi sarcoma
B. Blue rubber bleb naevus syndrome
C. Thrombocytopaenia
D. Malignant melanoma
E. Cherry haemangioma

E. Cherry haemangioma

The most likely diagnosis for the 'red spots' on the 55-year-old man's torso is **Cherry haemangioma**. This condition is characterized by small, bright red raised bumps that are usually found on the skin of adults aged over 30 years. The 'red spots' in the image provided are consistent with this description. Cherry haemangiomas are benign vascular proliferations and do not require treatment unless they become symptomatic or for cosmetic reasons.

Kaposi sarcoma is incorrect because it typically presents as purple, red, or brown blotches or tumours on the skin. It is a rare type of cancer that can occur in people with weakened immune systems such as those with AIDS or who have had organ transplants, which does not seem to be applicable to this patient based on the information given.

Blue rubber bleb naevus syndrome is also unlikely as it involves venous malformations that appear as compressible blue nodules and can occur anywhere on the body including internal organs. These lesions are typically blue rather than red and often present at birth or during early childhood.

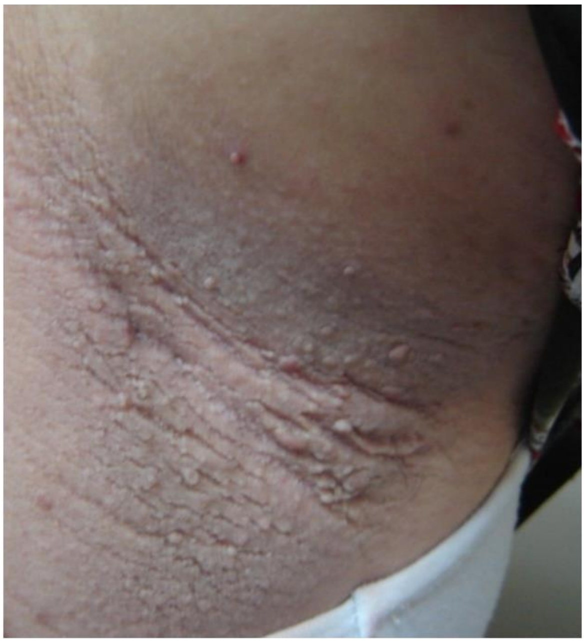
The option of **Thrombocytopaenia**, which translates to low platelet count, would usually present with petechiae (small red or purple spots), purpura (larger areas of bleeding into skin), easy bruising or mucosal bleeding rather than these isolated 'red spots'.

Finally, **Malignant melanoma** is an aggressive form of skin cancer that often appears as a new mole or changes in an existing mole rather than presenting as multiple 'red spots'. Melanomas typically have irregular borders, variations in colour within one lesion, larger diameter and evolving changes over time. This presentation does not match the image provided.

Question 66 of 182

A 50-year-old man presents to the dermatology clinic with a 1-month history of axillary skin discolouration. He finds it unpleasant to look at and is now complaining of increasing pruritus. He has no past medical history of note and smokes 15-20 cigarettes/day.

On examination, he is afebrile with a heart rate of 76 beats/min and a blood pressure of 130/65mmHg. His skin is examined, as shown below:



Given the diagnosis, what is most likely to be associated with this presentation?

A. Acromegaly
B. Acute lymphoblastic leukaemia
C. Addison's disease
D. Coeliac disease
E. Diabetes insipidus

A. Acromegaly

Acromegaly is correct. This patient has a diagnosis of acanthosis nigricans characterised by hyperkeratosis and hyperpigmentation of the skin, predominantly affecting the flexural skin folds such as the axillae and groin. The skin may have a velvety texture secondary to papillomatous overgrowth of the epidermis. Common underlying causes include metabolic diseases such as obesity, type 2 diabetes mellitus, polycystic ovarian syndrome and acromegaly.

Acute lymphoblastic leukaemia is incorrect. Haematological malignancies are associated with Sweet syndrome, characterised by fever and inflamed and blistered skin with mucosal involvement.

Addison's disease is incorrect. Due to excess ACTH, patients with Addison's disease may have the appearance of tanned skin due to the stimulation of melanocytes. However, it does not cause acanthosis nigricans.

Coeliac disease is incorrect. Coeliac disease is associated with dermatitis herpetiformis, a vesicular rash commonly seen on the extensor surfaces of the skin. This is not the rash that is demonstrated here.

Diabetes insipidus is incorrect. Diabetes mellitus is associated with acanthosis nigricans. However, diabetes insipidus is not associated with any dermatological manifestations.

Question 67 of 182

A 68-year-old lady is reviewed in the cardiology outpatient clinic following a recent admission for a non-ST-elevation myocardial infarction (NSTEMI). An echocardiogram performed during her inpatient stay showed mild-moderate LV systolic dysfunction with an ejection fraction of 40%.

Since discharge, she has been feeling well and has experienced no further episodes of chest pain. She reports only mild breathlessness on climbing two flights of stairs but draws your attention to an itchy rash on her lower legs. On examination, there are tense blisters covering her lower limbs bilaterally with minimal underlying oedema.

Which of her medications is most likely to be implicated?

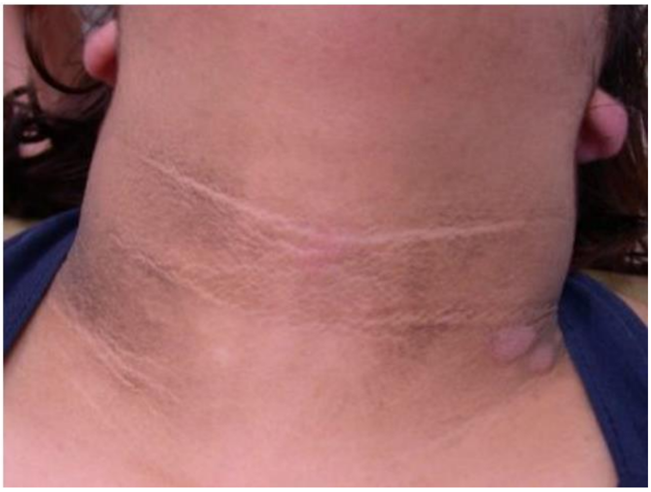
A. Spironolactone
B. Clopidogrel
C. Bisoprolol
D. Furosemide
E. Ramipril

D. Furosemide

The patient has developed bullous pemphigoid, of which furosemide is a classic cause. Penicillamine, captopril, and penicillin derivatives are other common causes. The condition may remit or even disappear on cessation of the offending agent.

Question 68 of 182

You notice an abnormality on the neck of a 40-year-old woman:



Which one of the following is most associated with this appearance?

- A. Lung cancer
- B. Acute pancreatitis
- C. Haemochromatosis
- D. Polycystic ovarian syndrome
- E. Digoxin use

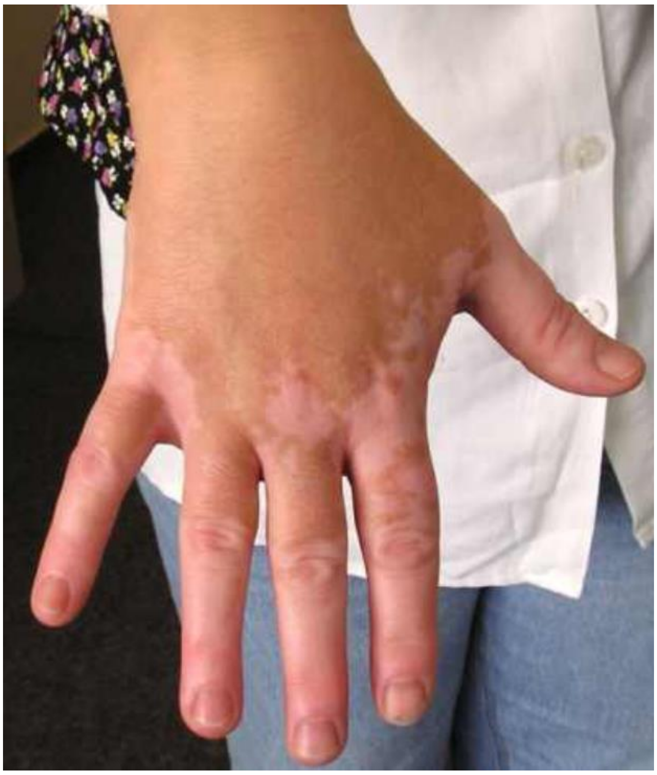
D. Polycystic ovarian syndrome

This patient has acanthosis nigricans which is associated with a number of hyperinsulinaemia states such as polycystic ovarian syndrome.

Whilst acanthosis nigricans can be associated with any type of cancer by far the most common malignant cause is gastrointestinal adenocarcinoma.

Question 69 of 182

A 21-year-old woman with a history of eczema presents with a change in the colour of her skin affecting the hands and feet symmetrically:



What is the most likely diagnosis?

- A. Excessive topical corticosteroid use
- B. Leprosy
- C. Tuberous sclerosis
- D. Vitiligo
- E. Pityriasis versicolor

D. Vitiligo

The correct answer is **Vitiligo**. Vitiligo is a condition that causes loss of skin colour in patches. It can affect the skin on any part of your body and may also affect hair, the inside of the mouth and even the eyes. The main symptom is a loss of pigment which produces white patches on the skin, often symmetrical, as seen in this patient.

The first incorrect option, **Excessive topical corticosteroid use**, can lead to changes in skin colour, but these are typically characterised by thinning of the skin and formation of stretch marks rather than depigmentation. Additionally, excessive use would likely not produce such symmetrical patterns.

Leprosy, another incorrect option, is an infectious disease caused by *Mycobacterium leprae*. It usually presents with hypopigmented or reddish patches with a definite loss of sensation. However, it's rare in the UK and does not usually present symmetrically.

Tuberous sclerosis is a genetic disorder that causes non-cancerous (benign) tumours to grow in many parts of your body. Skin manifestations include hypomelanotic macules (white patches), facial angiofibromas or shagreen patches but these do not have a symmetrical distribution.

Lastly, **Pityriasis versicolor** is an infection caused by yeast from the Malassezia family. This condition causes small, scaly patches of discoloured skin that might be lighter or darker than the surrounding skin. However, unlike vitiligo, pityriasis versicolor does not cause complete depigmentation and it tends to affect the trunk more commonly than the hands and feet.

Question 70 of 182

A 72-year-old woman presents with a new skin marking. She has noticed it recently and is fairly sure it was not present six months ago. It has not caused her any symptoms and has not been itching. On examination, it is a raised, slightly ulcerated, 7mm diameter lesion on her chest.

What clinical finding would make squamous cell carcinoma more likely than basal cell carcinoma?

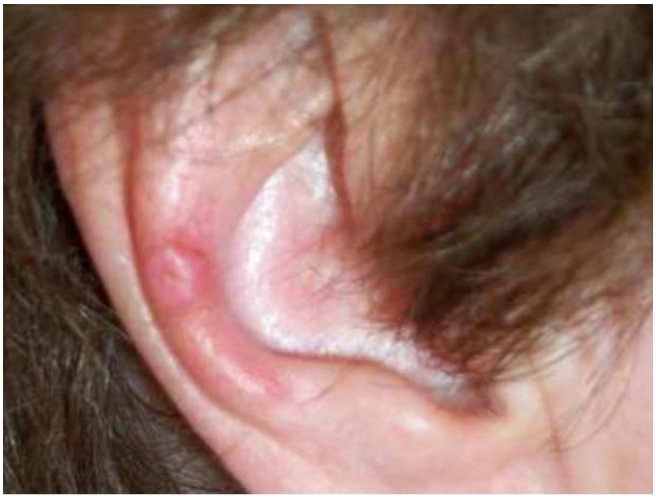
A. Telangiectasia
B. Not associated with ulceration
C. Pearly appearance
D. Erythematous base
E. Nodular edge

D. Erythematous base

The correct answer is erythematous base. Squamous cell cancers present as crusty enlarging lumps on a background of acinic keratosis on sun-exposed skin. They may ulcerate and are often tender or painful. Basal cell cancers usually have telangiectasia, pearly appearance and have a nodular edge.

Question 71 of 182

A 60-year-old man presents with a painful lesion on his right ear:



What is the most likely diagnosis?

A. Actinic keratosis
B. Pseudocyst of the auricle
C. Chondrodermatitis nodularis helicis
D. Basal cell carcinoma
E. Keratoacanthoma

C. Chondrodermatitis nodularis helicis

The correct answer is **Chondrodermatitis nodularis helicis**. This condition typically presents as a painful, inflamed nodule on the helix or antihelix of the ear. It most commonly affects middle-aged or elderly individuals and is thought to be related to pressure and sun exposure. The image provided shows a characteristic lesion of Chondrodermatitis nodularis helicis.

Moving on to the incorrect options:

Actinic keratosis is a precancerous skin condition characterised by scaly, crusty spots on the skin caused by damage from the sun's ultraviolet (UV) rays. While it may present in similar areas such as face, lips, ears, back of your hands, forearms, scalp or neck due to sun exposure, it does not typically cause painful lesions.

Pseudocyst of the auricle, also known as auricular pseudocyst, presents as a non-inflammatory, fluid-filled swelling on the anterior aspect of the pinna. It's usually painless unless infected and does not appear erythematous like in this case.

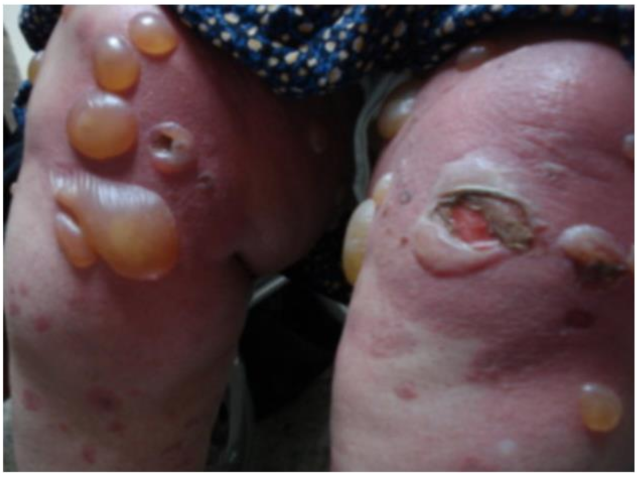
Basal cell carcinoma is a type of skin cancer that begins in the basal cells which produce new skin cells when old ones die off. It often appears as a slightly transparent bump on the skin, though it can also form as red patches or sores. While it could potentially present with similar characteristics to our patient's lesion (especially if ulcerated), BCCs are typically less painful than Chondrodermatitis nodularis helicis.

Lastly, **Keratoacanthoma** manifests as a round, flesh-coloured growth that might resemble a small volcano with a central plug of keratin. They are rapidly growing tumours that often develop over weeks to months which then may resolve spontaneously over many months. However, they are usually solitary and painless, unlike our patient's presentation.

Question 72 of 182

A 79-year-old woman presents to the dermatology clinic with a 2-week history of a blistering rash over her body. She describes the rash to be predominantly over the lower limbs and forearms. Her past medical history includes hypothyroidism and mild hypertension for which she takes levothyroxine and amlodipine daily.

On examination, her observations are within normal limits. Her rash is examined, as shown below:



Given the likely diagnosis, what findings are most likely to be seen on biopsy?

A. Acantholysis
B. IgA deposition
C. IgG and C3 deposition
D. Keratinocyte necrosis
E. Positive gram stain

C. IgG and C3 deposition

This patient has a diagnosis of bullous pemphigoid, given the presence of a blistering rash over the body, predominantly affecting the limbs. The image shows tense blisters that are full of fluid with surrounding erythema. There are discrete, raised erythematous lesions that have a similar appearance to urticaria-like weals. Unlike, pemphigus vulgaris, the oral mucosa is usually spared.

The risk of bullous pemphigoid increases with age and those with a history of autoimmune disease. In order to confirm the diagnosis of bullous pemphigoid, a skin biopsy may be taken which shows **IgG and C3 deposition at the dermo-epidermal junction** on immunofluorescence.

Acantholysis is incorrect. Acantholysis is the loss of cohesion between adjacent keratinocytes due to a loss of intercellular connections. It is seen in pemphigus vulgaris and is absent in bullous pemphigoid, making a skin biopsy a useful diagnostic test to distinguish between the two blistering diseases.

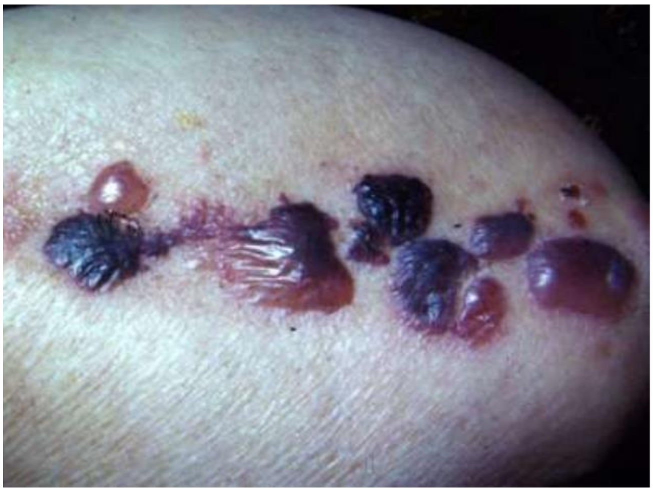
IgA deposition is incorrect. IgA deposition is seen in dermatitis herpetiformis, a condition associated with coeliac disease. It causes a vesicular eruption, most commonly on the shins but would not be responsible for the rash seen in this patient.

Keratinocyte necrosis is incorrect. Keratinocyte necrosis is classically seen in Steven-Johnson syndrome (SJS). SJS is a rare, life-threatening condition that is almost always caused by a drug reaction. It is associated with fever, flaccid blistering causing large amounts of skin detachment, erythema and shock. Although this patient has evidence of a blistering disease, they are otherwise stable, making SJS unlikely.

Positive gram stain is incorrect. Although these blisters can become infected with a bacterial organism, bullous pemphigoid is not an infective process and a positive gram stain is not a diagnostic feature of the disease.

Question 73 ofttt 182

An elderly man develops a blistering skin rash:



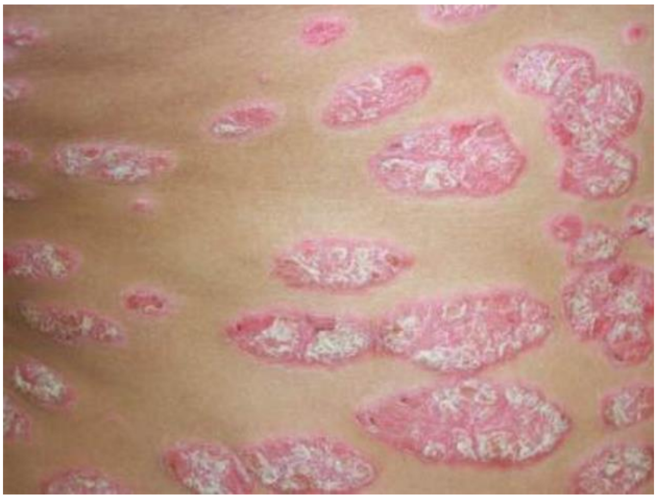
Which one of the following statements regarding the likely diagnosis is correct?

A. It is associated with coeliac disease
B. Eye involvement is the most common complication
C. Topical corticosteroids are the mainstay of treatment
D. It is less common than pemphigus vulgaris
E. The blisters usually heal without scarring

E. The blisters usually heal without scarring

Question 74 of 182

Please look at the image below:



Which one of the following is this patient most likely to go on and develop?

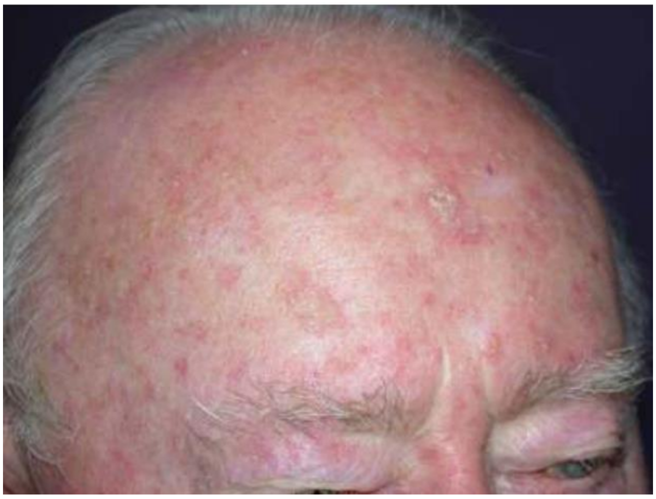
A. Parkinson's disease
B. Hypothyroidism
C. Rheumatoid arthritis
D. Sarcoidosis
E. Metabolic syndrome

E. Metabolic syndrome

Along with psoriatic arthritis, metabolic syndrome is one of the most common and significant complications of psoriasis.

Question 75 of 182

A 67-year-old man who is a retired builder presents following the development of a number of red, scaly lesions on his forehead. These were initially small and flat but are now erythematous and rough to touch.



What is the most likely diagnosis?

A. Pityriasis versicolor
B. Seborrhoeic keratosis
C. Polymorphous light eruption
D. Actinic keratoses
E. Malignant melanoma

D. Actinic keratoses

The most likely diagnosis for this 67-year-old retired builder presenting with red, scaly lesions on his forehead is **actinic keratoses**. Actinic keratoses are premalignant skin lesions caused by chronic exposure to ultraviolet (UV) radiation, typically seen in fair-skinned individuals and those with a history of outdoor work such as builders. The appearance of these lesions as erythematous, rough patches is consistent with actinic keratoses. In some cases, they can progress to squamous cell carcinoma if left untreated.

Pityriasis versicolor is an incorrect answer because it is a superficial fungal infection caused by the yeast *Malassezia*. It typically presents as hypopigmented or hyperpigmented scaly macules and patches on the trunk and upper extremities. The distribution and lack of pigmentary changes make pityriasis versicolor less likely in this case.

Seborrhoeic keratosis is also an incorrect answer. Seborrhoeic keratoses are benign epidermal growths that appear as waxy, brownish-black 'stuck-on' plaques. They commonly occur on the trunk and face but have a different appearance than the erythematous, rough lesions described in this case.

Polymorphous light eruption is not the correct answer either. Polymorphous light eruption is a delayed hypersensitivity reaction to UV radiation that presents as pruritic papules, vesicles, or plaques on sun-exposed skin areas shortly after sun exposure. While the patient's age and occupation might suggest sun exposure as a factor, the chronic nature of these lesions makes polymorphous light eruption less likely.

Finally, **malignant melanoma** would be an incorrect answer because malignant melanoma typically presents as an asymmetrical, irregularly bordered, variably coloured lesion with a diameter greater than 6mm. The red, scaly lesions described in this case do not fit the typical presentation of malignant melanoma.

Question 76 of 182

A 69-year-old man presents to the dermatology clinic for review due to a suspicious skin lesion. He noticed that a particular mole was of concern as it had been irritated and rubbed against his shirt. On his back, he has a 11x13mm brown lesion with an irregular border with a variation in colour. On view under a dermatoscope, there is no telangiectasia, the lesion looks like there are dark pigmented pin-prick spots within the brown lesion.

What feature of this description provides reassurance that the diagnosis is more likely to be benign?

A. The absence of telangiectasia
B. Irregular border of lesion
C. Presence of dark pigmented pin-prick spots
D. The size of the lesion
E. Variation in colour

C. Presence of dark pigmented pin-prick spots

Pseudo-comedones and pale spots help distinguish seborrhoeic lesions from malignant melanomas

The presence of dark pigmented pin-prick spots, sometimes described as pseudo-commodones, are consistent with seborrhoeic keratosis rather than malignant melanoma. Seborrhoeic keratosis can normally be distinguished clinically by a 'stuck-on' appearance, rather than an invasive one. The other features described are not reassuring and could be consistent with a melanoma, but it is also important to establish if there is change in the lesion.

Irregular shape, a size greater than 6mm, and variation in colour are all features which can be considered with skin malignancy. Telangiectasia is a feature of basal cell carcinoma rather than melanoma or seborrhoeic keratosis.

Question 77 of 182

A 32-year-old woman is reviewed in the emergency department. She has developed a very painful rash on her left arm. It is a burning pain which preceded the formation of a rash. She has felt low in energy with this and has noticed that the rash is intensely tender to light touch. Apart from this she feels well in herself. She has a past medical history of appendicitis at the age of 18 which was treated surgically, and depression for which she has been attending cognitive behavioural therapy. On examination, she has an erythematous rash within on the underarm on the left side, roughly 8cm in length. There are small vesicles within this. She is diagnosed with shingles, and told that she can infect people with chicken-pox. She wonders how long she will be infectious for.

What is the most appropriate advise?

A. She will be infectious until she starts aciclovir
B. She will be infectious up to two weeks from onset of rash
C. She will be infectious up to two months from onset of rash
D. She will be infectious until her vesicles have crusted over
E. She will no longer be infectious as vesicles have appeared

D. She will be infectious until her vesicles have crusted over

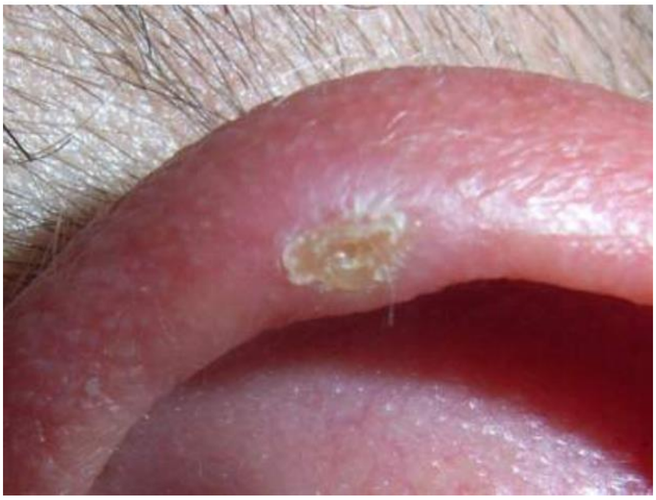
People with shingles should be advised that they are infectious until the vesicles have crusted over, usually 5-7 days following onset

This patient has shingles, acute reactivation of herpes zoster virus (HZV). HZV in the form of shingles is contagious, but exposure could cause chickenpox, rather than shingles. People who have had chickenpox previously would be considered immune, but adults and especially pregnant women who have not had chickenpox are at risk of serious infection.

The patient is infectious until the vesicles have crusted over.

Question 78 of 182

A 75-year-old asks you to have a look at a lesion on his right ear. It has developed slowly over the past few months and is tender to palpation.



What is the most likely diagnosis?

A. Cystic chondromalacia
B. Actinic keratosis
C. Chondrodermatitis nodularis helicis
D. Perichondritis
E. Keratin horn

C. Chondrodermatitis nodularis helicis

The correct answer is **Chondrodermatitis nodularis helicis**. This condition is a benign inflammatory process affecting the ear's cartilage and overlying skin. It typically presents as a tender nodule on the helix or antihelix of the ear, often in elderly patients. The slow development and tenderness to palpation of this patient's lesion are classic signs of chondrodermatitis nodularis helicis.

Cystic chondromalacia, also known as Relapsing Polychondritis, is an incorrect choice because it is a rare multi-system disorder characterized by recurrent episodes of painful inflammation and deterioration of cartilage throughout the body. It would not present as a solitary lesion on the ear; instead, it often affects multiple sites including ears, nose, joints, respiratory tract and eyes.

Actinic keratosis is another incorrect option. While actinic keratosis can indeed present as a rough, scaly patch on sun-exposed skin (such as the ear), it is usually not tender unless it has progressed into squamous cell carcinoma. Furthermore, actinic keratosis lesions generally develop more rapidly than what was described in this case.

The option **Perichondritis** does involve inflammation of the ear cartilage but it's incorrect because perichondritis is typically caused by trauma to the ear or following ear surgery. It presents acutely with severe pain, redness and swelling around the entire ear structure rather than a slowly developing single nodule.

Finally, **Keratin horn** refers to a projection from the skin resulting from overgrowth of keratin. These lesions are typically hard and non-tender which contrasts with this patient's presentation of a tender lesion.

Question 79 of 182

A 37-year-old gentleman is referred to the cardiology clinic with a 7-month history of bilateral foot pain. Initially, the pain only occurred towards the end of his daily commute; improving when he sat at his desk, but over the past 3 weeks his symptoms have progressed. The pain is now provoked by walking short distances and has occasionally occurred at rest. He describes being woken by the pain at night, although he is able to obtain relief by standing on the cool tiles in his bathroom. He eventually decided to see his GP when he developed similar pain in his right wrist 10 days ago.

His past medical history is unremarkable. There is no history of hypertension, dyslipidaemia or diabetes. He is a current smoker with a 20 pack year history. He drinks alcohol in moderation.

On examination, both feet are noted to be smooth and hairless. The right dorsalis pedis is difficult to feel and the medial 3 toes are cold and dusky. Urgent angiography is performed and shows a number of distal stenoses in the right limb affecting the anterior and posterior tibial arteries as well as the distal fibular artery. A number of similar stenoses are seen in the contralateral limb with 'corkscrew' collateral formation.

Blood tests are as follows:

Hb	130 g/l	Na +	139 mmol/l
Platelets	322*10 /l 9	K +	4.1 mmol/l
WBC	9.1*10 /l 9	Urea	5.3 mmol/l
Neuts	6.2*10 /l 9	Creatinine	113 µmol/l
Lymphs	2.1*10 /l 9	CRP	19 mg/l
Eosin	0.1*10 /l9		
MPO		negative	
PR3		negative	

anti-cardiolipin	negative

What is the most likely diagnosis?

A. Peripheral vascular disease
B. Microscopic polyangiitis
C. Takayasu arteritis
D. Thromboangiitis obliterans
E. Polyarteritis nodosa

NOT ANSWERED

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A 54-year-old man comes to the dermatology clinic for review. He has a hyperpigmented rash affecting the back of his neck, his axillae and his groin that has become more prominent over the past few weeks. There has been increased shortness of breath and decreased exercise tolerance over the past 3 months. He smokes 20 cigarettes per day and is treated for COPD with a high dose salmeterol / fluticasone combination inhaler. Examination reveals a blood pressure of 132/82 mmHg, and a pulse of 65 beats per minute. You confirm the rash. There is quiet wheeze on auscultation of the chest. Abdomen is soft and he complains of mild tenderness in epigastrium. His body mass index is 22 kg/m².

Investigations

Hb	90 g/l	Na +	140 mmol/l
Platelets	191 * 10 /l 9	K +	4.3 mmol/l
WBC	7.0 * 10 /l 9	Urea	7.2 mmol/l
Neuts	3.2 * 10 /l 9	Creatinine	110 µmol/l
Lymphs	1.4 * 10 /l 9	CRP	32 mg/l
ESR	72 mm/hr		

Which of the following is the most useful next investigation?

A. Bronchoscopy
B. Colonoscopy
C. Skin biopsy
D. Ultrasound abdomen
E. Upper gastrointestinal endoscopy

E. Upper gastrointestinal endoscopy

The most common malignancy associated with acanthosis nigricans is gastrointestinal adenocarcinoma

The character of the rash is most suggestive of a diagnosis of acanthosis nigricans. Given the history of smoking and the anaemia, the possibility of underlying gastric cancer is raised, for this reason, an endoscopy +/- biopsy of any lesion is the most useful next step.

Bronchoscopy isn't indicated, nor is a colonoscopy, because lung cancer and colon cancer are not as strongly associated with acanthosis. Ultrasound of the abdomen may reveal an epigastric mass, but it is a relatively insensitive way of detecting gastric cancer.

Skin biopsy reveals hyperkeratosis and papillomatosis, relatively minor hyperpigmentation is also seen.

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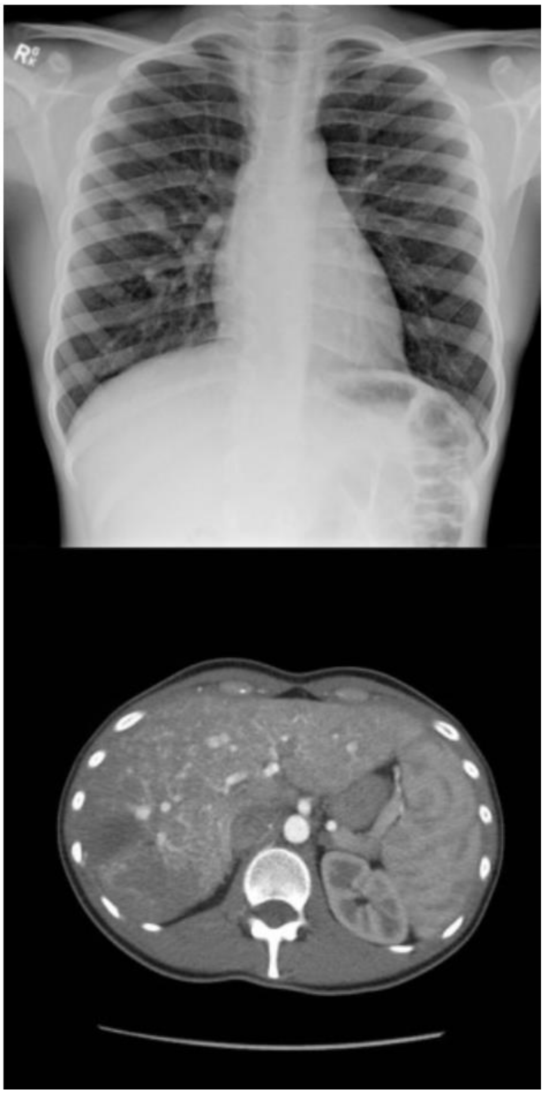
A 16-year-old male is investigated for recurrent epistaxis, haemoptysis and iron-deficiency anaemia. Blood tests show the following:

Hb	11.1 g/dl
Platelets	341 * 10 /l9
WBC	4.3 * 10 /l9
Bilirubin	33 µmol/l
ALP	131 u/l
ALT	54 u/l
γGT	135 u/l
Albumin	40 g/l

A chest x-ray and CT abdomen are requested:



What is the most likely diagnosis?

A. Acute lymphoblastic leukaemia
B. Metastatic cancer secondary to familial adenomatous polyposis
C. Hereditary haemorrhagic telangiectasia
D. Metastatic pleuropulmonary blastoma

E. Peutz-Jeghers syndrome

C. Hereditary haemorrhagic telangiectasia

The correct answer is **Hereditary haemorrhagic telangiectasia**. This is a rare autosomal dominant disorder characterised by the presence of multiple arteriovenous malformations (AVMs)

that lack intervening capillaries and result in direct connections between arteries and veins.

The chest x-ray shows multiple pulmonary nodules representing arteriovenous malformations, the largest in the right mid-zone. The CT scan shows multiple hepatic arteriovenous malformations.

The clinical presentation of recurrent epistaxis, haemoptysis, and iron-deficiency anaemia in this 16-year-old male is suggestive of Hereditary haemorrhagic telangiectasia (HHT), also known as Osler-Weber-Rendu syndrome. HHT can affect various organs, including the lungs, liver, brain, and gastrointestinal tract, causing bleeding.

The other options are less likely based on the given information:

Acute lymphoblastic leukaemia typically presents with symptoms related to bone marrow suppression, such as fatigue, pallor, fever or infection due to neutropenia. This patient's blood test results do not show any significant abnormalities in white blood cell count or platelet count that would be expected in acute leukaemia.

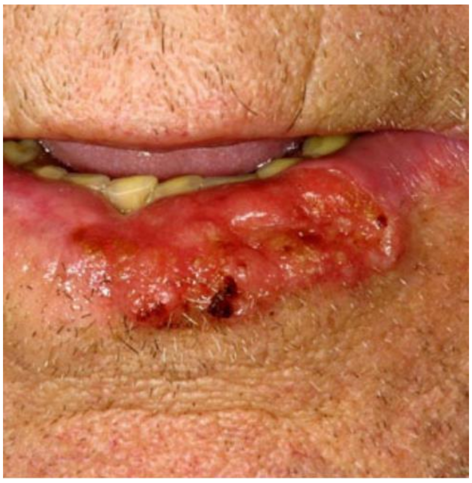
Metastatic cancer secondary to familial adenomatous polyposis is an unlikely diagnosis as this condition is primarily associated with colorectal cancers developing from numerous adenomatous polyps in the colon. The patient's presenting symptoms of recurrent epistaxis and haemoptysis do not fit this diagnosis.

Metastatic pleuropulmonary blastoma, a rare malignant mesenchymal tumour of childhood affecting the lung parenchyma and pleura, usually presents with respiratory distress or pneumonia-like symptoms rather than recurrent epistaxis and haemoptysis.

Finally, **Peutz-Jeghers syndrome** is characterised by hamartomatous gastrointestinal polyps and mucocutaneous pigmentation. While this syndrome can cause anaemia due to gastrointestinal bleeding, it does not typically present with recurrent epistaxis or haemoptysis as seen in this patient.

Question 82 of 182

A 75-year-old man presents with a 'sore' on his lip. This has been present for around four months and has been getting slowly worse. His past medical history includes ischaemic heart disease and chronic obstructive pulmonary disease. He still smokes around 20 cigarettes per day.



What is the most likely diagnosis?

A. Actinic keratosis
B. Leukoplakia
C. Squamous cell carcinoma
D. Mucocoele
E. Bowen's disease

C. Squamous cell carcinoma

The most likely diagnosis in this case is **Squamous cell carcinoma**. The patient's age, history of smoking, and the appearance of the lesion with its chronicity and progressive worsening are all suggestive of squamous cell carcinoma (SCC). SCC is a malignant tumour arising from keratinocytes in the epidermis. It commonly affects sun-exposed areas like the face and lips. Smoking is a significant risk factor for SCC development.

Actinic keratosis is an incorrect option because it typically presents as a rough, scaly patch on sun-exposed skin. It is a precursor to SCC but does not have the same appearance as the lesion described in this case.

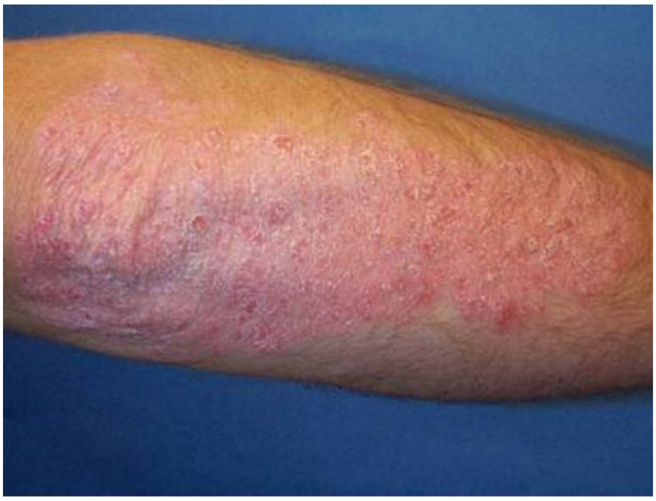
Leukoplakia is also incorrect as it usually presents as white patches or plaques on the oral mucosa, which cannot be scraped off. While leukoplakia can be associated with malignancy, it does not have the same clinical appearance as the lip sore described here.

Mucocoele is another incorrect choice because it presents as a painless, fluid-filled swelling that often occurs on the lower lip due to trauma or obstruction of salivary glands. The chronicity and worsening nature of the lesion in this case do not fit with a mucocoele.

Finally, **Bowen's disease** is an incorrect option because it typically appears as an erythematous, scaly patch with well-defined borders on sun-exposed skin. Although Bowen's disease represents squamous cell carcinoma in situ and shares some risk factors with invasive SCC, its clinical presentation differs from that described in this scenario.

Question 83 of 182

A 34-year-old man presents with a scaly rash on his elbows and knees that has been getting progressively worse for the past 3 months. He has tried using some of his wife's Diprobase cream which has helped the itching somewhat.



Following NICE guidance, what is the most appropriate next step in management?

A. Topical potent corticosteroid + vitamin D analogue
B. Topical potent corticosteroid
C. Refer for ultraviolet light therapy
D. Topical vitamin D analogue
E. Topical coal tar

A. Topical potent corticosteroid + vitamin D analogue

Topical potent corticosteroid + vitamin D analogue is first-line for chronic plaque psoriasis

The correct answer is **Topical potent corticosteroid + vitamin D analogue**. According to NICE guidelines, the first-line treatment for psoriasis in adults should be a combination of a topical corticosteroid and a vitamin D analogue. This combination has been shown to be more effective than using either treatment alone. The patient's presentation of a scaly rash on his elbows and knees is consistent with plaque psoriasis, and the use of a potent corticosteroid combined with a vitamin D analogue would provide both anti-inflammatory and keratolytic effects, helping to reduce symptoms and improve the appearance of the skin.

The option **Topical potent corticosteroid** alone may provide some relief from inflammation but would not address the abnormal keratinocyte proliferation seen in psoriasis. Additionally, using a corticosteroid alone increases the risk of skin atrophy and other side effects associated with longterm use.

Refer for ultraviolet light therapy is not the most appropriate next step as per NICE guidelines. Ultraviolet light therapy can be considered if topical treatments have failed or are contraindicated; however, this patient has not yet tried any targeted topical treatments for his psoriasis.

Using only a **Topical vitamin D analogue**, while it may help regulate keratinocyte proliferation, would not provide sufficient anti-inflammatory effects needed to manage this patient's symptoms effectively. As mentioned earlier, combining it with a potent corticosteroid provides better results.

Finally, **Topical coal tar** is an older treatment option for psoriasis that works by reducing inflammation and slowing down skin cell growth. However, it is less commonly used today due to its unpleasant smell, staining properties, and potential carcinogenic risks. It is also less effective than newer treatments such as topical corticosteroids and vitamin D analogues.

Question 84 of 182

A 64-year-old man attends with a severe facial skin rash. He describes recurrent redness on his face. This has been on and off for several years but has recently worsened and the rash is not going away. It is associated with burning and stinging sensations and occasional swelling around the eyes.

On examination, he has an erythematous rash over his cheeks, nasal bridge, and forehead. Within the erythema are numerous large pustules and some pitted scarring with interspersed papules and single plaque. He also has an erythematous rash over his neck and shoulders that is warm to the touch and has the appearance of sunburn.

Given the likely diagnosis, what would be the most appropriate treatment option for this patient?

A. Laser therapy
B. Topical brimonidine gel
C. Topical ivermectin
D. Topical ivermectin and oral doxycycline combination therapy
E. Topical ivermectin and oral doxycycline combination therapy and dermatology referral

D. Topical ivermectin and oral doxycycline combination therapy

Rosacea: a combination of topical ivermectin + oral doxycycline is first-line for patients with severe papules and/or pustules

The most likely diagnosis here is rosacea. Recurrent erythematous facial rash that comes and goes with the distribution described in the stem is typical of rosacea. It is commonly associated with flushing, papules, and pustules, in some cases, rhinophyma can develop. Another clue is that this patient has sunburn - the skin rash in rosacea is commonly exacerbated by sunlight and sunburn indicates significant sun exposure.

Topical ivermectin and oral doxycycline combination therapy is the correct answer. We have a persistent rash with a significant burden of papules and pustules, this would be deemed 'moderate to severe' so a combination of topical ivermectin and oral doxycycline would be advisable.

Topical ivermectin is incorrect. This would be a reasonable first-line option for patients with mild to moderate rash. 'Few' papules/pustules would be classed as 'mild', 'several' would be classed as 'moderate', and 'many' would be classed as 'severe' rosacea according to the National Rosacea Society. The presence of plaque also indicates 'severe' rosacea.

Laser therapy is incorrect. This would be guided by secondary care specialists and does not form part of the first-line management for rosacea, it is predominantly used for patients with extensive telangiectasia which is not a main issue for this patient.

Topical ivermectin and oral doxycycline combination therapy and dermatology referral is incorrect. It would be reasonable to trial optimal management in primary care before onward referral to dermatology specialists.

Topical brimonidine gel is incorrect. This topical alpha-adrenergic agonist is used to manage flushing associated with rosacea. It would not be of benefit in this case as it would not have efficacy in managing the pustules and papules. This patient has pustule and papule-predominant rosacea which is better managed with ivermectin, doxycycline, or alternative options such as metronidazole and topical azelaic acid.

Question 85 of 182

A 50-year-old woman attends your clinic with an itchy rash on her forearms. It constitutes several small violaceous polygonal papules, mainly clustered around the wrist flexors. Her background is notable for left ventricular failure, for which she was recently commenced on 5mg bisoprolol OD.

Bloods are unremarkable.

Which of the following is true in this condition?

A. Scalp lesions are common
B. Oral lesions have a better prognosis
C. Nail involvement is not seen
D. It is a type IV hypersensitivity reaction
E. Exhibits the Koebner phenomenon

E. Exhibits the Koebner phenomenon

Koebner phenomenon - Warts And ALL (warts, autoimmune, AIDS, lichen planus & lichen sclerosis)

This is the typical description of lichen planus. It is possible that bisoprolol has precipitated it - as can thiazide diuretics and anti-malarial drugs. Cutaneous lesions can take up to a year to fully heal and oral lesions can take much longer, with many requiring oral steroids and topical calcineurin inhibitors.

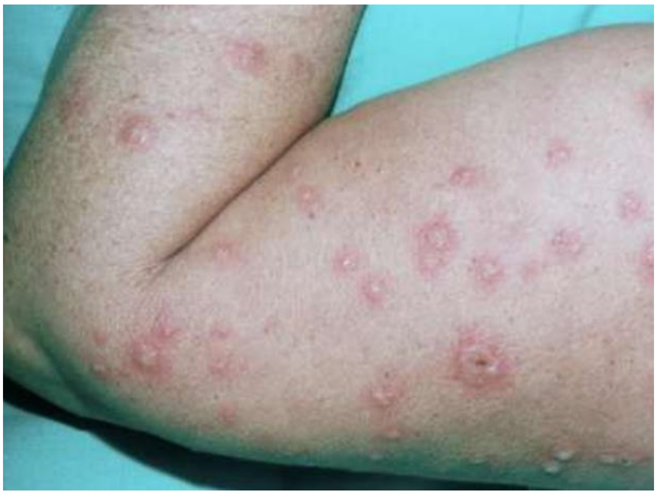
Lichen planus exhibits the Koebner phenomenon, in which minor trauma can cause new lesions. Other conditions that exhibit this phenomenon include viral warts, vitiligo, molluscum contagiosum and lichen sclerosis.

Nail involvement can be seen in lichen planus and is characterised by longitudinal grooving, which can be helpful at differentiating the condition from psoriasis.

Allergic contact dermatitis is a type IV hypersensitivity reaction and would have a more eczematous appearance.

Question 86 of 182

A middle aged man develops a non-pruritic rash after starting allopurinol therapy for gout. The rash develop within 24 hours and started on the back of his hands.



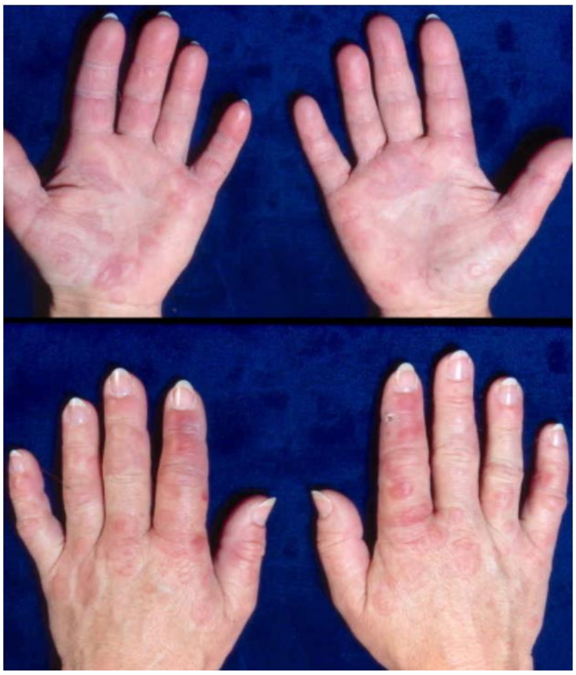
What is the most likely diagnosis?

A. A llopurinol-associated dermatitis
B. Plaque-type tophi
C. Erythema multiforme
D. Erythema marginatum
E. Eosinophilic folliculitis

C. Erythema multiforme

Question 87 of 182

A 40-year-old woman visits her general practitioner with a 2-day history of a new rash that has developed over her hands and forearms. She denies pain or itch and has no history of previous dermatological conditions. Over the last week, she reports feeling generally lethargic with lowgrade fevers and a dry cough. There is no past medical history of note and she takes no allergies. Her rash is examined, as shown below. There is no nail or mucosal involvement.



What is the most likely diagnosis?

A. Erythema marginatum
B. Erythema multiforme
C. Irritant contact dermatitis
D. Lichen planus
E. Plaque psoriasis

B. Erythema multiforme

This patient has multiple erythematous, round lesions on her hands. On closer inspection, some of these lesions have a characteristic appearance of a target lesion with an outermost erythematous ring and paler centre, in keeping with a diagnosis of erythema multiforme. Erythema multiforme is an acute, self-limiting skin eruption most commonly secondary to an infective trigger (e.g. herpes simplex virus or *Mycoplasma pneumoniae*). The history of low-grade fevers and a dry cough further support an infective component of this patient's rash. The rash appears abruptly and affects the upper limbs most often before spreading down towards the trunk. A history of lethargy and lowgrade fevers suggests a recent infection that may have triggered the onset of the rash.

Erythema marginatum is a manifestation of acute rheumatic fever. It is most commonly seen in children and is rare in adults. It presents as annular erythematous macules or papules that spread outwards with central clearance. The lesions are not itchy or painful and may be asymptomatic.

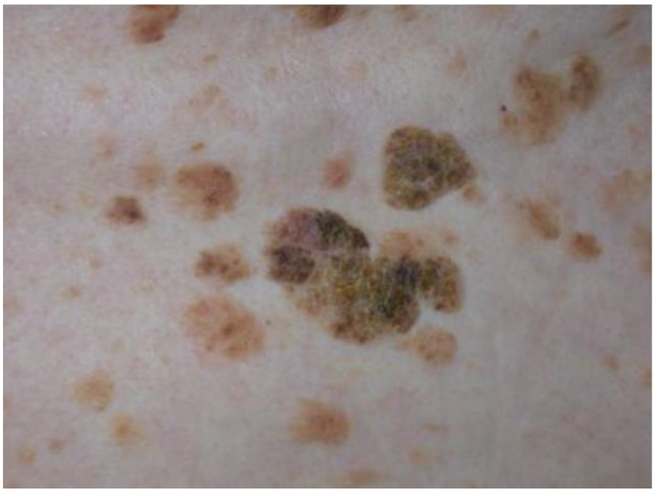
Irritant contact dermatitis is an inflammatory skin condition secondary to contact with irritants to the skin (e.g. detergents). The rash manifests as an erythematous and dry rash that may feel painful or itchy. There is no history of exposure to irritants in this question and the rash does not appear to be dry or flaky which makes irritant contact dermatitis less likely.

Lichen planus is a chronic inflammatory skin condition characterised by purplish papules and plaques commonly found on the front of the wrists. They are often shiny and may have fine white lines covering the surface (Wickham striae). This description does not match the rash in the photograph, making it the incorrect answer.

Plaque psoriasis is a chronic inflammatory skin condition that gives rise to well-defined red plaques with an overlying silver scale. Common features include itch and painful fissures. The rash in the image does not show the presence of erythematous plaques. Furthermore, this patient's rash has had an acute onset which is atypical of psoriasis that has a much slower onset.

Question 88 of 182

A 68-year-old man asks you to 'check his moles' during an admission. He was admitted three days ago with a non-ST elevation myocardial infarction. You look at the following area of skin on his back:



What is the most likely diagnosis?

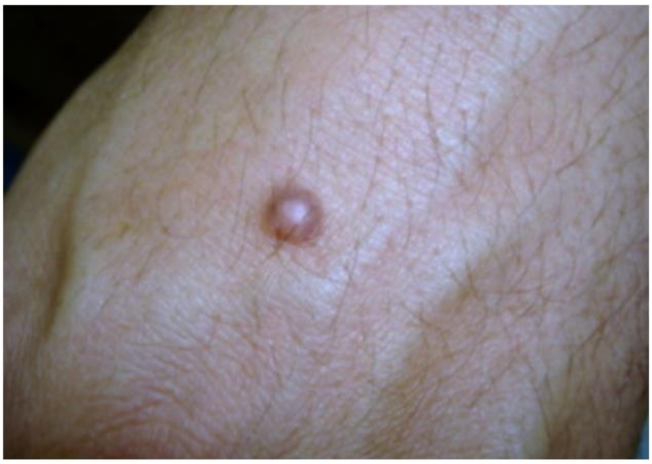
A. Actinic keratosis
B. Benign mole
C. Bowen's disease
D. Malignant melanoma
E. Seborrhoeic keratosis

E. Seborrhoeic keratosis

This lesion has the typical warty, 'stuck-on' appearance of a seborrhoeic keratosis.

Question 89 of 182

A 34-year-old man presents with a small lump on his hand. He says that it has been present for several months. He cannot remember a clear onset, although does remember a thorn-related injury on that hand whilst gardening. On examination, the lump appears as shown below:



It is approximately 8mm in diameter and not tender.

Given the most likely diagnosis, which of the following should happen next?

A. Aspiration
B. Biopsy
C. Cryotherapy
D. Excision

E. Observation

E. Observation

This photo is indicative of a dermatofibroma, commonly found in young people - more so on the legs, rather than on the hands as seen here. It is a solitary, firm papule, usually less than 1cm in diameter. It has a regular border, shape, colour and is symmetrical - there are no concerning features regarding its appearance. In this case, as the lesion is not tender or irritating, it can simply be observed for now and the patient reassured.

Aspiration is inappropriate - dermatofibromas are solid lesions, not fluid-filled.

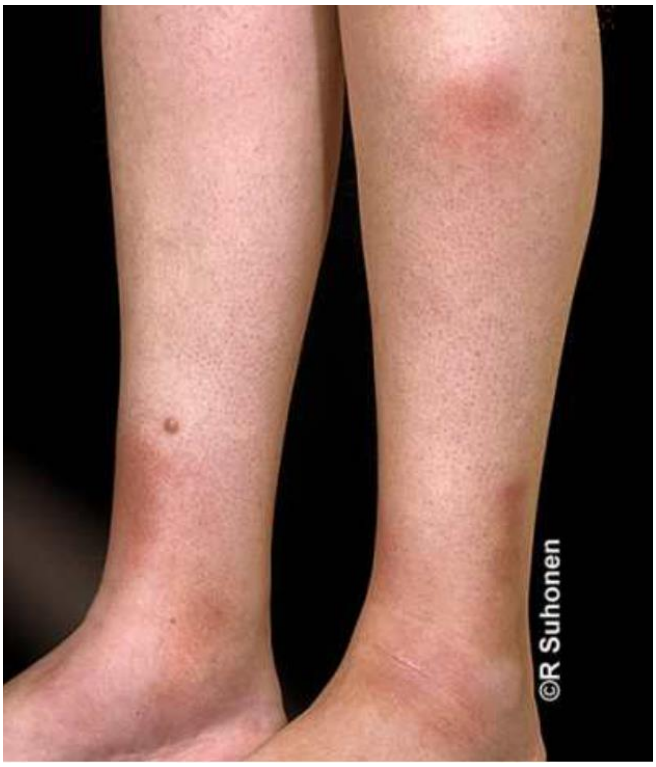
A biopsy may be undertaken if any atypical features trigger concern for a more serious diagnosis. However, in this instance, we can see the lesion looks entirely regular and typical.

Cryotherapy is a suitable option for treatment if the lesion is bothersome, according to the British Association of Dermatologists. In this scenario, there are no troublesome features mentioned and so the patient should be reassured at this stage.

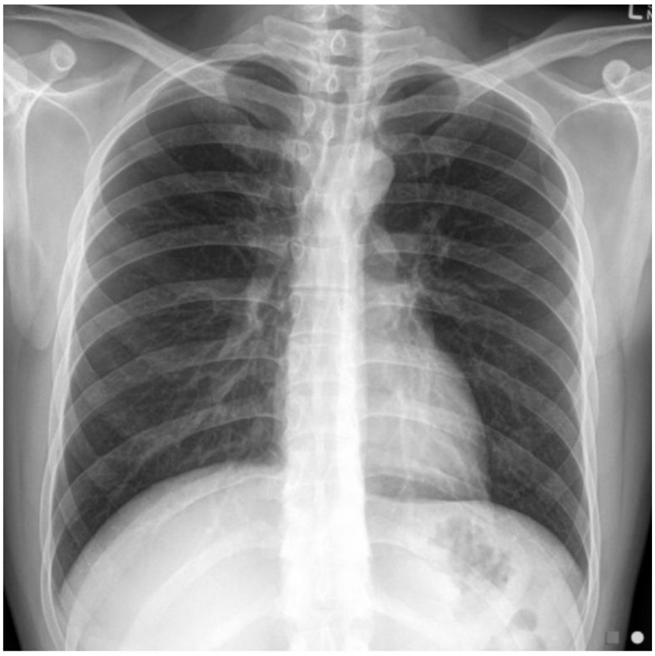
Excision is another suitable option for treatment if bothersome. However, in this scenario, the lesion causes no issues and so should simply be observed for now.

Question 90 of 182

A 30-year-old woman presents with a painful 'rash' on her shins



These have been present for the past 2 weeks. There is no past medical history of note although she had a course of penicillin V for a bout of tonsillitis around a month ago. She also takes Cerazette (a progestogen-only pill) for contraception. A chest x-ray is requested:



What is the most likely cause of the skin lesions?

- A. Lymphoma
- B. Sarcoidosis
- C. Progestogen-only pill
- D. Penicillin
- E. Tuberculosis

D. Penicillin

The likely diagnosis here is erythema nodosum (EN). The chest x-ray is completely normal making penicillin the likely culprit in precipitating skin lesions. The combined oral contraceptive pill is linked to EN rather than the progestogen-only pill.

Question 91 of 182

A 32-year-old man with autism spectrum disorder presents to the general medical clinic. He was referred by his GP for urgent review whilst awaiting a dermatology appointment. The patient has been experiencing a severe eczema flare which has not been controlled despite the use of emollients twice each day, strong topical corticosteroids for five days and non-sedating antihistamines. He has no other medical problems. He has become very concerned about the spreading rash and feels a high level of anxiety with it. On examination, there is spreading erythema from flexural folds on arms and legs.

What is the most appropriate treatment option whilst awaiting dermatology opinion?

- A. Admit for IV hydrocortisone 200mg QDS
- B. Start prednisolone 30mg OD for one week
- C. Start prednisolone 30mg OD long-term with bone protection
- D. Advise for bandaging
- E. Start urea-containing emollient

B. Start prednisolone 30mg OD for one week

Prednisolone treatment for eczema flares needs several weeks of treatment and then tapering off to prevent rebound

The most appropriate action for this patient is for a trial of oral prednisolone. NICE advises one week of 30mg OD whilst awaiting dermatology review, which can be extended as necessary, but this should be done under close review. This would be especially done for people who are suffering psychological distress from severe eczema. Admission is not necessary but would be if the patient was unstable or had eczema herpeticum. Bandaging would be a better approach for weeping or lichenified eczema. and urea-containing emollients are useful when greater hydration is needed for pruritic skin in the absence of severe inflammation.

Question 92 of 182

A 30-year-old lady was admitted with a painful ulcer on her right lower limb. At first, the patient described developing a painful erythematous area which then rapidly broke down to form a painful ulcer. The edges of the ulcer had a purple discolouration. She did recall recently knocking this area whilst riding her bike to work. She had recently been considering seeing her GP as she had been opening her bowels around eight times a day with some mucus and she had noticed some mild abdominal pain. She attributed this to irritable bowel syndrome which she is believed to have had since the age of 20 years.

Her past medical history included migraines and a right deep vein thrombosis after a flight to America. Her father had a family history of diabetes. She had a biopsy of the ulcer which showed heavy neutrophil infiltration.

What is the most likely diagnosis?

A. Pyoderma gangrenosum
B. Erythema nodosum
C. Necrobiosis lipoidica
D. Leishmaniasis
E. Venous ulcer

A. Pyoderma gangrenosum

This patient has Pyoderma gangrenosum with underlying undiagnosed inflammatory bowel disease.

Pyoderma gangrenosum is a rare, non-infectious, inflammatory disorder. It is an uncommon cause of very painful skin ulceration. It may affect any part of the skin, but the lower legs are the most common site. In 50% there is no underlying cause but in 10-15% it is associated with ulcerative colitis or Crohn's disease. Other associations include rheumatoid arthritis, myeloproliferative disorders, malignancy, and Wegener's granulomatosis.

Pyoderma gangrenosum is classified as a neutrophilic dermatosis. Neutrophilic dermatoses are skin conditions characterised by dense infiltration of neutrophils in the affected tissue and this is often seen on biopsy.

Pyoderma gangrenosum usually starts quite suddenly, often at the site of a minor injury as in this patient's case and this is known as pathergy. It may start as a small pustule, red bump or bloodblister. The skin then breaks down resulting in an ulcer which is often painful. The edge of the ulcer is often described as purple, violaceous and undermined.

Diagnosis of Pyoderma gangrenosum can be difficult. Diagnosis is often made by its characteristic appearance, associations with other diseases, the presence of pathergy, histology results and when other diseases have been ruled out. Histology is not specific and can vary depending on time and site of the specimen. It is helpful in ruling out other causes of an ulcer. The ulcer should be swabbed and cultured for micro-organisms and fungi.

Treatment is mainly steroid (0.5-2mg/kg) therapy which is slowly tapered over 1-2 months. Immunosuppressants are useful as a steroid sparing agent and maintenance therapy. These may include ciclosporin, methotrexate, mycophenolate mofetil and cyclophosphamide. Surgery should be postponed until the disease process is controlled on immunosuppression to risk worsening of the disease (pathergy).

Necrobiosis lipoidica is a skin disorder which can affect the shins. This most commonly occurs in patients with insulin dependent diabetics, although it may occur in non-diabetic patients. These lesions are characteristically yellowish brown patches which appear slowly over a few months. The patch is often pale, shiny and has telangiectasia present. They are often painless. Ulceration can occur if the patch has a minor injury to the area.

Erythema nodosum is often described as red lumps that form on the shins, but can also occur on the forearms and thighs. It is an inflammatory disorder affecting subcutaneous fat (panniculitis). Patients commonly develop these in association with bacterial infections (commonly mycoplasma pneumonia), streptococcal throat infections, sarcoidosis, tuberculosis, drugs such as the oral contractive pill, NSAIDS and salicylates, pregnancy and inflammatory bowel disease.

The lesions may appear in a range of sizes and number. The nodules are often painful, warm and red on first appearance. Later they may change to a purple colour and appear more like a bruise. This bruise like colour often evolves over the next 1-2 weeks and then subsides by 3rd to 6th week. The patient may also complain of a fever, arthralgia and malaise.

Question 93 of 182

A 33-year-old lady in her third trimester of her first pregnancy comes to see you regarding a rash she has developed. It started over her abdomen but has now spread over her upper thighs and buttocks. You examine her and see the rash is erythematous, and there are small papules evident which have coalesced into patches in some areas. Around her abdomen, the rash is most marked in areas related to her stretchmarks and around the umbilicus seems to be spared; there are no blisters evident.

What is the most likely diagnosis?

A. Polymorphic eruption of pregnancy
B. Atopic eruption of pregnancy
C. Pemphigoid gestationis
D. Psoriasis
E. Obstetric cholestasis

A. Polymorphic eruption of pregnancy

The answer here is the polymorphic eruption of pregnancy. This is a condition usually occurs in the third trimester, often only in a first pregnancy, and typically there are pruritic urticarial papules that

coalesce into plaques. These often start on the abdomen, particularly related to striae but may also extend elsewhere. Around the umbilicus is typically spared.

Atopic eruption of pregnancy is most common in patients that have eczema. It is most common in the first trimester and it is typically eczematous lesions that break out, unlike in this case. Pemphigoid gestationis is not the answer as it is a very rare condition that is characterised by initial erythematous urticarial papules that progress to form bullae especially over the abdomen. Obstetric cholestasis is not associated with a rash so this is unlikely. The history and lesions are not typical of psoriasis so this is not the diagnosis here.

Question 94 of 182

A 65-year-old Caucasian lady presents with a new skin lesion to the forearm which has been present for 4 weeks. She reports she first noticed it after sustaining a minor abrasion to the area, and it has quickly increased in size since. On examination, there is a 10mm raised, symmetrical nodular lesion, which has a large keratinised central core. The surrounding skin appears normal, with no other similar lesions.

What is the most likely diagnosis?

A. Basal cell carcinoma
B. Actinic keratosis
C. Pyogenic granuloma
D. Molluscum contagiosum
E. Keratoacanthoma

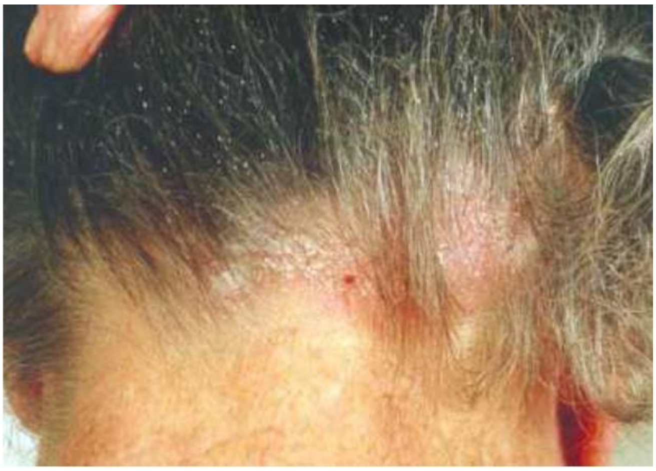
E. Keratoacanthoma

Keratoacanthoma are fast-growing skin lesions which may appear in areas of sun damaged skin. They may also develop after a minor skin injury. They typically start as a small, round fleshy nodule before developing a keratinised central core. Though they may spontaneously regress, they are difficult to distinguish clinically from a squamous cell carcinoma, and therefore should be referred for surgical removal.

Basal cell carcinomas tend to be slow growing lesions. Actinic keratoses tend to be scaly plaques. Pyogenic granuloma is another fast-growing lesion which occur at the site of minor skin injury, but are vascular, bleeding easily. Molluscum contagiosum are round lesions with a small central punctum caused by a pox virus that tend to appear in crops, mainly in children.

Question 95 of 182

A 47-year-old woman complains of an itchy neck and scalp:



This skin condition is though to occur as a result of a reaction to:

A. Trichophyton rubrum
B. Trichophyton schoenleinii
C. Microsporum audouinii
D. Candida albicans
E. Malassezia furfur

E. Malassezia furfur

Seborrhoeic dermatitis - an inflammatory reaction to *Malassezia furfur*

The correct answer to this question is ***Malassezia furfur***. This yeast-like fungus is a part of the normal skin flora but can overgrow in certain conditions, leading to various skin diseases including Pityriasis versicolor, seborrheic dermatitis and dandruff. The image provided shows signs of seborrheic dermatitis on the scalp and neck which is typically associated with an overgrowth of *Malassezia furfur*. It is thought that this organism causes irritation and inflammation in susceptible individuals, leading to the characteristic flaky, itchy skin.

The other options listed are all organisms that can cause skin infections, but they are not typically associated with the symptoms and presentation described in the question.

Trichophyton rubrum is a dermatophyte fungus that causes athlete's foot (tinea pedis), jock itch (tinea cruris) and ringworm (tinea corporis). These conditions present with a well-defined red, scaly rash often accompanied by itching. However, these rashes usually appear on the feet, groin or body rather than the scalp or neck.

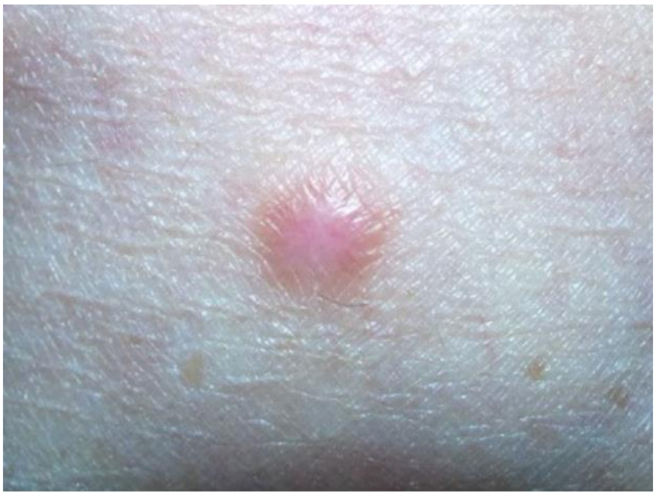
Trichophyton schoenleinii is another dermatophyte fungus, which primarily causes tinea capitis (scalp ringworm). While this condition does affect the scalp, it presents differently from seborrheic dermatitis. Tinea capitis typically causes patchy hair loss and a scaly scalp rather than generalised itching and flaking.

Microsporum audouinii, also a cause of tinea capitis, would similarly result in patchy hair loss rather than widespread itching and flaking. It's important to note that while both *T. schoenleinii* and *M. audouinii* can cause scalp infections, they are more commonly seen in children rather than adults.

Finally, ***Candida albicans***, a type of yeast fungus, can cause various infections including oral thrush and vaginal yeast infections. While it can affect the skin causing candidal intertrigo or diaper rash for example; it's less likely to be responsible for an itchy neck and scalp as described in this scenario.

Question 96 of 182

A 33-year-old woman asks you to look at a 'spot' on her arm that has been present for several months. It has not changed recently and is completely asymptomatic.



What is the most likely diagnosis?

A. Leiomyoma
B. Basal cell carcinoma
C. Amelanotic malignant melanoma
D. Dermatofibroma
E. Nodular prurigo

D. Dermatofibroma

The most likely diagnosis for the lesion presented in the image is a **Dermatofibroma**. Dermatofibromas are common benign skin nodules that often develop after minor skin injuries. They are typically asymptomatic, firm to touch, and can be brownish or purplish in colour. The patient's history of having a 'spot' on her arm for several months without any changes or symptoms is consistent with this diagnosis.

The first option, a **Leiomyoma**, is less likely as these are smooth muscle tumours that usually present as painful lesions when exposed to cold or pressure, which does not match the patient's presentation of an asymptomatic lesion.

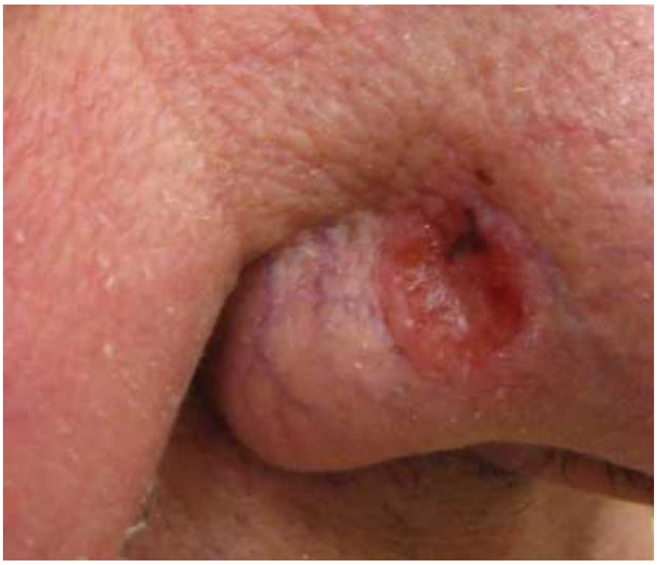
The second option, a **Basal cell carcinoma**, is also unlikely. Although it is one of the most common forms of skin cancer, it usually presents as a pearly nodule with telangiectasia (visible blood vessels) on sun-exposed areas. It may ulcerate and bleed but rarely metastasises. This does not fit with the clinical picture described here.

An **Amelanotic malignant melanoma**, the third option, is a type of skin cancer that lacks pigment. While it can appear similar to the lesion in question, amelanotic melanomas tend to change rapidly over weeks to months and may have irregular borders and surface characteristics. Given this woman's stable lesion over several months without any symptoms, an amelanotic melanoma seems unlikely.

Finally, **Nodular prurigo** could present as multiple intensely itchy nodules predominantly found on extensor surfaces such as arms and legs. However, given that this woman has no mention of itchiness or multiple lesions, nodular prurigo would be an unlikely diagnosis for her condition.

Question 97 of 182

A 62-year-old man presents with a lesion on the right side of his nose. He is unsure how long it has been there.



What is the most likely diagnosis?

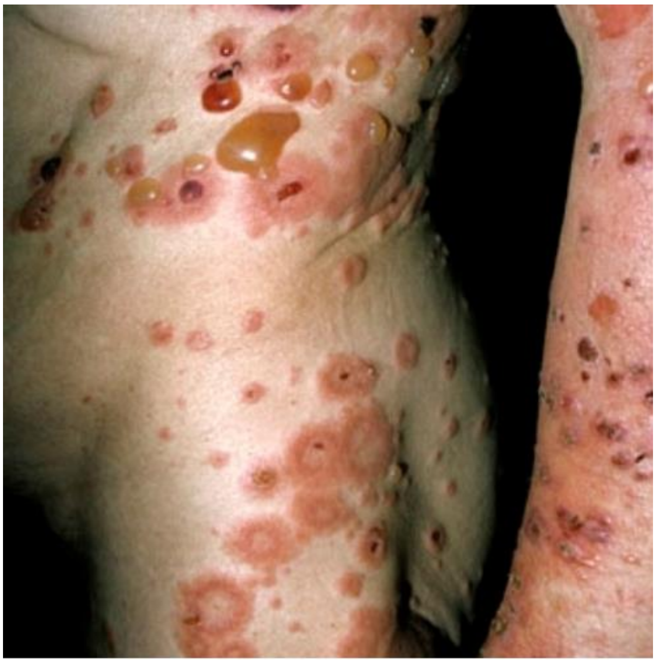
A. Desmoplastic trichoepithelioma
B. Squamous cell carcinoma
C. Impetigo
D. Basal cell carcinoma
E. Actinic keratosis

D. Basal cell carcinoma

The rolled, pearly edges with telangiectasia surrounding a central crater make basal cell carcinoma the most likely diagnosis.

Question 98 of 182

An elderly man develops a generalised pruritic rash:



Which one of the following is the mainstay of treatment?

A. Gluten free diet
B. Phototherapy
C. Oral corticosteroids
D. Long-term oral antibiotics
E. Potent topical corticosteroids

C. Oral corticosteroids

Question 99 of 182

A 59-year-old man with extensive small bowel stricturing Crohn's disease underwent a large small bowel resection. It was complicated by an anastomotic breakdown which required a further resection and jejunostomy formation. He was referred to a local intestinal failure unit and, after a period of assessment, was discharged home on parenteral nutrition. He gained weight and made good progress, although an intermittently high-output stoma persisted. When reviewed in clinic after a year on home parenteral nutrition, he complained of hair loss, a skin rash, and impairment of taste. Examination revealed a superficial scaling erythematous patchy rash that was most prominent in intertriginous areas and periorally.

Which is the most likely nutrient deficiency?

A. Niacin
B. Selenium
C. Vitamin A
D. Vitamin E
E. Zinc

E. Zinc

Zinc deficiency causes a characteristic skin rash (mainly at the intertriginous and perioral areas), alopecia, taste impairment, glucose intolerance, and diarrhoea

Zinc deficiency causes a characteristic skin rash (mainly affecting the intertriginous and perioral areas), alopecia, taste impairment, glucose intolerance, and diarrhoea. In addition to the above symptoms, deficiency also causes acne, visual problems, impaired ability to smell, subtle cognitive and memory changes, and increased susceptibility to respiratory infections. Secondary skin infections may develop with extensive skin rashes. Nail changes, including white spots, bands, lines, or leuconychia, may also be evident. Diarrhoea, stomal and fistula losses increase the loss of zinc.

In contrast to the other options, vitamin A deficiency causes night blindness and eventually xerophthalmia. It can also lead to impaired immune function, cancer, and birth defects. Vitamin E deficiency can lead to problems such as spinocerebellar ataxia and myopathies, and anaemia, due to oxidative damage to red blood cells. Selenium deficiency has been seen in patients with malabsorption and in those on total parenteral nutrition. The effects include myocardial necrosis leading to congestive cardiomyopathy. There is increased susceptibility to infection and can also cause disruption of thyroid metabolism (hypothyroidism). Niacin deficiency causes pellagra, dermatitis, alopecia, oedema, and glossitis. Psychological changes include emotional problems, aggression, insomnia, confusion, and dementia. Neurological signs include weakness, ataxia, paralysis, and peripheral neuritis. Diarrhoea and a dilated cardiomyopathy may also occur.

Question 100 of 182

A 82-year-old man presents to dermatology clinic with changes on his scalp. He is a bald man and has noticed that he has had thickened and abnormal skin. He has a long history of sun exposure from having been living in Spain for five years previously but has now returned to the UK permanently. He has also a history of smoking, but quit 15 years ago, and has also quit drinking. He has been diagnosed with COPD, fractured NOF, gout and hypertension. He normally uses ipratropium and seretide, as well as allopurinol, ramipril, amlodipine and alendronic acid.

On examination of his scalp he has erythematous skin with thickened plaques. It is mildly tender, and looks slightly irritated.

What is the most appropriate treatment?

A. Emollients
B. Topical hydrocortisone
C. Miconazole shampoo
D. Topical 5-FU
E. Salicylate cream

D. Topical 5-FU

Topical 5-FU, imiquimob or difclofenac can be used as first line treatment for actinic keratosis

This patient has a typical presentation of actinic keratoses. Actinic keratoses, also known as solar keratosis, is an area of sun-damaged skin which is considered an early form of cutaneous squamous cell carcinoma. There are several treatment options available, including topical 5-FU, topical diclofenac, cryotherapy and topical imiquimod.

Topical hydrocortisone and emollients are used for eczema. Miconazole shampoo is used for tinea and other infections. Salicylate creams can help with scales, especially in psoriasis.

Question 101 of 182

A 72-year-old man presents to the general medical clinic with his daughter. She is concerned about him because she has developed shingles and is concerned that he may catch shingles from her, and wonders if he could have a shingles vaccination. The patient is well and has no rash. He has a past medical history of type 2 diabetes which is diet controlled, high cholesterol, previous stroke without residual weakness, as well as a fractured hip repaired with hemiarthroplasty. He normally takes aspirin, atorvastatin and paracetamol.

He believes that he has had chicken pox when he was young but does not think that he has ever had shingles or a shingles vaccination.

What is the most appropriate plan?

A. Offer oral aciclovir

B. Offer shingles vaccination

C. Explain that he is not eligible for shingles vaccination and should monitor for symptoms of shingles

D. Arrange admission for IV aciclovir

E. Offer oral corticosteroids

B. Offer shingles vaccination

People aged between 70-79 years old are eligible for shingles vaccine

The patient is not at risk of catching shingles from another person with shingles, but is eligible for shingles vaccination. This is the most appropriate plan available. It could be argued that he should be asked to monitor for symptoms only as he is not of increased risk of shingles, but as he is eligible for vaccination and has inquired this should be offered.

Oral aciclovir can be used to treat acute shingles, whilst ophthalmic shingles would need hospital admission for IV aciclovir or ganciclovir.

Question 1 of 82

A 37-year-old gentleman is referred to the cardiology clinic with a 7-month history of bilateral foot pain. Initially, the pain only occurred towards the end of his daily commute; improving when he sat at his desk, but over the past 3 weeks his symptoms have progressed. The pain is now provoked by walking short distances and has occasionally occurred at rest. He describes being woken by the pain at night, although he is able to obtain relief by standing on the cool tiles in his bathroom. He eventually decided to see his GP when he developed similar pain in his right wrist 10 days ago.

His past medical history is unremarkable. There is no history of hypertension, dyslipidaemia or diabetes. He is a current smoker with a 20 pack year history. He drinks alcohol in moderation.

On examination, both feet are noted to be smooth and hairless. The right dorsalis pedis is difficult to feel and the medial 3 toes are cold and dusky. Urgent angiography is performed and shows a number of distal stenoses in the right limb affecting the anterior and posterior tibial arteries as well as the distal fibular artery. A number of similar stenoses are seen in the contralateral limb with 'corkscrew' collateral formation.

Blood tests are as follows:

Hb	130 g/l	Na +	139 mmol/l
Platelets	322*10 /l 9	K +	4.1 mmol/l
WBC	9.1*10 /l 9	Urea	5.3 mmol/l
Neuts	6.2*10 /l 9	Creatinine	113 µmol/l
Lymphs	2.1*10 /l 9	CRP	19 mg/l
Eosin	0.1*10 /l9		

MPO	negative
PR3	negative
anti-cardiolipin	negative

What is the most likely diagnosis?

A. Peripheral vascular disease
B. Microscopic polyangiitis
C. Takayasu arteritis
D. Thromboangiitis obliterans
E. Polyarteritis nodosa

D. Thromboangiitis obliterans

The patient presents with symptoms of arterial insufficiency; however, his age, lack of vascular risk factors and upper limb involvement should alert you to the fact this may not be a simple case of peripheral vascular disease.

Both microscopic polyangiitis and polyarteritis nodosa are multisystem vasculitic disorders characterised by constitutional symptoms such as weight loss, fever, anorexia, and fatigue. Renal involvement and skin rash are common, as is an association with p-ANCA (MPO) antibodies and elevation of the inflammatory markers.

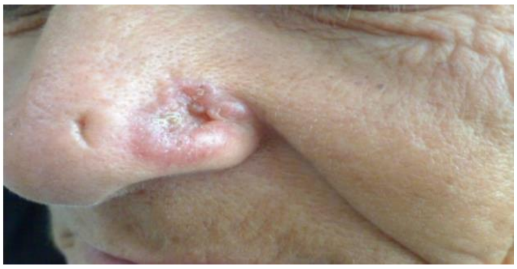
Takayasu arteritis is a large vessel vasculitis that affects the aorta and its branches. It is more common in females and would not typically cause the isolated small and medium vessel stenoses described above.

Thromboangiitis obliterans is an inflammatory disorder of the small to medium-sized arteries and superficial veins in the distal extremities. Segmental arterial inflammation and thrombus formation lead to peripheral limb ischaemia, with the disease often pursuing an aggressive course. The aetiology is unclear but the condition is strongly associated with smoking, and disease progression is closely linked to continued tobacco use. It most commonly affects men aged 20-40.

The diagnosis is made on the basis of a characteristic history and examination, angiographic findings and the exclusion of proximal emboli and vasculitic disorders. Typical findings on angiography include segmental occlusions of the distal arteries in the hands and feet, 'corkscrew' collateral vessels and the lack of widespread atherosclerotic disease. Thromboangiitis obliterans is, therefore, the most appropriate answer to the question.

Question 2 of 82

A 66-year-old man presents to the GP with the following lesion on his nose, which he says has been growing over the course of several years:



He is otherwise well in himself, and takes amlodipine for hypertension.

Given the likely diagnosis, which of the following is the most appropriate, definitive management?

A. Conventional surgery
B. Cryotherapy
C. Fluorouracil
D. Imiquimod
E. Mohs surgery

E. Mohs surgery

The most appropriate option is Mohs surgery. The lesion shown here is a basal cell carcinoma (BCC), which are characterised by slow growth and local invasion. BCCs are typically found on sunexposed sites, such as the nose in this scenario. The picture here depicts the typical features of a BCC - it is a papule with 'pearly, rolled edges' and a depressed, crater-like central area which has followed ulceration. BCCs often have associated telangiectasia, which is not so obvious in this photo. Definitive management involves surgical removal. Mohs surgery is the preferable option for cosmetically challenging locations such as the face, removing very small amounts at a time, thus leaving the smallest scar.

Conventional surgery almost always results in complete cure. However, it should be reserved for areas where tissue sparing is not important, as it is likely to result in significant scarring. For sensitive areas such as the face, Mohs surgery is more appropriate.

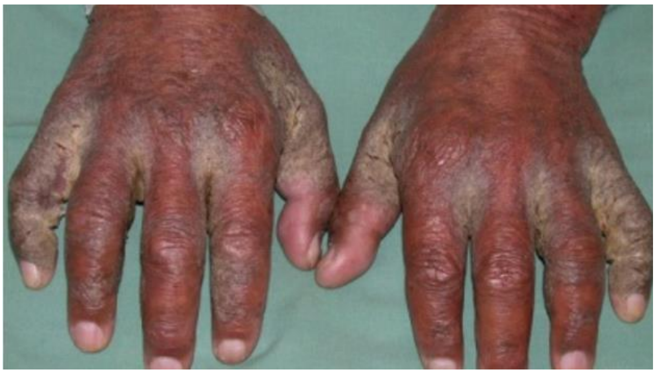
Cryotherapy is a potential option if the BCC is not advanced and does not extend deeply upon palpation. This BCC, however, appears to have ulcerated and left a crater visible in the photo, which suggests it is more advanced. Furthermore, the question asks for 'definitive' management - surgery is the most definitive route for BCCs.

Fluouracil is a topical option, often used when multiple BCCs are present. It can significantly reduce the size and appearance of the lesions over time, with repeated application. It is not, however, the most definitive management.

Imiquimod is another topical option and has more evidence supporting its efficacy than fluouracil. It has a good cure rate, although is not as definitive or successful as surgery.

Question 3 of 82

A 45 year old man with mild learning difficulties and newly diagnosed advanced HIV (CD4 count 65 /mm3) attends A&E from his residential home. He has an intensely itchy rash on his hands, arms, groin, legs and feet. He was recently commenced on Atripla (Emtritcitabine, Tenofavir, Efavirenz) and co-trimoxazole.



There is extensive crusted scaling on his arms, groins and hands. On closer examination there are grey burrow marks between the webs of his fingers and toes. There are itchy nodules extensively over the palms and backs of his hands.

What is the most appropriate treatment?

A. Isolate and apply topical malathion cream for 24 hours
B. Isolate and stop co-trimoxazole
C. Discharge with topical steroids
D. Change his antiretroviral therapy to a second line regimen
E. Isolate and give ivermectin orally

E. Isolate and give ivermectin orally

The correct answer is **Isolate and give ivermectin orally**. This patient has a clinical presentation consistent with scabies, which is caused by the mite *Sarcoptes scabiei*. Immunocompromised patients are at risk of developing crusted scabies (formerly known as Norwegian scabies). It typically begins as poorly defined red patches that then develop into thicker, scaly plaques between the fingers, under the nails, or diffusely over palms and soles. Mites can also potentially collect in nail beds, causing the nail plates to split.

In patients with advanced HIV, such as this patient with a CD4 count of 65/mm3, scabies can be more severe and difficult to treat. Ivermectin is an effective oral treatment for scabies and is particularly useful in cases where topical treatments may be less effective or challenging to apply.

Isolate and apply topical malathion cream for 24 hours would be an appropriate treatment option for uncomplicated scabies in an immunocompetent individual. However, given the severity of this patient's infection and his immunocompromised state due to advanced HIV, oral ivermectin would be a more suitable choice.

Isolate and stop co-trimoxazole is not an appropriate management plan because stopping cotrimoxazole will not address the underlying cause of the rash - scabies. Co-trimoxazole was likely prescribed as prophylaxis against opportunistic infections due to the patient's low CD4 count.

Discharge with topical steroids would not effectively treat the underlying scabies infection. Topical steroids may provide temporary relief from itching but do not eradicate the mites causing the infection.

Change his antiretroviral therapy to a second-line regimen is not indicated in this scenario. There is no evidence to suggest that changing antiretroviral therapy would impact this patient's scabies infection or improve his symptoms. The focus should be on treating the scabies infection with appropriate medication, such as oral ivermectin.

Question 4 of 82

A 52-year-old gentleman presents with a lesion on his tongue. He has noticed feeling 'something odd'in his tongue and noticed a white rash on the side of the tongue which he is unable to wipe away. He has a past medical history of recurrent herpes simplex sores, HIV, hypertension and an ankle fracture requiring surgery. He takes only losartan. On examination, there is a thickly furrowed white lesion on the side of his tongue.

What is the most appropriate treatment that would help resolve the lesion?

A. Antifungal treatment
B. Antiretroviral treatment
C. Long-term antibiotics
D. Oral steroids
E. Surgical resection

B. Antiretroviral treatment

The correct answer is antiretroviral treatment. This patient with is described as having leukoplakia. A white lesion on the side of the tongue is most commonly candida or lichen planus, but the fact that the lesion can't be rubbed off makes leukoplakia a more likely diagnosis, especially in the context of HIV. A biopsy should be done to exclude a squamous cell cancer. Despite the lesion being caused by EBV, treating the underlying HIV causes improvement.

Question 5 of 82

A 55-year-old gentleman was referred to the urgent outpatient dermatology clinic with a flare up of long standing psoriasis. HIs GP had diagnosed him with psoriasis five years ago. It usually affected the extensor surfaces of his elbows and was well controlled with a combination of topical vitamin D analogue and topical corticosteroid. For the last four weeks, however, the rash had become extensive across his chest and back. Despite an intensive trial of topical high potency corticosteroid and further topical vitamin D analogue, his rash had seemed to be worsening, leading to his referral.

His past medical history comprised hypercholesterolaemia and hypertension which were diagnosed at a well man check by his GP three months ago. As he also suffered from palpitations which were thought to be induced by anxiety, he was commenced on propranolol modified release 80mg OD, as well as simvastatin 40mg OD. He also suffered from chronic low back pain for the last few years and used naproxen 500mg BD with lansoprazole 30mg OD for the last five years with good effect. He also admitted to using over the counter aspirin 75 mg OD for the last nine years as his friend was prescribed this by her doctor.

Examination revealed a widespread rash comprising of multiple scaly erythematous discrete plaques across his chest, abdomen, back and extensor surfaces of his elbows and knees. He was otherwise systemically well.

Which is the single most likely causative agent responsible for the deterioration?

A. Propranolol
B. Simvastatin
C. Naproxen
D. Lansoprazole
E. Aspirin

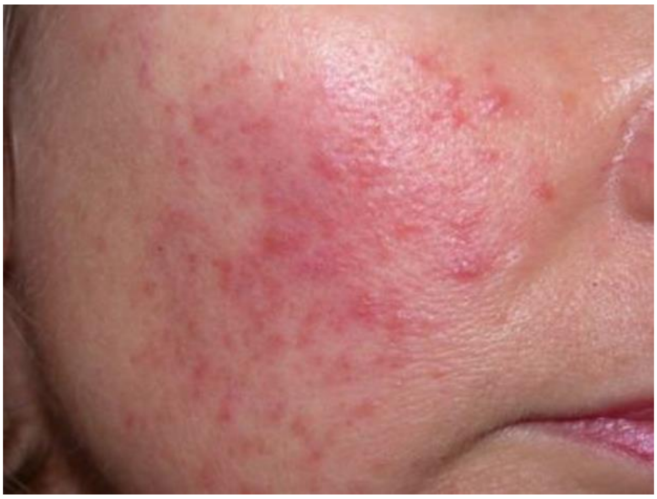
A. Propranolol

Beta-blockers are known to exacerbate plaque psoriasis

Psoriasis may be exacerbated by a variety of causes. With respect to drugs, the most common suspects are lithium and beta blockers. Other agents include antimalarials, ACE inhibitors and non-steroidal anti-inflammatories. The patient was indeed using naproxen and aspirin. However, he was commenced on propranolol recently with a subsequent exacerbation of his psoriasis. The most suitable answer, therefore, is propranolol.

Question 6 of 82

A 49-year-old woman complains of 'spots' on her cheeks. She has tried using her daughter's 'Clearasil' but this has had no effect.



What is the most likely diagnosis?

A. Seborrhoeic dermatitis
B. Systemic lupus erythematous
C. Perioral dermatitis
D. Late-onset acne vulgaris
E. Acne rosacea

E. Acne rosacea

Rosacea features:

- nose, cheeks and forehead
- flushing, erythema, telangiectasia → papules and pustules

The correct answer to this question is **Acne rosacea**. This condition, also known as rosacea, is a common skin disease that often begins with a tendency to blush or flush more easily than other people. The redness can slowly spread beyond the nose and cheeks to the forehead and chin. Even the ears, chest, and back can be red all the time. The image shows typical features of Acne rosacea: erythema (redness) of the cheeks with some papules (small bumps) and pustules. It's important to note that this condition is different from acne, which typically includes blackheads and whiteheads - these are not present in rosacea.

Seborrhoeic dermatitis is an incorrect option because it usually presents with greasy-looking, flaky skin in areas rich in sebaceous glands such as the scalp, eyebrows, sides of the nose, behind the ears and middle of the chest. It does not typically present with spots on cheeks.

Systemic lupus erythematosus (SLE) could potentially cause a malar rash ('butterfly' rash) on cheeks but this would usually also involve the bridge of the nose which isn't mentioned in this case. Additionally, SLE-related rashes are photosensitive and there was no mention of sunlight exacerbating symptoms in this scenario.

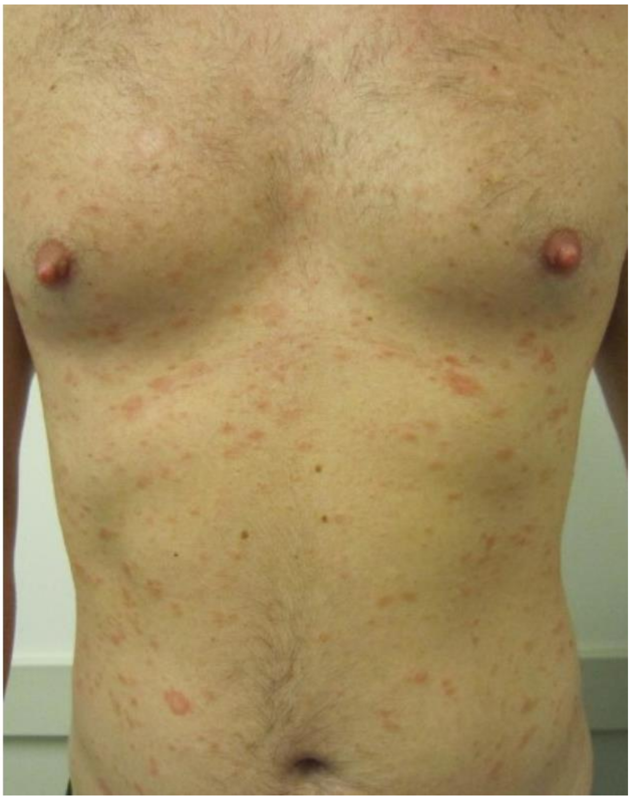
Perioral dermatitis, as its name suggests, tends to occur around the mouth area (although sometimes it can affect areas around the eyes or nose). It presents as groups of itchy or tender small red papules. It's unlikely in this case due to the location being described as 'cheeks'.

Lastly, **Late-onset acne vulgaris**, while possible at any age including adulthood ('late-onset'), would typically include comedones (blackheads & whiteheads), which were not mentioned here. Furthermore, over-the-counter treatments like Clearasil often have some effect on acne vulgaris but none on rosacea according to the patient's history.

Question 7 of 82

A 19-year-old man is referred to the dermatology clinic by his GP with a 2-week history of a rash over his torso. He has tried applying topical emollients but has seen no improvement in his symptoms. The rash is not painful and only mildly itchy. He denies any preceding illness or recent travel and there is no past medical history of note and no known allergies.

On examination, his observations are recorded within normal limits. His rash is examined, as shown below:



Given the likely diagnosis, what is the recommended management for this patient?

A. Oral phenoxymethylpenicillin
B. Reassurance and discharge
C. Topical 1% hydrocortisone
D. Topical ketoconazole
E. UVB phototherapy

B. Reassurance and discharge

Reassurance and discharge is correct. This patient has multiple small, oval-shaped, red plaques that are located on the trunk. Looking closely at these lesions, some appear to have a fine peripheral scale. This patient has a diagnosis of pityriasis rosea, a self-limiting rash that is characterised by a large round 'herald patch', usually found on the chest, abdomen, or back. It mostly affects children and young adults and is thought to be precipitated by bacterial and viral infections (e.g. human herpes virus 6 and 7).

Oral phenoxymethylpenicillin is incorrect. This is used in the treatment of Streptococcal infections (e.g. tonsillitis). Streptococcal infections are associated with guttate psoriasis that presents as an eruption of teardrop-shaped erythematous, scaly lesions. In contrast, the lesions of pityriasis rosea have a fine scale confined to the peripheries of the lesion. Furthermore, given the lack of signs of a streptococcal throat infection, phenoxymethylpenicillin is unlikely to be of any benefit.

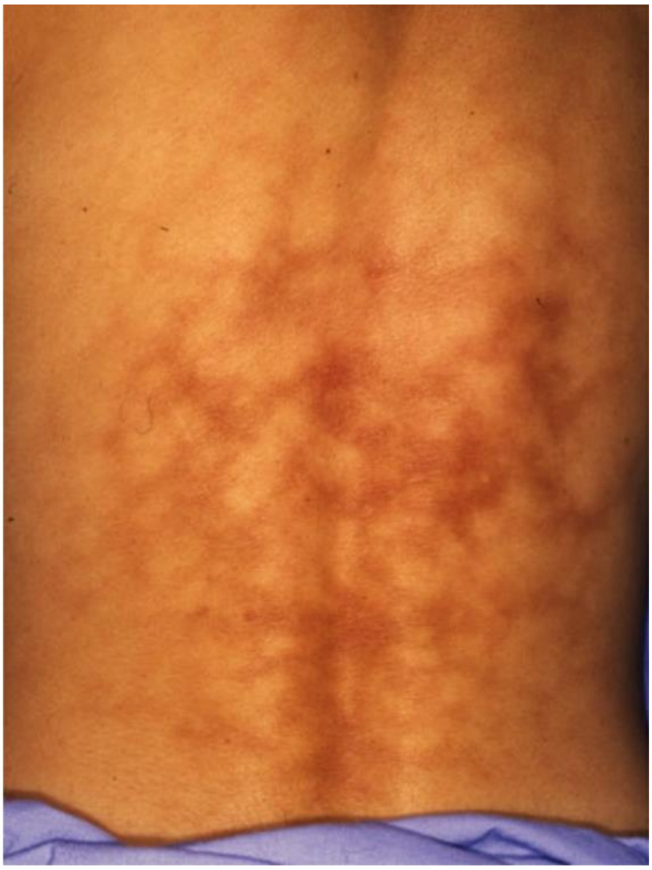
Topical 1% hydrocortisone is incorrect. Topical corticosteroids are usually used in the treatment of eczematous diseases that typically present as a dry, itchy and scaly rash which is not seen here. They can also be used in the management of localised chronic plaque psoriasis. However, it plays no role in the management of pityriasis rosea.

Topical ketoconazole is incorrect. Topical ketoconazole is used in the treatment of fungal infections such as pityriasis versicolor. The rash is mostly seen on the trunk, neck and upper limbs. The patches can vary in colour from pink, brown to pale (typically paler than the surrounding skin). They are usually asymptomatic but can occasionally be itchy. Heat and moisture are risk factors for developing the rash.

UVB phototherapy is incorrect. This is used in the management of chronic plaque psoriasis and guttate psoriasis that cannot be controlled with initial topical management. However, it is not used in the management of pityriasis rosea.

Question 8 of 82

A 34-year-old man with a long history of back pain asks you to have a look at his back. His wife has noticed a rash.



What is the most likely diagnosis?

A. Pityriasis rosea
B. Erythema multiforme
C. Erythema ab igne
D. Pityriasis versicolor
E. Cold urticaria

C. Erythema ab igne

This is a typical erythema ab igne rash. He may have been applying a hot water bottle to his lower back to try and relieve the pain.

Question 9 of 82

A 22-year-old female with a background history of iron deficient anaemia and eczema presents with a skin rash. She takes ferrous sulphate 200mg twice daily but no other medication. On examination, she has an itchy bullous rash consisting of papules and blisters on the knees and elbows. Direct immunofluorescence of a skin biopsy shows immunoglobulin A (IgA) deposition in a granular pattern within the upper dermis.

Which of the following would be the most appropriate treatment?

A. Emollients and topical hydrocortisone
B. Dapsone and a gluten-free diet
C. Oral steroids
D. Emollients and a vitamin D analogue
E. A gluten-free diet and topical hydrocortisone

B. Dapsone and a gluten-free diet

Dermatitis herpetiformis is typically treated with a gluten-free diet and topical dapsone

The clue here is the history of a young person who is iron-deficient and develops a rash which is not typical of eczema or psoriasis. Coeliac disease can often present with iron deficiency and no bowel manifestations and practitioners are lucky if their patient develops the characteristic rash of dermatitis herpetiformis, pointing them to the diagnosis.

Question 10 of 82

A 82-year-old female is brought into hospital after she was witnessed by her carer to 'twitch all 4 limbs' and lose consciousness for about 5 minutes. She is known to have previously had a stroke, resulting in mild right sided weakness 5 years previously and is a type 2 diabetic. She is diagnosed with a first generalised seizure and is prescribed lamotrigine. Four days after her admission, whilst awaiting a new package of care, she complains of a sore throat. The next day, you note peeling skin from her bottom lip and a new erythematous, well-defined macule on her trunk.

What is the most likely diagnosis?

A. Stevens-Johnson syndrome
B. MRSA cellulitis
C. Impetigo
D. Scabies
E. Necrobiosis lipoidica

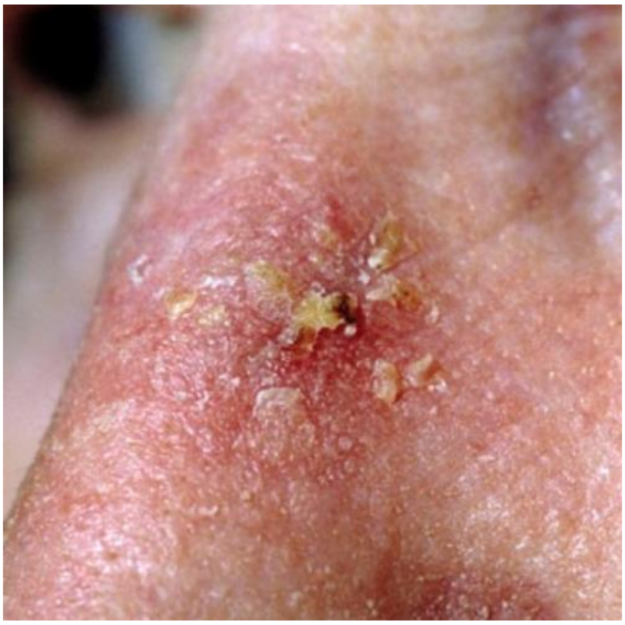
A. Stevens-Johnson syndrome

The clinical syndrome of a sore throat, macular erythematous rash and mucous involvement is suggestive of Stevens-Johnson/ toxic epidermal necrolysis spectrum disorder.

Although Stevens-Johnson syndrome is a classic, well-recognised side effect of lamotrigine, it must be remembered that rashes commonly occur in up to 7% of all patients starting lamotrigine but only a small proportion develop Stevens-Johnson syndrome.

Question 11 of 82

A 67-year-old man presents with a rough, scaly lesion on his nose. He has a long history of sunrelated damage to his skin and has had similar lesions on his temples treated previously:



Which one of the following is he at an increased risk of developing if the lesion is not treated?

A. Pyoderma gangrenosum
B. Seborrhoeic keratosis
C. Malignant melanoma
D. Basal cell carcinoma
E. Squamous cell carcinoma

E. Squamous cell carcinoma

Actinic keratoses are premalignant lesions which may develop into squamous cell carcinomas if not treated.

Question 12 of 82

A 45-year-old woman is admitted with hallucinations. She has a long history of alcohol dependency and is being treated by her GP for hypothyroidism. Further questioning of her husband reveals a 3-month history of diarrhoea, difficulty swallowing and an itchy rash on her arms. Prior to this she had been irritable and suffered from regular bouts of vomiting.

The examination is limited as the patient is aggressive . There is a pigmented, scaly rash on her arms and neck. Heart sounds are normal, her chest is clear and her abdomen soft. Neurological examination reveals generalised weakness.

Blood tests show:

Hb	142 g/l
Platelets	200 * 10 /l9
WBC	5.3 * 10 /l9
Na +	140 mmol/l
K +	4.2 mmol/l
Urea	6 mmol/l
Creatinine	84 µmol/l
TSH	4.0 U/mL
Free T3	3.1 pg/mL
Free T4	1.5 ng/L

What is the most likely diagnosis?

A. Beriberi
B. Thyroxine toxicity
C. Systemic lupus erythematosus (SLE)
D. Pellagra

D. Pellagra

Niacin (B3) deficiency is characterised by dermatitis, diarrhoea and dementia, a condition known as pellagra

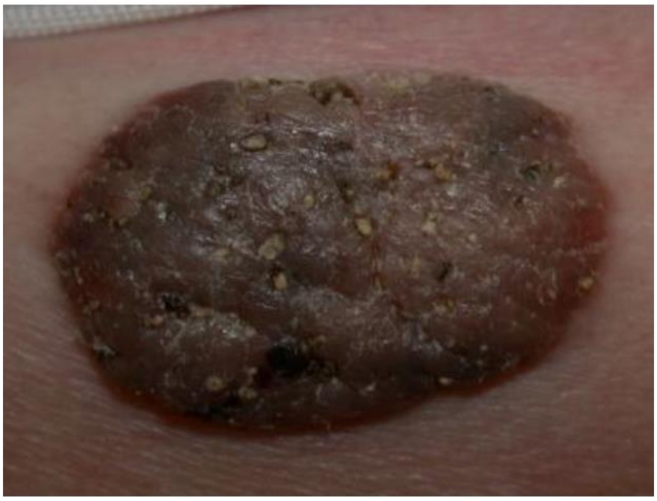
This lady has pellagra, caused by niacin (vitamin B3) deficiency. It occurs primarily in countries such as India and China, in which corn is a staple food. In the UK it is uncommon, and largely seen in patients with alcohol dependency or anorexia nervosa. Niacin is absorbed from foods such as beans and eggs and is also converted from amino acid tryptophan. Pellagra may manifest in carcinoid syndrome, in which tryptophan is converted to serotonin.

Over-treatment or deliberate overdose of thyroxine could cause some of this patient's symptoms, but her thyroid function tests are normal. SLE and Darier's disease both cause a light-sensitive rash, but would not cause the gastrointestinal symptoms. Cognitive disturbance may be seen in SLE.

Pellagra is distinguished from beriberi by the presence of a rash and gastrointestinal symptoms. Like beriberi, it can lead to confusion and memory deficits. Alcoholic patients may, of course, present with both. Other signs include dilated cardiomyopathy, oesophagitis and ataxia.

Question 13 of 82

A 60-year-old man presents with a lesion on his back which has increased in size over the past year. His past medical history includes ischaemic heart disease and spinal stenosis. There is no personal or family history of skin cancer.



What is the most likely diagnosis?

A. Malignant melanoma
B. Seborrhoeic keratosis
C. Bowen's disease
D. Dermatosis papulosa nigra
E. Actinic keratosis

B. Seborrhoeic keratosis

Question 14 of 82

A 36-year-old presents with a 4 months history of episodic generalised abdominal discomfort, nausea and vomiting. He reports no gastrointestinal symptoms prior to this period. He denies eating any unusual foods or recent travel within the past 2 years. His diarrhoea appears to be watery and he denies any blood stools. Eight months ago, he underwent a bone densimetry scan after breaking his hip and both femurs playing football, revealing a T-score of <-2.8. On examination, you notice brown papules, measuring 2 to 3 mm on each lower leg. When you press along the lesion and the surrounding skin, you note a red raised line is elicited. Abdominal examination reveals hepatosplenomegaly. His admission blood tests are as follows:

Hb	98 g/l
Platelets	195 * 10 /l9
WBC	4.9 * 10 /l9
ESR	70 mm/hr
Na +	138 mmol/l
K +	4.6 mmol/l
Adjusted calcium	2.81 mmol/l
Phosphate	1.3 mmol/l
CRP	22 mg/l
Serum tryptase 379 ng/ml (< 20)	
Complement	normal
C1 inhibitor	normal
Serum gastrin	normal
24 hour urinary 5-HIAA	normal

What is the most likely unifying diagnosis?

A. Zollinger-Ellison syndrome
B. Carcinoid syndrome
C. Hereditary angioedema
D. Cutaneous mastocytosis
E. Systemic mastocytosis

E . Systemic mastocytosis

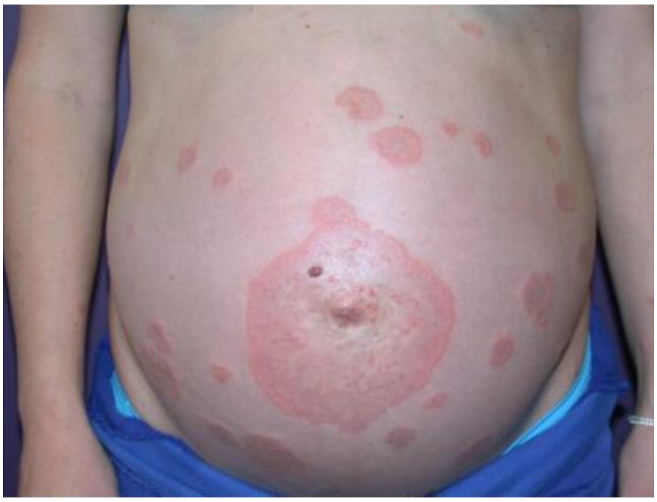
Localised erythema and urticaria on scratching, rubbing or stroking of the skin as described is classically known as Darier's sign, a manifestation of mast cell mediator release following physical irritation, classically seen in mastocytosis, a disorder of excessive mast cells. The next distinction is whether this represents symptoms limited to the skin or involves extracutaneous sites, hence the classification of cutaneous and systemic mastocytosis respectively. Systemic symptoms are a result of mast cell mediator and IgE release, which can cause gastrointestinal, neuropsychiatric and anaphylactic symptoms. A mild anaemia, as in this patient, is common in half of all systemic mastocytosis patients. Infiltration of spleen, liver and lymph nodes are noted in physical examinations as organomegaly. Excessive bleeding can also be an issue as a result of excess heparin release from mast cells. Osteoporosis in young patients should make you suspicious of bone marrow involvement.

Diagnosis of cutaneous mastocytosis is made upon signs and symptoms alone. Systemic mastocytosis is diagnosed according to a WHO criteria of major and minor symptoms: the major criterion involves the presence of mast cells in extracutaneous organs such as the liver and bone marrow, while minor criteria relies on serum tryptase over 20, surface markers of mast cells, atypical bone marrow mast cell morphology and a specific kit mutation. In the case of this patient, serum tryptase is significantly elevated in association with systemic involvement.

An important differential is that of hereditary angioedema, caused by a deficiency of C1 inhibitor deficiency and transient painful episodes of skin, laryngeal and bowel oedema. In this case, C1 inhibitor and complement are normal. Similarly, carcinoid syndrome should produce increased levels of urinary 24 hour 5HIAA secondary to excessive serotonin release. Lastly, Zollinger-Ellison syndrome only rarely presents with diarrhoea. Serum tryptase is almost always normal with elevated gastrin levels.

Question 15 of 82

A 27-year-old woman who is 34 weeks pregnant presents with an itchy, blistering rash over her abdomen. Initially she had a red rash around her umbilicus but it later spread.



What is the most likely diagnosis?

A. Pemphigoid gestationis
B. Seborrhoeic dermatitis
C. Polymorphic eruption of pregnancy
D. HELLP syndrome
E. Pompholyx

A. Pemphigoid gestationis

The correct diagnosis for this patient is **Pemphigoid gestationis**. This condition, also known as Herpes gestationis, is a rare autoimmune blistering disorder that occurs in pregnancy. It typically presents during the second or third trimester with pruritic, urticarial plaques and tense vesicles or bullae. The rash often starts around the umbilicus and then spreads to other parts of the body, which matches the description provided in the question. There may be remissions and exacerbations throughout pregnancy, with flares often occurring at delivery.

Seborrhoeic dermatitis is an incorrect answer. While it can cause an itchy rash, it typically affects areas of the body rich in sebaceous glands such as the scalp, eyebrows, sides of the nose and behind ears rather than the abdomen. Moreover, its presentation does not include blisters.

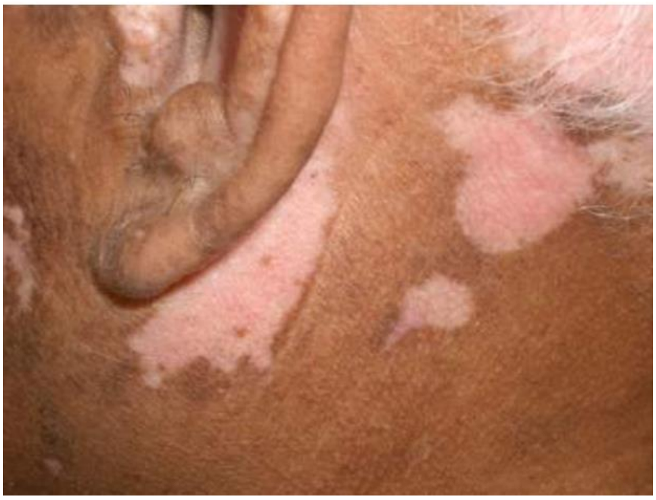
Polymorphic eruption of pregnancy, formerly known as PUPPP (pruritic urticarial papules and plaques of pregnancy), is another incorrect option. Although it does present with intense itching during late pregnancy, its distinguishing feature is that it usually spares the umbilical area - unlike in this case where the rash started from around the umbilicus.

HELLP syndrome (Hemolysis Elevated Liver enzymes Low Platelet count) is also not correct. HELLP syndrome is a severe form of pre-eclampsia characterised by haemolysis, elevated liver enzymes and low platelet count. It may sometimes present with epigastric or right upper quadrant pain due to liver swelling but a blistering rash would not be a typical presentation.

Lastly, **Pompholyx**, also known as dyshidrotic eczema, primarily affects hands and feet causing small blisters along the sides of fingers or toes as well as palms or soles - not the abdomen or around the umbilicus which makes it an unlikely diagnosis for this case scenario.

Question 16 of 82

Please look at the image below:



Which one of the following is LEAST likely to have a role in the management of this patient?

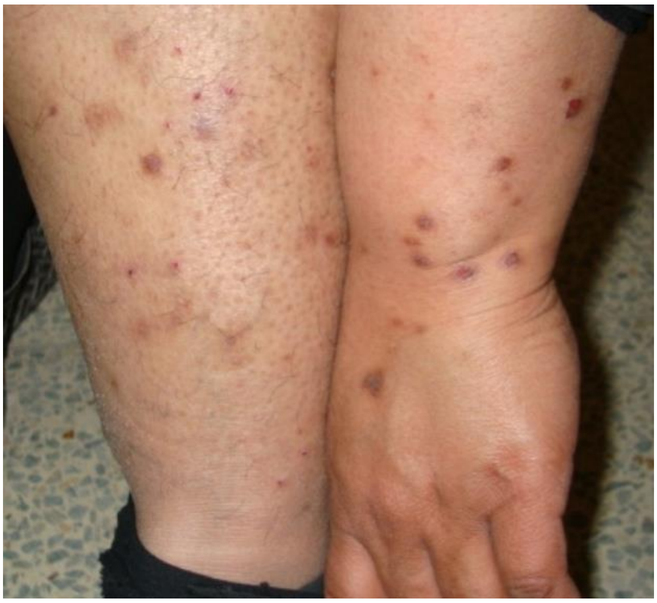
A. Sun block
B. Topical tacrolimus
C. Phototherapy
D. Topical ketoconazole
E. Topical corticosteroids

D. Topical ketoconazole

There is no role for antifungal therapy in vitiligo.

Question 17 of 82

A 63-year-old woman presents with an itchy rash on her arms and legs and also mentions that she has a white discolouration on her tongue and cheeks. The rash has been present for the last 2 months and over-the-counter emollients have not helped. This woman works in a bakery and wears gloves but she took the last 2 weeks off work. Also, she has been in a stable relationship since her divorce 10 years ago.



What is the most likely diagnosis?

A. Eczema
B. Lichen planus
C. Lichen simplex
D. Prurigo nodularis
E. Syphilis

B. Lichen planus

The image shows a typical lichen planus rash. The image shows a plaque-like erythematous rash distributed on the hand, arm, and leg. There are some small areas of desquamation where the skin appears peeled and exposes the dermal layer (most prominently here on the proximal arm). Lichen planus can also be described as having shiny red or purple papules although it is difficult to tell if any lesions are raised in this image.

Lichen planus - this is the correct answer. This typically affects people over 40 years old and about 50% of cases affect the oral mucosa. Fine white or grey lines or dots may be seen overlying the rash and oral mucosal lesions, otherwise named Wickham's striae. The cause is not well understood but it is thought to be a T-cell-mediated immune disorder. The diagnosis is mainly clinical but a skin biopsy will show globular deposits and T-cell infiltration. Most cases resolve spontaneously within a year but topical steroids can help with symptoms.

Eczema - this is incorrect as eczema can appear with excoriations and lichenification, there is often more involvement on the flexor surfaces. Topical steroids and emollients are the treatment. It might be expected that emollient use or staying away from working at the bakery would help (contact allergen) instead of the history in the stem.

Lichen simplex - this is incorrect. In lichen simplex, one or more plaques with lichenification will appear in an area that is heavily scratched. The itch/scratch cycle perpetuates the rash. Topical steroids and scratch avoidance are the treatment.

Prurigo nodularis - this is incorrect as this will appear as pruritic nodules often on the extremities. This is a chronic skin condition characterised by very itchy firm lumps. It can appear anywhere on the body but is most common in a symmetrical distribution on the arms, legs, back, and torso. There is a correlation between prurigo nodularis with atopy and internal disease.

Syphilis - this is incorrect. Syphilis is caused by the spirochete *Treponema pallidum*. It is primarily sexually transmitted and can cause many signs and symptoms similar to other diseases and so is nicknamed 'the great imitator'. This kind of rash is more typical of secondary infection which follows an incubation period of 10-90 days. One would expect other symptoms to accompany this stage like fever, headache, malaise, myalgia, arthralgia, and lymphadenopathy. There is no likely history of any sexual transmission or other systemic symptoms in the stem, making this answer less likely.

Question 18 of 82

A 57-year-old man has returned to the emergency department after struggling to cope with pain from shingles, diagnosed two days ago. He was started on aciclovir, paracetamol and codeine, but was still struggling and therefore yesterday was started on neuropathic analgesia with gabapentin, but has found this to be ineffective. He has a past medical history of oesophagitis, atrial fibrillations and gout. He normally takes omeprazole, warfarin, bisoprolol and allopurinol.

What would be the most appropriate plan?

A. Admit for IV aciclovir
B. Admit for IV ganciclovir
C. Stop all opiates
D. Add an NSAID
E. Add prednisolone

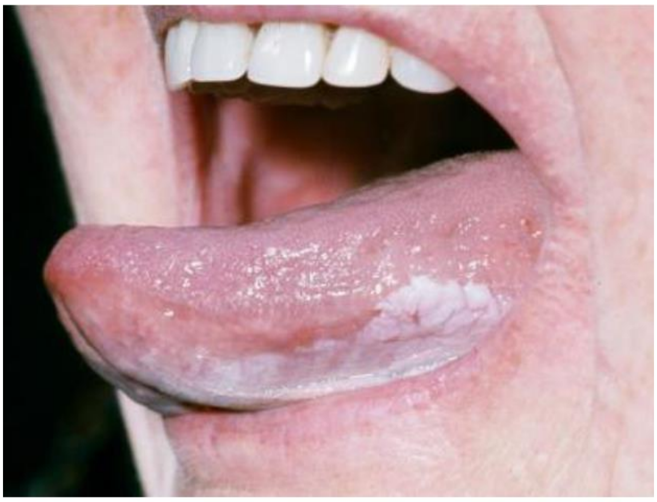
E. Add prednisolone

Corticosteroids can be used in refractory pain in shingles if simple analgesia and neuropathic analgesia do not help, but only for acute shingles

This patient has been suffering with severe pain due to acute shingles despite management with paracetamol, codeine and neuropathic agent. Adding an NSAID would be inappropriate due to this history of gastritis, whilst IV antiviral treatment would not make any difference in regards to the pain management. NICE suggests prednisolone can be used to help in the management of pain in acute shingles, and this can be offered for this patient.

Question 19 of 82

A 52-year-old man asks you to look at the side of his tongue. The white patches have been present for the past few months and are asymptomatic. He is a smoker who is known to have type 2 diabetes mellitus.



What is the most likely diagnosis?

A. Candidiasis
B. Squamous cell carcinoma
C. Lichen sclerosus
D. Oral leukoplakia
E. Geographic tongue

D. Oral leukoplakia

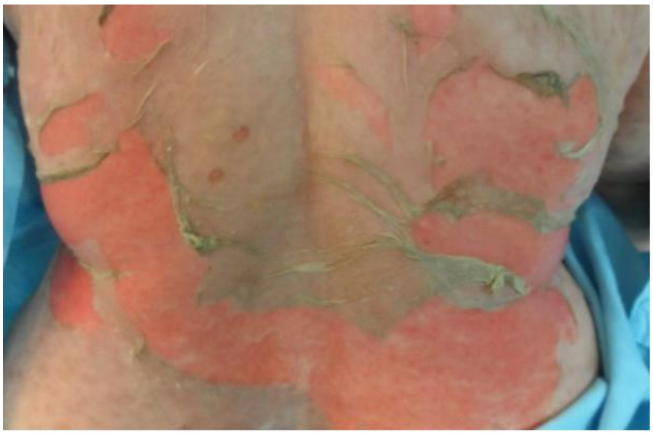
The asymptomatic and prolonged nature of the symptoms goes against a diagnosis of candidiasis. Lichen planus (rather then sclerosus) is a differential diagnosis but tends to have a slightly different appearance - typically a symmetrical white lace-like pattern on the buccal mucosa. Squamous cell carcinoma is not the most likely diagnosis as only around 1% of oral leukoplakias become malignant.

This patient should be referred for a biopsy to confirm the diagnosis.

Question 20 of 82

A 42-year-old female originally presents to her GP with a small skin lesion on her forearm after being bitten by her new kitten 4 days previous. There was some redness with minimal discharge at the site, but the patient denied any systemic symptoms. Her GP send off a swab of the area and started the patient on a course of amoxicillin.

Initial the patient felt the wound appeared to be healing but after a few further days, she became systemically unwell with fever, sweating and feeling weak. She noticed her skin appeared to be peeling all over her body (see below) and her considered family rushed her into hospital. On review of her GP records, the wound swab culture grew *Staphylococcal aureus*.



Other than supportive care what other management has been shown to have the most benefit for this patient's condition long term?

- A. Corticosteroids
- B. IV antibiotics
- C. Intravenous immunoglobulin
- D. Skin graft
- E. Surgical debridement

C. Intravenous immunoglobulin

This patient has presented with toxic epidermal necrolysis (TEN) most likely secondary to

amoxicillin started by her GP. The specific pathophysiology of TEN is unknown but it is thought to be due to an immune response and a drug precipitate is attributed to 80-95% of cases. Patients are systemically unwell and the epidermis separates resulting in a peeling like appearance as seen in this case. Management involves stopping the precipitating drug and supportive care, often requiring intensive care. Recent research has shown intravenous immunoglobulin therapy is effective in reducing mortality and morbidity and so is commonly used as first-line management. Other treatments options including immunosuppressive agents and plasmapheresis have also be used but are normally second-line.

Although the patient had a previous skin infection it is not the cause of their acute deterioration. The rapid onset following starting a new drug (in this case an antibiotic) is in keeping with a TEN reaction. IV antibiotics would be required in the differential diagnosis of staphylococcal scaled skin syndrome (SSSS) however this is less likely in this case despite the patients confirmed *Staphylococcal aureus* infection. SSSS is rarely seen in adults and is less severe than TEN. Also in SSSS the cleavage site is the intraepidermal as opposed to the TEN which involves necrosis of the full epidermal layer as seen in this patient. Antibiotics are sometimes used prophylactically in TEN and are often required later as patients are at high risk of infection, but they are not required initially.

Immunosuppressive medication such as corticosteroids have been used in TEN but intravenous immunoglobulin is more effective and therefore is commonly used as first-line.

Skin grafts may be required much later in the course of TEN to promote healing if there is extensive skin disruption however it is not required in all cases and the benefits on mortality and morbidity are unclear.

Surgical debridement is not commonly required as TEN is not due to an infection. Secondary infection can occur but again debridement is rarely required.

Question 21 of 82

An 18-year-old male university student presents to the emergency department due to itching. He has been experiencing worsening itching of his hands and arms after two months of living in student accommodation with two other students. He has found the itching to be progressively worse and not manageable with over the counter chlorphenamine. He has had no other medical problems and takes no regular medications. He is an economics student and has no exposure to any chemicals. On examination there are linear burrows on the sides of some of his fingers on both right and left sides. In additions, there is evidence of lots of scratching, but does not appear infected. He is prescribed permethrin 5% as treatment.

What advise should be given in its application?

- A. Apply once to whole body
- B. Apply once to whole body and repeat in one week
- C. Apply once to whole body and repeat in one week and advise household contacts to dosame
- D. Apply daily to whole body for one week
- E. Apply daily to whole body for one week and advise household contacts to do same

C. Apply once to whole body and repeat in one week and advise household contacts to dosame

This patient likely has scabies. Scabies is more common in unkempt environments, which can occur in student housing. The diagnosis is strongly supported by severe, persisting pruritus in the presence of linear burrows. Treatment is with permethrin 5% but care must be given to ensure that the patient understands the treatment protocol or treatment failure is likely.

In scabies, advise all close contacts to be treated as well as the patient twice, with applications one week apart

Question 22 of 82

A 32-year-old lady attends with a facial rash for several weeks. She has an erythematous rash which looks greasy and has a fine scale over her face affecting her cheeks, nasolabial folds, eyebrows, nasal bridge and scalp.

What is the most likely diagnosis?

- A. Acne rosacea
- B. Systemic lupus erythematous (SLE)
- C. Eczema
- D. Seborrhoeic dermatitis
- E. Psoriasis

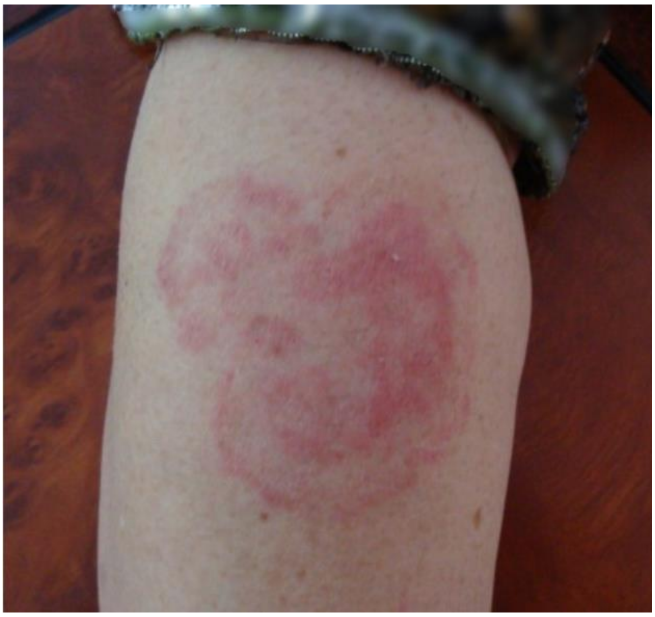
D. Seborrhoeic dermatitis

An itchy rash affecting the face and scalp distribution is commonly caused by seborrhoeic dermatitis

The description describes seborrhoeic dermatitis. The involvement of the nasolabial folds differentiates it from acne rosacea which typically spares this area and tends to include telangiectasia and pustules.

Question 23 of 82

A 26-year-old man presents with a lesion on his leg that he thinks has been present for a couple of weeks. It is somewhat itchy. He is otherwise fit and well, has no significant past medical history and takes no regular medications. The rash is shown below:



Given the likely diagnosis, what is the definitive management?

- A. Oral doxycycline
- B. Topical emollient
- C. Topical fusidic acid
- D. Topical hydrocortisone
- E. Topical ketoconazole

E. Topical ketoconazole

The rash here is characteristic of tinea corporis, colloquially known as ringworm. This is a fungal infection affecting the trunk and limbs. It has a characteristic appearance of a circular red patch with a raised edge and central hyperpigmentation as seen here, leading to the 'ring' appearance. Given its fungal nature, the most appropriate treatment is therefore topical ketoconazole. If the rash were significantly widespread, an oral antifungal agent may be more appropriate.

Oral doxycycline would be an appropriate treatment for Lyme disease. There was no history of a tick bite in this question, and the lesions are much more indicative of ringworm than of erythema migrans, which would instead present with a red papule or macule that gradually enlarges to form a bull's eye-like ring.

Topical emollient is insufficient. Whilst this may relieve the itch associated with the rash, it will not treat the fungal cause of ringworm.

Topical fusidic acid is inappropriate. This is an antibiotic and would be used for bacterial infections, such as mild impetigo or a mildly infected wound. It would not help in treating ringworm.

Topical hydrocortisone may be used short-term to relieve inflammation. The lesion in this photo does not look particularly inflamed. It is not the definitive management for this condition; an antifungal cream is required. In fact, prolonged use of steroid creams can actually worsen fungal rashes.

Question 24 of 82

A 24-year-old woman presents with new skin lesions.

She was the recipient of a renal transplant a year ago following IgA nephropathy. After several modifications to her immunosuppressive therapy, her graft function is stable with an eGFR of 80ml/min/1.73m². She also has a past history of hypothyroidism for which she takes levothyroxine.

She had first noticed the lesions 3 months ago. She had noted them a few years previously but on that occasion, they had disappeared of their own accord. They are not itchy.

On examination she has well demarcated depigmented patches on her arm, her shoulders and her upper back; the lesions shine a yellow-green colour under Woods lamp.

What is the most likely diagnosis?

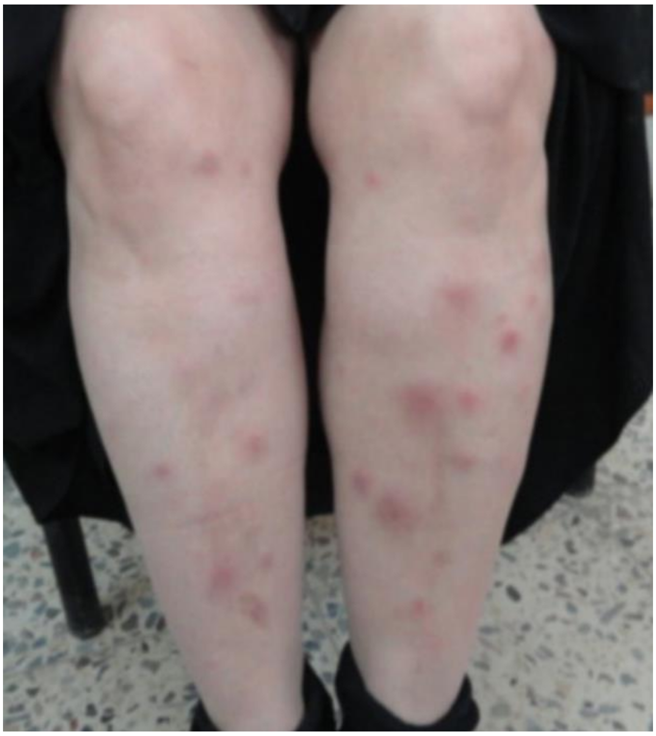
- A. Vitiligo
- B. Pityriasis alba
- C. Idiopathic guttate hypomelanosis
- D. Pityriasis versicolor
- E. Seborrhoeic dermatitis

D. Pityriasis versicolor

The yellow-green fluorescence under Woods lamp is the key factor here; none of the other answers fluoresces in this way. Hence vitiligo is not correct and the history of hypothyroidism (an autoimmune illness that may be associated with vitiligo) is irrelevant. The description is not consistent with seborrhoeic dermatitis, and the distribution of the rash is not consistent with either pityriasis alba or seborrhoeic dermatitis which typically affect the face.

Question 25 of 82

A 33-year-old woman presents with several days of tender lesions overlying her shins. She feels generally well in herself, aside from feeling slightly feverish. She has a past medical history of depression, anxiety, acid reflux, and ulcerative colitis, including a recent exacerbation that required oral prednisolone. She recently completed a course of doxycycline for a chest infection. Her regular medications include sertraline, azathioprine, and the combined oral contraceptive pill. Her rash appears as follows:



Given the likely diagnosis, which of the following is the most likely cause?

A. Azathioprine
B. Combined oral contraceptive pill
C. Doxycycline
D. Prednisolone
E. Sertraline

B. Combined oral contraceptive pill

The diagnosis here is erythema nodosum. This patient has the typical appearance of multiple erythematous, nodular lesions over the shins which appear inflamed. They are also described as being tender. These usually resolve within 6 weeks without scarring. There are several causes, including some medications - of the list above, the combined oral contraceptive pill is most strongly associated with the development of erythema nodosum.

Erythema nodosum is associated with inflammatory bowel disease. There have been a few case studies of azathioprine hypersensitivity being one possible explanation for this association. However, the combined oral contraceptive pill remains a much more likely option.

Doxycycline is not known to trigger erythema nodosum. Many antibiotics are, however, implicated in the condition, including penicillins and sulphonamides.

Prednisolone is not linked to the development of erythema nodosum. It is sometimes used to aid the resolution of the condition if symptoms persist.

Sertraline is a selective serotonin reuptake inhibitor used for anxiety and depression. Newer antidepressants are rarely linked to erythema nodosum, with fluoxetine, paroxetine, and venlafaxine being more strongly implicated than sertraline (although still relatively rarely linked compared to the combined pill).

Question 26 of 82

A 17-year-old male presents with a new skin condition which his mum noticed when they were on holiday in Spain. On examination, he has skin type V, with multiple small patches of depigmentation to the upper back. The patches appear mildly flaky but they are asymptomatic. He is usually well and has never had this condition before.

Which of the following is the most likely diagnosis?

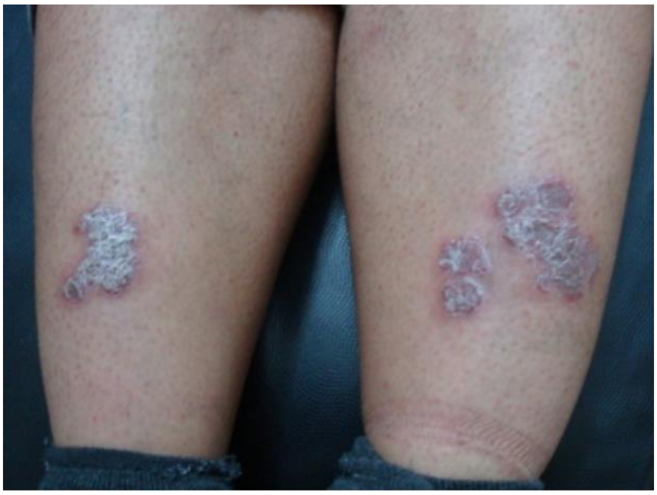
A. Pityriasis rosea
B. Atopic eczema
C. Vitiligo
D. Guttate psoriasis
E. Pityriasis versicolor

E. Pityriasis versicolor

This is a typical history of pityriasis versicolor, a skin condition caused by an overgrowth of Malassezia yeast. It is most common in young people, especially males. It causes multiple patches of skin discoloration, mainly to the trunk. The patches may appear pale brown, pink, or may appear depigmented especially in patients with dark skin. They may also be mildly flaky and itchy. The condition can often present after spending time in sunny, humid environments. It is treated with topical antifungals eg. ketoconazole shampoo.

Question 27 of 82

A 32-year-old woman presents with a rash over her shins. It has gradually been getting worse, despite moisturising. The rash is shown below:



She is otherwise fit and well and has no significant past medical history. She takes the combined oral contraceptive pill and uses a salbutamol inhaler for asthma.

Which of the following is the most likely diagnosis?

A. Chronic plaque psoriasis
B. Discoid lupus
C. Erythema nodosum
D. Lichen planus
E. Tinea corporis

A. Chronic plaque psoriasis

The correct answer is chronic plaque psoriasis, which is the most common presentation of psoriasis. The photo demonstrates a typical example of this condition - erythematous plaques covered with a silvery-white scale. There is a clear delineation between the plaques and the surrounding normal skin. What is slightly atypical about this photo is the location - generally, it presents on extensor surfaces such as elbows and knees. These lesions appear to be on the shins.

Discoid lupus would appear somewhat similar, but lesions would be less scaly and more regularly shaped. It is localised to above the neck in 80% of patients, or generalised across the body in the remaining 20% - unlike this presentation on the shins.

Erythema nodosum is inflammation of subcutaneous fat, presenting as tender red nodules on the shins - whilst the location here is correct, the appearance is not. The combined oral contraceptive pill is a known causative factor but, although this patient is taking the pill, the appearance is far more likely to be psoriasis.

Lichen planus is characterised by shiny, flat-topped, violaceous papules which are often clustered around the wrists and ankles. Rather than appearing scaly as in this photo, the papules are crossed by fine white lines called Wickham striae.

Tinea corporis is a fungal infection of the skin, affecting any part of the body. It is colloquially known as ringworm. Its appearance differs from the photo here - rather than scaly plaques, it has a red, ring-shaped appearance with central hypopigmentation.

Question 28 of 82

A 54-year-old female presented to the dermatology clinic with a history of worsening itchy rash. She has a history of psoriasis which had been quite well controlled but had worsened significantly in the last week. On examination, there are multiple well-demarcated oval, red/pink elevated lesions with overlying silvery scales over her elbows, knees, legs and scalp. She revealed that her GP had started her on a new tablet 2 weeks ago.

Which of the following drugs is likely to have resulted in the above presentation?

A. Citalopram
B. Lithium
C. Omeprazole
D. Ranitidine
E. Cimetidine

B. Lithium

Psoriasis: lithium may trigger an exacerbation

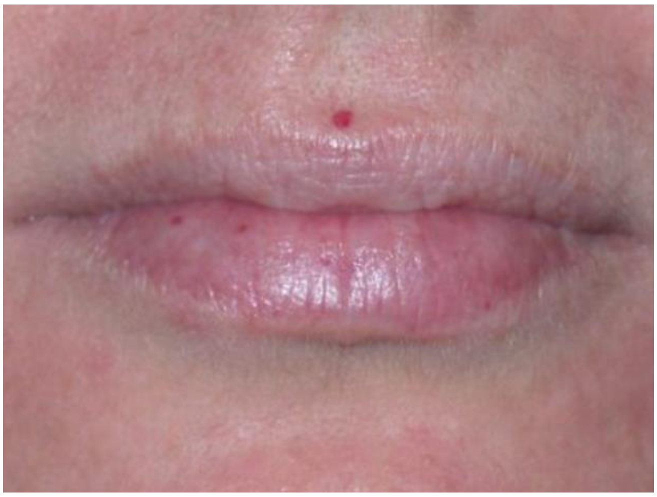
Psoriasis can be exacerbated by certain drugs such as:

- lithium
- beta blockers
- antimalarials
- non-steroidal anti-inflammatory drugs
- ACE-inhibitors
- antibiotics such as tetracycline and penicillin

Withdrawal of systemic steroids can also exacerbate psoriasis.

Question 29 of 82

This 21-year-old woman has a history of recurrent epistaxis:



What is the most likely underlying diagnosis?

A. Idiopathic thrombocytopenic purpura
B. Peutz-Jeghers syndrome
C. Anorexia nervosa
D. Combined oral contraceptive pill use
E. Hereditary haemorrhagic telangiectasia

E. Hereditary haemorrhagic telangiectasia

The correct answer is **Hereditary haemorrhagic telangiectasia**. This condition, also known as Osler-Weber-Rendu syndrome, is a genetic disorder that leads to abnormal blood vessel formation in the skin, mucous membranes and often in organs such as the lungs, liver and brain. Recurrent epistaxis (nosebleeds) are a common symptom because of the fragile blood vessels in the nasal lining.

Idiopathic thrombocytopenic purpura is incorrect. It's an autoimmune disorder where platelets are destroyed by antibodies leading to purpura (small red or purple spots caused by bleeding into the skin), but not typically recurrent nosebleeds. Epistaxis can occur but it's usually accompanied by other signs of bleeding like petechiae or ecchymosis.

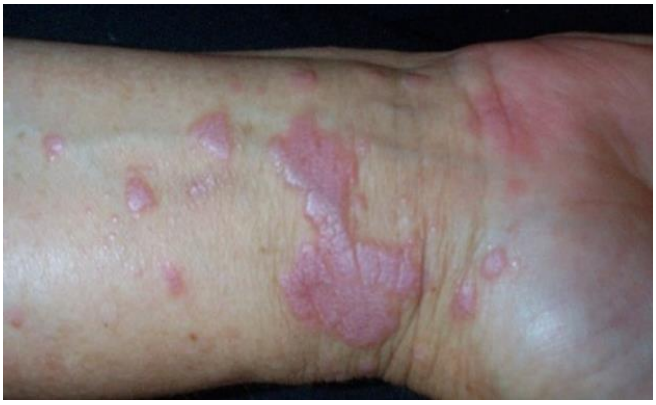
Peutz-Jeghers syndrome is also incorrect. This genetic condition results in the development of polyps in the digestive tract and hyperpigmented macules on lips, oral mucosa and digits. While these patients have an increased risk for intestinal bleeding, they do not typically present with recurrent epistaxis.

Next, **Anorexia nervosa** doesn't fit this clinical picture either. Although patients with anorexia nervosa may experience nosebleeds due to dry nasal passages from dehydration, it is not a characteristic feature of this eating disorder.

Lastly, **Combined oral contraceptive pill use** can increase risk of thromboembolic events due to increased oestrogen levels which promote coagulation but it does not cause recurrent epistaxis.

Question 30 of 82

A 43-year-old man presents with a three week history of an itchy rash on the flexor surfaces of his arms. He is generally fit and well and has no history of skin problems.



What is the most likely diagnosis?

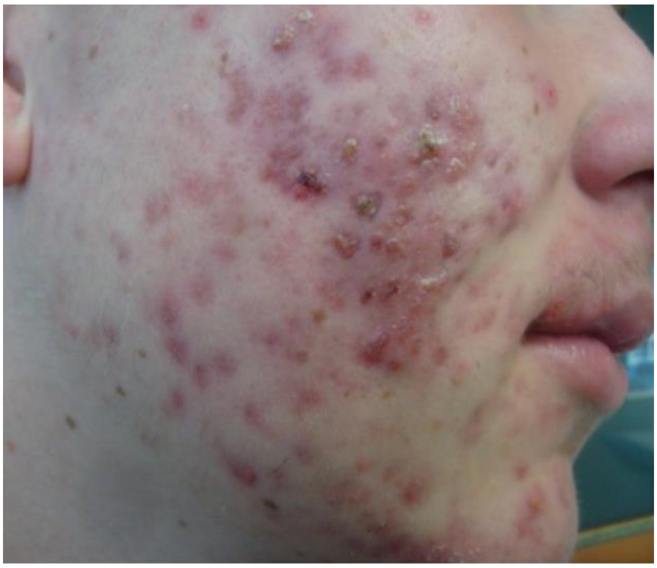
A. Morphea
B. Lichen sclerosus
C. Lichen planus
D. Psoriasis
E. Idiopathic urticaria

C. Lichen planus

Question 31 of 82

A 17-year-old boy presents to the dermatology clinic with a facial rash. He reports that the rash has been worsening over the past 6 weeks despite regular facial washing and multiple treatments including topical benzoyl peroxide and clindamycin. He now feels uncomfortable going outside due to feeling uncomfortable with his appearance. His current medications include oral lymecycline which was prescribed by his GP that he has been taking for the last 3 months.

His rash is examined, as shown below:



Given the underlying diagnosis, what is the first-line recommended management for this patient?

A. Continue oral lymecycline for further 3 months
B. Oral isotretinoin
C. Topical corticosteroids
D. Topical fusidic acid
E. Topical isotretinoin

B. Oral isotretinoin

Oral isotretinoin is correct. This patient has a diagnosis of acne vulgaris given the presence of inflammatory macules, papules and pustules in the photograph of this young male. Acne vulgaris is very common, particularly in adolescence. Associated features include comedones which are skin-coloured papules that can either be open (blackheads) or closed (whiteheads). Comedones are associated with increased sebum production and are formed when sebaceous ducts and hair follicles become blocked by debris. Looking closely at this patient's face, the areas of erythema are localised around a pilosebaceous unit. In those with acne vulgaris that has not responded to firstline measures and that are at risk of scarring, NICE guidelines recommend the use of oral isotretinoin.

Continue oral lymecycline for further 3 months is incorrect. Guidelines recommend that oral antibiotics should only be continued in the management of acne if the patient is seeing some improvement in their symptoms. As this patient is seeing a progression of symptoms despite antibiotics, an alternative treatment is needed.

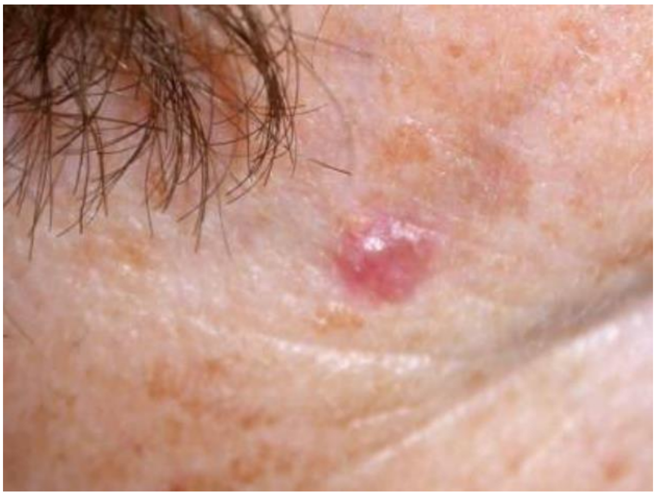
Topical corticosteroids is incorrect. This is used in the management of inflammatory skin lesions such as eczema or psoriasis. It is not used in the management of acne vulgaris.

Topical fusidic acid is incorrect. This is used in the management of superficial bacterial infections such as impetigo and mild cellulitis. Although topical antibiotics can be used in the management of acne vulgaris, fusidic acid is not recommended as per NICE guidelines.

Topical isotretinoin is incorrect. This is used in the initial stages of acne management and would not be recommended for the clearance of severe acne that has failed to respond to topical and oral antibiotics.

Question 32 of 82

A 78-year-old woman presents with a raised skin lesion on her face:



What is the most likely diagnosis?

- A. Keratoacanthoma
- B. Basal cell carcinoma
- C. Actinic keratosis
- D. Squamous cell carcinoma
- E. Malignant melanoma

B. Basal cell carcinoma

The correct answer is **Basal cell carcinoma**. It is the most common type of skin cancer and typically presents in elderly individuals on sun-exposed areas, such as the face.

Keratoacanthoma is an incorrect choice. Although it can appear similar to basal cell carcinoma, Keratoacanthomas tend to grow rapidly over weeks to months and then involute, leaving a scar. They also often have a central keratin-filled crater which is not apparent in this image.

The option **Actinic keratosis** is also incorrect. Actinic keratoses are precancerous lesions that result from chronic sun exposure and present as rough, scaly patches on sun-exposed skin. They are typically flat rather than raised and lack the pearly appearance seen in this image.

Squamous cell carcinoma would be an incorrect answer too. While Squamous cell carcinomas can present as raised lesions on sun-exposed skin, they more commonly appear as erythematous scaly plaques or ulcerated nodules rather than the pearly nodule shown here.

Finally, **Malignant melanoma** is unlikely given this presentation. Malignant melanomas generally present as pigmented lesions with asymmetry, irregular borders, colour variation and diameter greater than 6mm (the 'ABCD' criteria). This lesion does not exhibit these characteristics.

Question 33 of 82

A 30-year-old cleaner presents with a four-week history of recurrent vesicular lesions on her hands. She describes these lesions as initially appearing on the sides of her fingers before progressing to more generalised vesicles on her palms. They then break down and become intensely itchy, dry and cracked. She has tried using over the counter moisturisers but these have not helped.

She finds that the cleaning products she works with makes the rash worse and has been wearing rubber gloves to try and avoid this happening. She has also taken the past two weeks off work due to the cracked and inflamed skin on her hands.

She has no past medical history of note but does describe an allergy to nickel at the age of 14 around the time of her first ear piercings.

She currently takes the oral contraceptive pill.

On examination, you find linear 1-2mm vesicles along most of her digits with evidence of lichenification, cracking and inflamed skin. There is no evidence of any other skin rash elsewhere.

What is the most likely diagnosis?

- A. Digital Herpes Simplex
- B. Irritant contact dermatitis
- C. Pompholyx
- D. Pustular psoriasis
- E. Allergic contact dermatitis

C. Pompholyx

This is a classic history of pompholyx eczema. It typically affects the hands (80% of the time) or the feet (12%), or both and presents with small vesicles along the digits and palms/soles. These then de-roof causing intense itching and drying of the skin.

Pompholyx eczema has been associated with a nickel allergy.

The other answers can also present with vesicular eruptions on the hands but the clinical presentations are different.

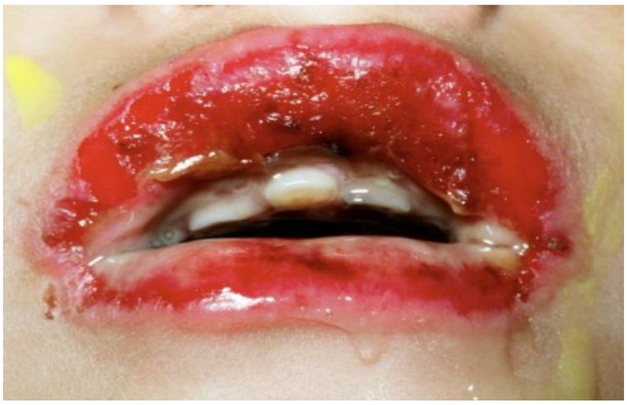
Herpetic whitlow would lead to a painful outcrop of vesicles and would be unlikely to cause lesions at multiple sites in an immunocompetent person.

Pustular psoriasis is an acute form of psoriasis which leads to widespread pustules with systemic upset.

Irritant and allergic contact dermatitis can present similarly to pompholyx eczema but are as a result of contact with an irritant or allergen. As a cleaner and with a nickel allergy she could be susceptible to both these conditions. However the fact that she has been wearing rubber gloves and has removed herself from potential contacts suggests that a specific chemical is unlikely to be the cause of her symptoms.

Question 34 of 82

A 27-year-old man presents as he is feeling generally unwell. Around 5 days ago he started having a sore throat associated with myalgia and lethargy. Yesterday he developed a fever and over the course of the past 24 hours a rash has appeared. His past medical history includes anxiety and atopic eczema. On examination he has a widespread erythematous rash, with many of the lesions having a 'target' appearance. Some of the lesions are becoming bullous. His pulse is 120/min and temperature 38.4º. He also has conjunctivitis and some ulceration and sloughing around the mouth:



What is the most likely diagnosis?

A. Eczema herpeticum
B. Pemphigus vulgaris
C. Erythema multiforme major
D. Impetigo
E. Necrotizing fasciitis

C. Erythema multiforme major

The correct answer in this case is **Erythema multiforme major**. Erythema multiforme is an acute, immune-mediated skin condition that typically presents with target-like lesions on the skin and mucous membranes. In this patient, the history of a sore throat followed by fever, myalgia, and lethargy along with the appearance of a widespread erythematous rash with target lesions and mucosal involvement (conjunctivitis and oral ulceration) strongly suggest erythema multiforme major. This condition can be triggered by infections (commonly herpes simplex virus or mycoplasma pneumoniae) or medications.

Eczema herpeticum is an incorrect option as it is a complication of atopic eczema where there is an extensive infection with herpes simplex virus. It presents with clusters of painful vesicles on an erythematous base that may rupture and form erosions. Although the patient has a history of atopic eczema, the presentation with target lesions and mucosal involvement makes erythema multiforme major more likely.

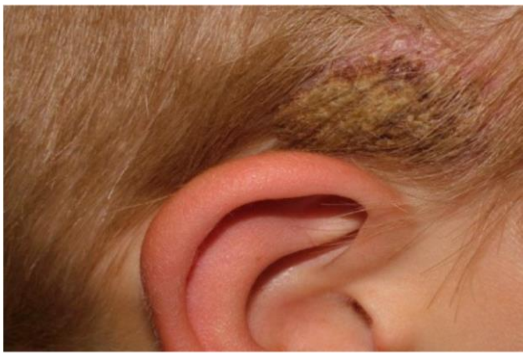
Pemphigus vulgaris is an autoimmune blistering disorder characterized by intraepidermal blisters involving both the skin and mucous membranes. The clinical presentation may include bullae that rupture easily, leaving painful erosions. However, the sudden onset of symptoms following a sore throat and fever in this case make it less likely than erythema multiforme major.

Impetigo is a superficial bacterial skin infection caused mainly by *Staphylococcus aureus* or *Streptococcus pyogenes*. It presents as honey-colored crusted lesions or small fluid-filled blisters that may rupture. The widespread rash involving multiple areas of the body along with systemic symptoms such as fever and myalgia make erythema multiforme major a more appropriate diagnosis.

Lastly, **necrotizing fasciitis** is a rapidly progressive bacterial infection involving the subcutaneous tissue and fascia. It presents with severe pain, erythema, and edema that rapidly progress to necrosis and systemic symptoms such as fever and hypotension. The presence of target lesions, mucosal involvement, and the absence of severe pain or rapid progression of symptoms in this case make erythema multiforme major more likely than necrotizing fasciitis.

Question 35 of 82

A 12-year-old boy is brought with a persistent 'scab' above his right ear. His mother reports that it has slowly developed over the past few weeks and initially started out as a patch of 'dandruff'. He is systemically well and their is no past medical history of note. The lesion is shown below:



What is the most likely diagnosis?

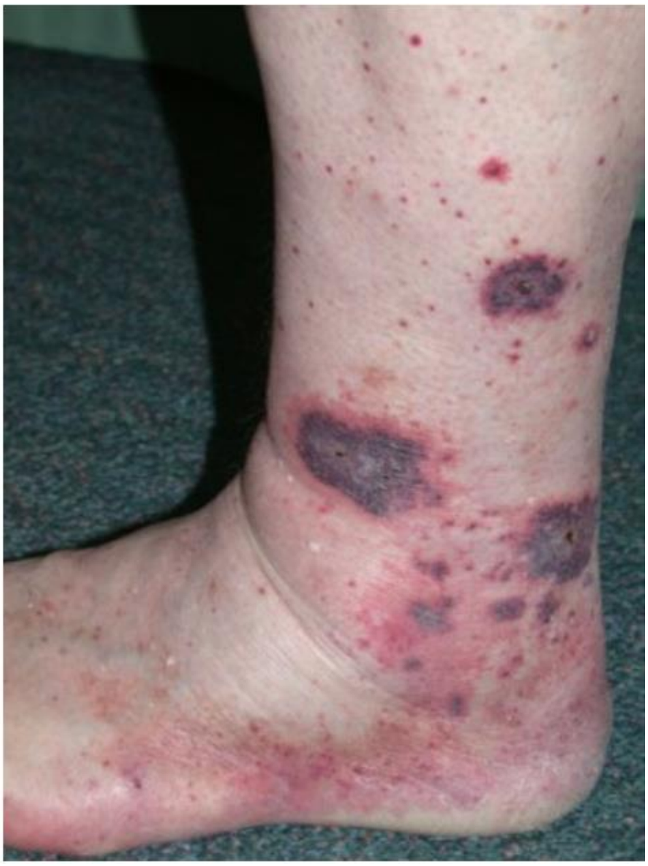
A. Seborrhoeic dermatitis
B. Trichotillomania
C. Impetigo
D. Scalp psoriasis
E. Kerion

E. Kerion

The kerion has developed as a result of an untreated tinea capitis infection. Systemic antifungals are usually required.

Question 36 of 82

A 64-year-old man presents with a 'rash' on his legs which has developed over the past few days:



He complains of feeling generally 'run-down' but review of systems is unremarkable.

What is the most likely underlying cause?

A. Vasculitis
B. Erythema multiforme
C. Necrotising fasciitis
D. Kaposi sarcoma
E. Venous eczema

A. Vasculitis

Kaposi sarcoma may cause similar skin changes to the larger lesions but would not typically cause petechiae.

Vasculitis is commonly limited to the skin and may be caused by infections, drugs, autoimmune disorders and malignancy.

Question 37 of 82

A 20-year-old woman presents to the medical clinic for review. She had been started by her GP on bisoprolol following an episode of palpitations and has subsequently developed a rash affecting her elbows, knees, navel genital area. It has been itchy but not painful, and she has not had any further episodes of palpitations. She has previously had eczema as a young child but this disappeared before the age of five. She has no allergies as far as she knows. On examination, she has a well defined erythematous rash of extensor surfaces of her elbows, knees, umbilicus and groin.

What is the most likely diagnosis?

A. Allergy to bisoprolol
B. Psoriasis
C. Topical dermatitis
D. Eczema
E. Cellulitis

B. Psoriasis

Flexural psoriasis is characterised by well defined, erythematous, non-painful rash

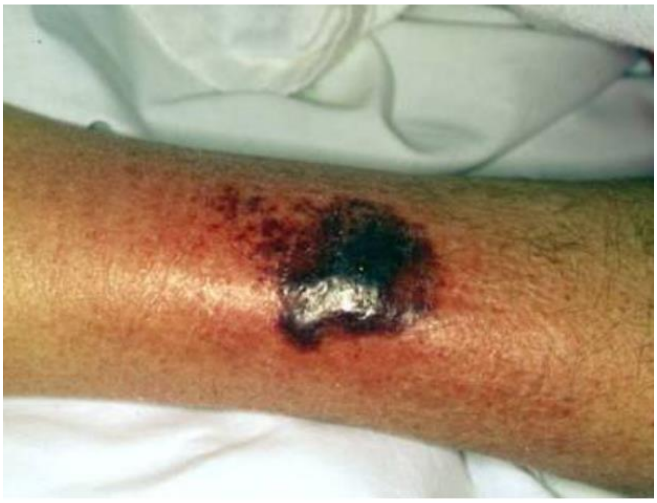
This is a patient who has developed a well-defined erythematous and pruritic rash of extensor surfaces following the use of a beta-blocker. The most likely explanation is psoriasis. In the progression of the condition, the typical plaques can form. For this patient, the first presentation has been triggered by beta-blockers; this is a known effect of beta-blockers which limits their use for people with psoriasis.

This is a very unlikely description of a drug allergy. The onset is faster with a systemic rash involving the trunk which is macular and poorly defined.

In topical dermatitis, there are defined areas of inflammation in areas of contact with an irritant. Eczema is also unlikely; eczema affects flexural rather than extensor surfaces.

Question 38 of 82

A 24-year-old man with a history of ulcerative colitis presents due to an expanding 'blood blister' on his lower leg:



What is the most likely diagnosis?

A. Pyogenic granuloma
B. Arterial leg ulcer
C. Necrotising fasciitis
D. Venous leg ulcer
E. Pyoderma gangrenosum

E. Pyoderma gangrenosum

The correct answer is **Pyoderma gangrenosum**. Pyoderma gangrenosum is a rare, inflammatory skin condition often associated with systemic diseases such as inflammatory bowel disease (IBD), which includes ulcerative colitis. The patient's history of ulcerative colitis and the appearance of the lesion - a rapidly enlarging, painful ulcer with a violaceous border - are characteristic features of pyoderma gangrenosum. It is also important to note that pyoderma gangrenosum can be precipitated by minor trauma or surgical procedures.

Pyogenic granuloma, although it can appear similar to pyoderma gangrenosum, typically presents as a small, red, vascular lesion that bleeds easily. They are usually solitary and occur after minor trauma. While they can occur anywhere on the body, they most commonly appear on the hands and face.

An **Arterial leg ulcer** would be an unusual presentation in such a young patient without risk factors for peripheral arterial disease (PAD). These ulcers typically present on the lateral aspect of the ankle or foot and are characterised by severe pain, especially at night or with elevation of the limb. Additionally, there would often be signs of peripheral arterial disease such as absence of pulses or claudication.

Necrotising fasciitis is a life-threatening soft tissue infection causing rapidly progressive skin necrosis and systemic toxicity. Although it could theoretically present similarly to this case, it would likely have been accompanied by severe systemic symptoms like fever and hypotension which are not mentioned in the scenario.

A **Venous leg ulcer** tends to occur in older patients with a history of chronic venous insufficiency or deep vein thrombosis (DVT). The ulcers usually develop around the medial malleolus and are often accompanied by other signs of chronic venous insufficiency such as varicose veins, oedema and skin changes like hyperpigmentation or lipodermatosclerosis.

Question 39 of 82

A 20-year-old man is reviewed in dermatology clinic. He has been suffering from severe worsening of his eczema symptoms. He normally manages with emollients and topical steroids of moderate to severe strength. Recently he has had a severe flare, which has not even responded to a trial of oral prednisolone over one week. He is suffering from severe pruritus and irritation of his skin. On examination, his flexural surfaces on his arms and legs are inflamed and show evidence of scratch marks.

What non-steroidal option may help control his flare?

A. Alitretinoin
B. Topical tacrolimus
C. Oral tacrolimus
D. IV tacrolimus
E. Topical salicylic acid

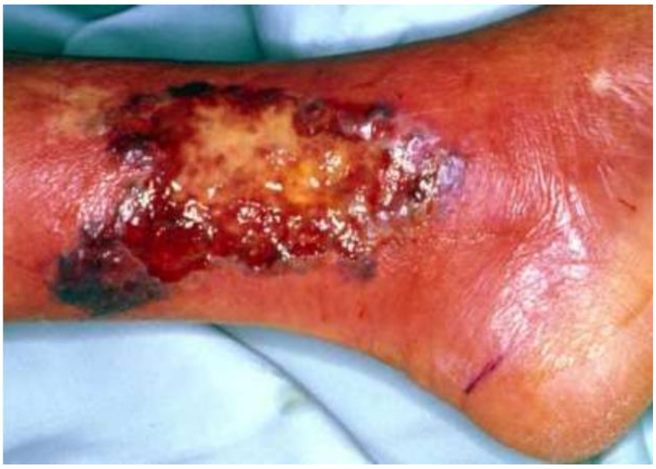
B. Topical tacrolimus

Calcineurin inhibitors are an alternative to topical steroids in eczema

This patient has a severe flare of eczema not responding to emollients and strong steroid therapy as evidence by failure of response to prednisolone. Calcineurin inhibitors, such as tacrolimus, can be used by dermatologists for this. Topical tacrolimus is preferred, as oral and IV tacrolimus are used to prevent and treat organ rejection rather than to treat eczema. Salicylic acid is used for scales in psoriasis, whilst alitretinoin would only be used for severe refractory hand eczema.

Question 40 of 82

The lesion below started as a small red papule which grew in size before starting to ulcerate:



Which one of the following conditions is most associated with this skin condition?

A. Rheumatoid arthritis
B. Sarcoidosis
C. Primary herpes simplex virus infection
D. Tuberculosis
E. Thyrotoxicosis

A. Rheumatoid arthritis

Question 41 of 82

A 50-year-old male patient with a history of severe psoriasis has previously tried treatment with topical corticosteroids, vitamin D analogue therapy and phototherapy with little effect.

On examination he has multiple well circumscribed indurated erythematous plaques with overlying white scale over the majority of the trunk and forearms.

Blood tests reveal:

Hb	135 g/l
Platelets	405 * 10 /l9
WBC	8.2 * 10 /l9
Bilirubin	12 µmol/l
ALP	70 u/l
ALT	27 u/l
Albumin	35 g/l
Na +	140 mmol/l
K +	5.1 mmol/l
Urea	8.2 mmol/l

Creatinine	112 µmol/l

What is the most appropriate therapy?

A. Infliximab
B. Adalimumab
C. Ciclosporin
D. Methotrexate
E. Etanercept

D. Methotrexate

NICE CG154 addresses the approach to systemic therapy for psoriasis and is outlined below:

Offer systemic non-biological therapy to people with any type of psoriasis if:

- It cannot be controlled with topical therapy and
- It has a significant impact on physical, psychological or social wellbeing and one or more of the following apply:

psoriasis is extensive (for example, more than 10% of body surface area affected or a PASI score of more than 10) or psoriasis is localised and associated with significant functional impairment and/or high levels of distress (for example severe nail disease or involvement at high-impact sites) or phototherapy has been ineffective, cannot be used or has resulted in rapid relapse (rapid relapse is defined as greater than 50% of baseline disease severity within 3 months).

Choice of drugs:

- Offer methotrexate as the first choice of systemic agent for people with psoriasis who fulfil the criteria for systemic therapy (above)
- Offer ciclosporin as the first choice of systemic agent for people who fulfil the criteria for systemic therapy (above) and who:

need rapid or short-term disease control (for example a psoriasis flare) or have palmoplantar pustulosis or

are considering conception (both men and women) and systemic therapy cannot be avoided.

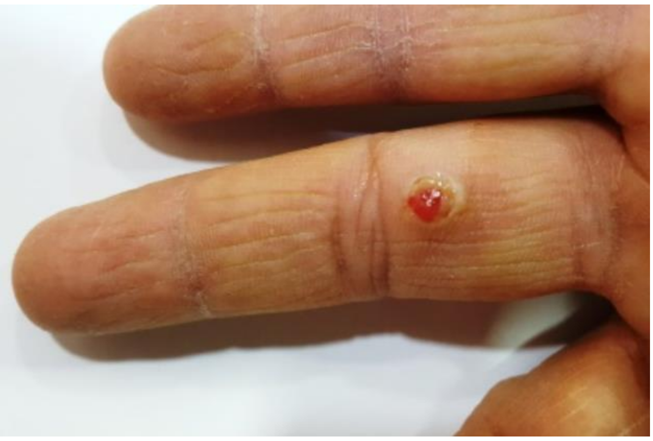
- Consider changing from methotrexate to ciclosporin (or vice-versa) when response to the first-choice systemic treatment is inadequate.
- Consider acitretin for adults, and in exceptional cases only for children and young people, in the following circumstances:

if methotrexate and ciclosporin are not appropriate or have failed or for people with pustular forms of psoriasis.

Question 42 of 82

You are reviewing a 33-year-old woman with a lesion on her finger. The lesion has been present for 6 weeks, initially starting small before quickly growing in size. There is no history of trauma. There is no past medical history and her only regular medication is folic acid. She is 28 weeks pregnant with her second child and has had no complications during her pregnancy.

An image of the lesion is shown below.



What is the most appropriate management for the lesion?

A. Cauterisation
B. Cryotherapy
C. Curettage
D. Excision
E. Watch and wait

E. Watch and wait

The image shows a well-defined, ulcerated papule alongside a history of rapid growth, in keeping with a pyogenic granuloma. These are found to be more common after trauma, in pregnancy, and in young adults. Given that the above scenario does not allude to any trauma, the likely cause for this lesion is pregnancy.

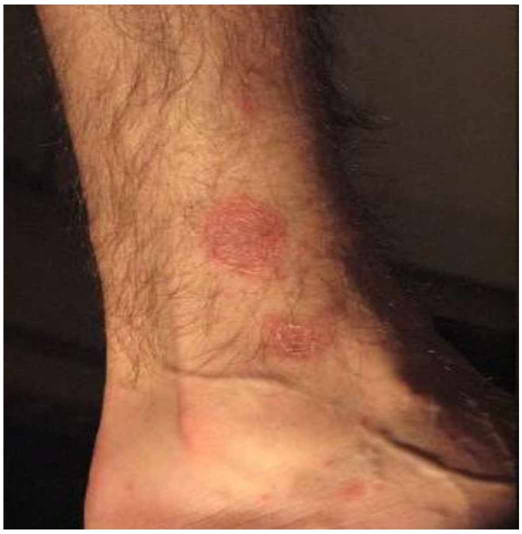
Traumatic pyogenic granulomas can be treated with cauterisation, cryotherapy, curettage, or excision.

The above scenario is a non-traumatic pyogenic granuloma likely secondary to pregnancy and so the most appropriate initial step in management is 'watch and wait' as lesions often self-resolve in pregnancy.

Question 43 of 82

A 32-year-old man presents to the dermatology clinic with a rash on his lower legs. He reports that an initial lesion appeared 2 weeks ago, followed by further smaller lesions over his body. There is no history of itch or pain and he denies having a fever. His past medical history includes anxiety for which he takes propranolol and he has no known allergies.

His rash is examined, as shown below:



What is the recommended management for this patient?

A. Reassurance and discharge
B. Topical 1% hydrocortisone
C. Topical fusidic acid
D. Topical ketoconazole
E. UVB phototherapy

A. Reassurance and discharge

This patient has multiple small, oval-shaped, red plaques that are located on the lower limbs.

Looking closely at these lesions, some appear to have a fine peripheral scale. This patient has a diagnosis of pityriasis rosea, a self-limiting rash that is characterised by a large round 'herald patch', usually found on the chest, abdomen, or back. It mostly affects children and young adults and is thought to be precipitated by bacterial and viral infections (e.g. human herpes virus 6 and 7) although this has not definitely been proved yet.

Reassurance and discharge is correct. Pityriasis rosea is a self-limiting rash and treatment is not usually required.

Topical 1% hydrocortisone is incorrect. Topical corticosteroids are usually used in the treatment of eczematous diseases that typically present as a dry, itchy and scaly rash which is not seen here. They can also be used in the management of localised chronic plaque psoriasis. However, it plays no role in the management of pityriasis rosea.

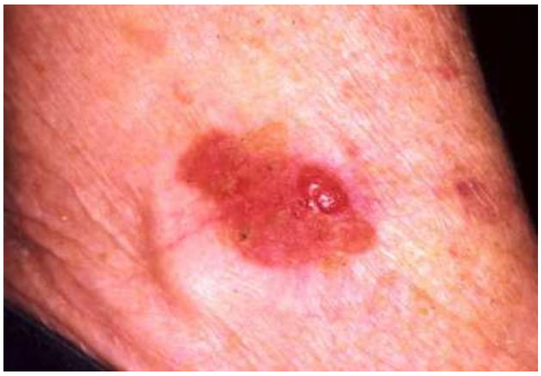
Topical fusidic acid is incorrect. This is used in treating superficial bacterial infections including insect bites, stings and impetigo. However, there is no surrounding erythema or pustular discharge to suggest that this rash is bacterial in nature.

Topical ketoconazole is incorrect. Topical ketoconazole is used in the treatment of fungal infections such as pityriasis versicolor. The rash is mostly seen on the trunk, neck and upper limbs. The patches can vary in colour from pink, brown to pale (typically paler than the surrounding skin). They are usually asymptomatic but can occasionally be itchy. Heat and moisture are risk factors for developing the rash.

UVB phototherapy is incorrect. This is used in the management of chronic plaque psoriasis and guttate psoriasis that cannot be controlled with initial topical management. Importantly, this patient is taking propranolol which is known to exacerbate psoriasis. Psoriasis has a similar appearance to pityriasis rosea. However, psoriasis has a coarser silvery scale with surrounding lichenification. However, the history of an isolated herald patch before the onset of the remaining rash is suggestive for pityriasis rosea for which UVB phototherapy is not a treatment.

Question 44 of 82

A 59-year-old man presents with a new skin lesion which has developed over the past few months:



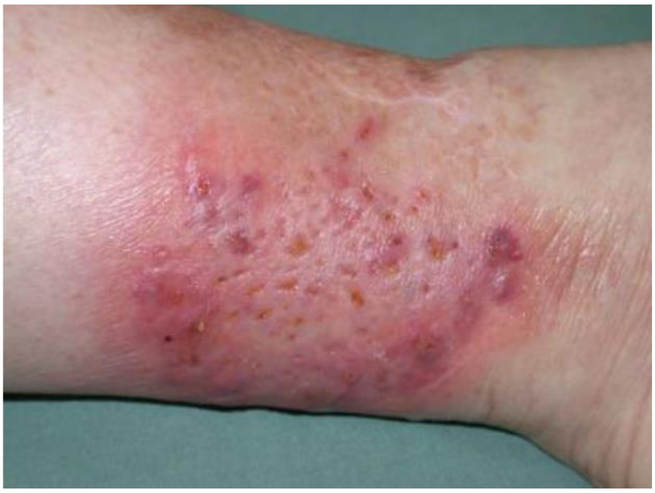
You plan to refer the patient to dermatology. What is the most likely diagnosis?

A. Malignant melanoma
B. Actinic keratosis
C. Bowen's disease
D. Basal cell carcinoma
E. Seborrhoeic keratosis

C. Bowen's disease

Question 45 of 82

You review a 31-year-old woman who has had Crohn's disease for the past 12 years. She is currently on infliximab therapy.



What is the most likely diagnosis?

A. Pyoderma gangrenosum
B. Acute febrile neutrophilic dermatosis
C. Squamous cell carcinoma
D. Pyogenic granuloma
E. Behcet's disease

A. Pyoderma gangrenosum

The correct answer is **Pyoderma gangrenosum**. Pyoderma gangrenosum is a neutrophilic dermatosis that commonly occurs in patients with inflammatory bowel disease, such as Crohn's. It presents as painful, necrotic ulcers often found on the lower extremities. The pathophysiology involves an abnormal immune response leading to neutrophil infiltration of the skin, resulting in ulceration. Management typically involves systemic corticosteroids and immunosuppressants like infliximab.

Acute febrile neutrophilic dermatosis, also known as Sweet's syndrome, is incorrect. Although it is also a neutrophilic dermatosis like pyoderma gangrenosum, it usually presents with fever and elevated inflammatory markers alongside the abrupt onset of tender erythematous skin lesions. It can be associated with malignancies and infections but less commonly with Crohn's disease.

Squamous cell carcinoma is not the most likely diagnosis in this case. While patients with chronic inflammatory conditions may have an increased risk for squamous cell carcinoma over time, the acute presentation of painful necrotic ulcers is more indicative of pyoderma gangrenosum.

The option **Pyogenic granuloma** is incorrect as well. Pyogenic granulomas are benign vascular lesions that present as rapidly growing red papules or nodules, often after minor trauma or during pregnancy. They do not typically present with ulceration and are not associated with Crohn's disease.

Finally, **Behcet's disease** could potentially cause similar skin lesions due to its vasculitis component; however, other symptoms such as oral aphthous ulcers, genital ulcers and ocular inflammation would likely be present too. Furthermore, Behcet's disease has no established association with Crohn's disease, unlike pyoderma gangrenosum.

Question 46 of 82

A 21-year-old woman presents to the emergency department with an itchy rash. It developed over the course of the day which she spent at the beach with friends.

She complains of an itching and burning sensation on her upper arms, trunk and thighs.

On examination there are crops of 3mm pink papules over the symptomatic areas, sparing her face and her hands.

What is the most likely diagnosis?

A. Scabies
B. Systemic lupus erythematosus
C. Xeroderma pigmentosum
D. Photoallergic contact dermatitis
E. Polymorphic light eruption

E. Polymorphic light eruption

Photoallergic contact dermatitis is a toxic or allergic reaction that occurs when certain chemicals are applied to the skin and subsequently exposed to the sun. It is unlikely to spare the hands and face.

Xeroderma pigmentosum is a rare hereditary defect of the enzyme system that repairs DNA after damage from ultraviolet rays, resulting in extreme sensitivity to sunlight and a tendency to develop skin cancer. It typically presents in youth with multiple basal cell carcinomas and other skin malignancies.

The photosensitive rash of systemic lupus erythematosus classically includes the the butterflyshaped rash that appears over the bridge of the nose and both cheeks.

The rash associated with scabies often appears as erythematous papules on the trunk and limbs with vesicles on palms and soles and irregular tracks in the web spaces between the fingers.

Question 47 of 82

A 27-year-old man was seen in the dermatology clinic with an itchy, vesicular rash over buttocks and proximal forearms. He was otherwise well.

A subsequent skin biopsy and direct immunofluorescence demonstrate granular IgA at the dermal-epidermal junction. Given the most likely diagnosis, he was started on dapsone and given specific diary advice.

Six weeks later he complains of worsening fatigue. What is the most important investigation?

A. Full blood count
B. Echocardiogram
C. ECG
D. Urine protein:creatinine ratio
E. Creatine kinase

A. Full blood count

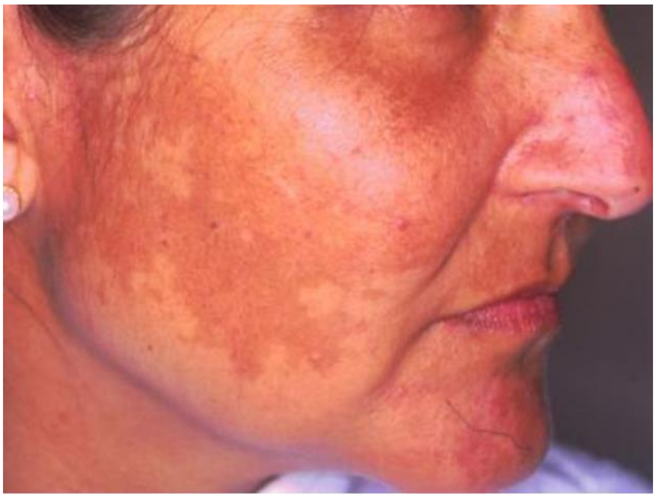
The patient has dermatitis herpetiformis - an inflammatory cutaneous condition closely associated with gluten sensitive enteropathy (coeliac disease).

Dapsone is an antibacterial agent that can provide temporary symptomatic relief as a gluten-free diet is established. The main side effect is that of haemolytic anaemia - so regular full blood counts are essential. Other side effects include peripheral neuropathy and rarely agranulocytosis.

The remaining options are clearly not correct if the candidate is aware of the side effects of dapsone - a MRCP favourite - as is the presentation of dermatitis herpetiformis without overt diarrhoea.

Question 48 of 82

This woman complains of a 'rash' on her cheeks. She has recently started taking hormone replacement therapy for menopausal symptoms.



What is the most likely diagnosis?

A. Vitiligo
B. Seborrhoeic dermatitis
C. Acne rosacea
D. Melasma
E. Systemic lupus erythematosus

D. Melasma

The correct answer is **Melasma**. Melasma, also known as chloasma, is a common skin condition of adults in which light to dark brown or greyish pigmentation develops, mainly on the face. The image shows hyperpigmented patches on the cheeks, a typical presentation of melasma. It is more common in women and often related to hormonal changes such as pregnancy or hormone treatments like hormone replacement therapy (HRT).

Vitiligo is incorrectly identified here because it presents with depigmented patches rather than hyperpigmented. Vitiligo is an autoimmune condition that causes loss of melanocytes leading to white patches on the skin.

Seborrhoeic dermatitis would be incorrect as well. Seborrhoeic dermatitis typically presents with a red, scaly, itchy rash usually located on the scalp, eyebrows, nasolabial folds and chest. It does not cause hyperpigmentation.

The diagnosis of **Acne rosacea** would be unlikely given the absence of papules, pustules or rhinophyma which are characteristic for this condition. Rosacea generally affects middle-aged women and presents with flushing and persistent erythema along with telangiectasia.

Lastly, **Systemic lupus erythematosus (SLE)** can indeed present with a facial rash known as malar rash or 'butterfly' rash but this tends to be erythematous and sparing nasolabial folds, unlike melasma which often involves these areas. Additionally, SLE usually comes with other systemic symptoms like joint pain and fatigue which were not mentioned in this scenario.

Question 49 of 82

A 74-year-old male presents to the acute medical take with left sided chest pain, worse on inspiration. On examination, you note his nails are yellow and clubbed with bilateral non-pitting oedema. Examination of his chest reveals reduced air entry of his left lung to mid zone, dull to percussion, with no wheeze.

He has a known past medical history of bronchiectasis diagnosed 2 years ago, for which he has been admitted 3 times in the past 5 months. He reports weight loss of about 8kg in the past 9 months. He last underwent an echocardiogram 2 years ago, during which a left ventricular ejection fraction of 56% was demonstrated.

A chest radiograph demonstrates a large left pleural effusion and a mark has been made by ultrasound for a pleural tap.

Pleural fluid analysis results are as follows:

pH	7.42
Protein	40 g/L (serum 58)
LDH	130 IU/L (serum 181)
Glucose	3.5 mmol/l (serum 6.2)
Amylase	22 u/l (serum 32)
Triglycerides	1.90 mmol/L (serum 2.2)
Cytology	awaited
AFB	awaited

What is the unifying diagnosis?

A. Parapneumonic effusion
B. Yellow nail syndrome
C. Malignant pleural effusion
D. Oesophageal rupture
E. Congestive cardiac failure

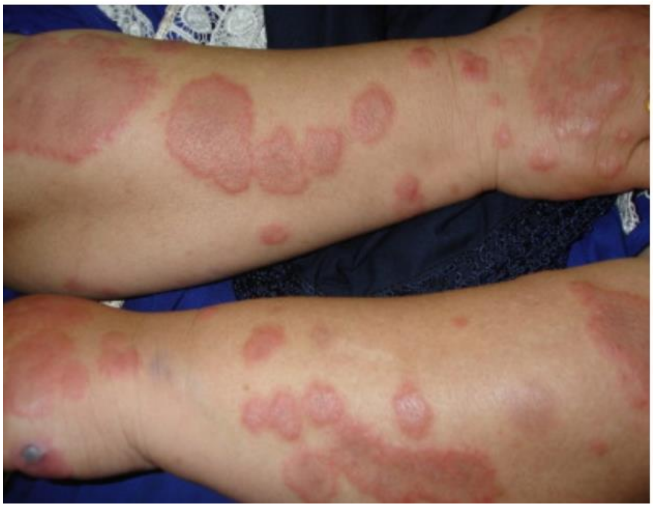
B. Yellow nail syndrome

Diagnosis here is dependent on subtle details in the history and pleural fluid analysis: the patient has bronchiectasis, pleural effusion (exudate), lymphoedema (not pitting oedema), chylothorax (note the significantly raised triglycerides in the pleural fluid) and of course, yellow nails! The patient has yellow nail syndrome, characterised by nail dystrophy or discolouration, lymphoedema and pleural effusions. A parapneumonic and malignant effusion are reasonable differentials but do not explain the triglycerides. Oesophageal rupture classically increases pleural amylase. Lastly, congestive cardiac failure classically produces a transudate.

Question 50 of 82

A 34-year-old woman presents to the general practitioner with a 2-day history of an erythematous rash over her upper limbs. She describes waking up yesterday morning with multiple lesions over her arms. The rash is not itchy or painful and has not resolved despite regular emollient use. She has no past medical history of note.

On examination, she appears well. There is a crusted cold sore on her top lip. Her chest is clear and her heart sounds are normal. She has a soft abdomen. Her rash is examined and appears as shown below:



What is the most likely diagnosis?

A. Eosinophilic granulomatosis with polyangiitis
B. Erythema marginatum
C. Erythema multiforme
D. Plaque psoriasis
E. Urticaria

C. Erythema multiforme

This patient has multiple well-demarcated, round target lesions with an outermost erythematous ring and paler centre in keeping with a diagnosis of erythema multiforme. Erythema multiforme is an acute, self-limiting skin eruption most commonly secondary to an infective trigger (e.g. herpes simplex virus or *Mycoplasma pneumoniae*). The rash appears abruptly and affects the upper limbs most often before spreading down towards the trunk. One of this patient's examination findings included a cold sore, suggesting a recent infection with herpes simplex virus, the likely trigger of her rash.

Urticaria presents as an itchy rash with multiple weals over the skin. They usually appear pale or light pink and may be surrounded by a red flare. The most common trigger is an allergic reaction, although viral infections may also precipitate urticaria. Urticaria does not cause the target lesions seen in the above image, making it an unlikely diagnosis.

Eosinophilic granulomatosis with polyangiitis is a small-vessel vasculitis with symptoms including asthma, allergic rhinitis and constitutional symptoms of weight loss, fevers and night sweats. Skin involvement can manifest in up to 50% of patients and include blisters, petechiae and ulceration. This is not seen in the image above. Furthermore, there is nothing in this patient's clinical history to suggest an underlying diagnosis of vasculitis.

Erythema marginatum is a manifestation of acute rheumatic fever. It is most commonly seen in children and is rare in adults. It presents as annular erythematous macules or papules that spread outwards with central clearance. The lesions are not itchy or painful and may be asymptomatic.

Plaque psoriasis is a chronic inflammatory skin condition that gives rise to well-defined red plaques with an overlying silver scale. Common features include itch and painful fissures. The rash in the image does not show the presence of erythematous plaques. Furthermore, this patient's rash has had an acute onset which is atypical of psoriasis that has a much slower onset.

Question 51 of 82

A 72-year-old gentleman presents with a peeling and erythematous rash on both of his hands. He has noticed slight tingling in the same area but denies pain. He has a past medical history of colorectal cancer which was managed surgically with an anterior resection. He developed local recurrence of his cancer five months post-operatively. He is now on palliative chemotherapy with capecitabine and oxaliplatin every two weeks in order to control progression. His last treatment was one week ago. He has a history of type two diabetes mellitus, depression and has had a cholecystectomy when he was younger. On examination he appears frail and cachectic, but systemically well. His hands are shown to have patchy palmar erythema with mild desquamation. A similar rash is present on the soles of his feet.

Observations:

Saturations	97%
Respiratory rate	17/min
Blood pressure	132/73mmHg
Heart rate	71/min
Temperature	37.3°C

What is the most likely diagnosis?

A. Erythromelalgia
B. Palmoplantar psoriasis
C. Palmar-plantar erythrodysesthesia
D. Paraneoplastic syndrome
E. Cellulitis

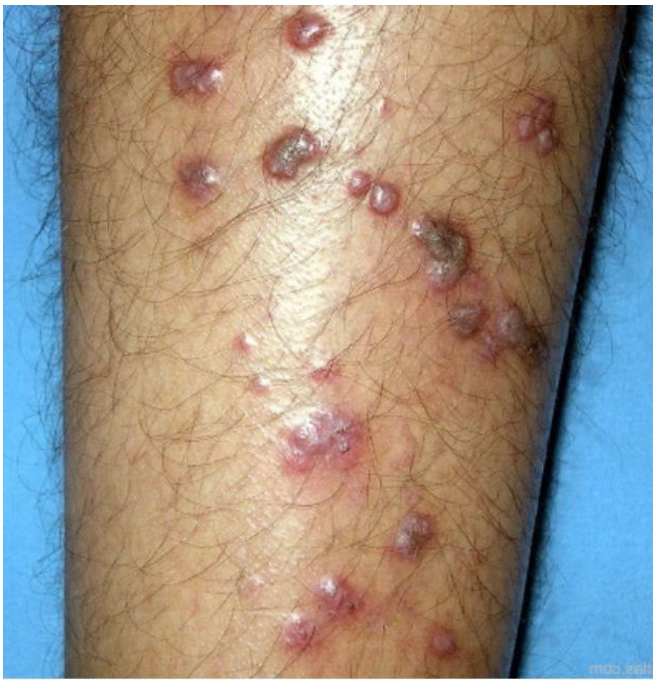
C. Palmar-plantar erythrodysesthesia

The answer is palmar-planter erythrodysesthesia. The distribution of a dysesthesic erythematous rash with desquamation of the palms and soles is a classic description of palmar-plantar erythrodysesthesia during chemotherapy. Cellulitis, paraneoplastic syndromes, and erythromelalgia do not fit this distribution, and palmoplantar psoriasis is associated with itching.

Question 52 of 82

A 53-year-old woman is seen in the dermatology clinic with a 4-week history of an itchy rash on her lower legs. She has tried various antihistamines and a cold compress, but these have not relieved her symptoms. There is no history of pain or bleeding and she is systemically well. Her past medical history includes chronic hepatitis C infection. She takes no regular medication.

On examination, her rash appears as shown below.



What is the most likely diagnosis?

A. Bullous pemphigus
B. Lichen planus
C. Lichen sclerosus
D. Molluscum contagiosum
E. Pemphigus vulgaris

B. Lichen planus

The photograph above shows multiple round, well-circumscribed, raised purple lesions. On closer inspection, subtle white markings can be visualised on top of the lesions, otherwise known as Wickham striae. The clinical history and appearance of the rash are in keeping with a diagnosis of lichen planus. Lichen planus is typically an itchy rash that appears as flat-topped plaques. The colour of the rash is dependent on skin tone. However, in fairer skin tones, the rash is more likely to look purple in colour. Rarely, nail involvement can be seen which includes thinning of the nail bed to onycholysis. Infection with hepatitis C is a risk factor for developing lichen planus. The treatment of choice is potent topical corticosteroids.

Bullous pemphigus is an autoimmune blistering disease commonly seen in those over 60 years. Typically a non-specific rash is seen for several weeks before the development of tense blisters between the dermis and epidermis. Any part of the skin can be affected, although the flexor aspects are more commonly affected.

Lichen sclerosus is most commonly diagnosed in women over the age of 50. It is a chronic dermatological disorder of the genital and peri-anal skin. It is possible for lichen sclerosus to overlap with other skin conditions such as lichen planus or psoriasis. However, the absence of vulval symptoms in this patient makes the diagnosis less likely.

Molluscum contagiosum is a viral condition that is commonly seen in childhood or the immunocompromised. It presents as clusters of round papules with an umbilicated centre and each papule contain a thick creamy material. There is nothing in this patient's history to suggest immunosuppression and therefore molluscum contagiosum is unlikely.

Pemphigus vulgaris is an autoimmune disease characterised by painful blisters and erosions on the skin. Mucous membranes are also involved and are usually the first site to be affected during the onset of the disease, followed by epidermal involvement. It typically arises in those aged between 30-60 years. Unlike bullous pemphigus, pemphigus vulgaris presents as flaccid blisters which easily rupture to cause painful erosions. These are not seen in the image above making the diagnosis less likely.

Question 53 of 82

A 32-year-old man attends his GP with pruritus of the genital area. Additionally, he reports painful intercourse with his long-term partner. Examination reveals a whitish scarred meatus with a white plaque on phimotic foreskin.

What is the most likely diagnosis?

A. Candidiasis
B. Vitiligo
C. Lichen planus
D. Lichen sclerosus
E. Erythroplasia of Queyrat

D. Lichen sclerosus

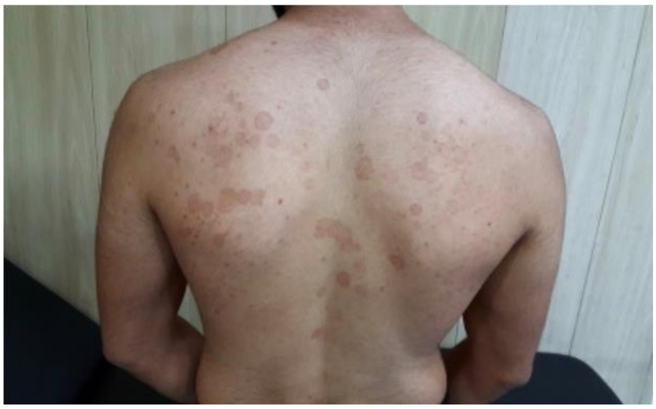
Candidiasis will typically cause erythema on the meatus and discharge. Vitiligo would not be expected to cause painful intercourse or result in phimosis.

Penile lichen planus usually presents with purple papules in a ring around the glans. Comparatively, lichen sclerosus usually affects the glans and foreskin, resulting in white papules or plaques. It can cause phimosis.

Erythroplasia of Queyrat, also known as an in-situ squamous cell carcinoma of the penis, is a penile intraepithelial neoplasia. It typically occurs in men above 50 years of age, with symptoms including erythema, pain, bleeding and ulcers.

Question 54 of 82

A 21-year-old man presents to the dermatology clinic with a 10-day history of a widespread rash over his torso, neck and upper limbs. He reports that the rash developed during a backpacking holiday in South East Asia. There is no history of fever, pain or itch and other than a mild gastrointestinal illness whilst abroad, has been well in himself. He thinks he may have had a similar rash before last summer on a family holiday to Portugal. His rash is examined, as shown below:



What is the most likely diagnosis?

A. Guttate psoriasis
B. Pityriasis rosea
C. Pityriasis versicolor
D. Tinea corporis
E. Urticaria

C. Pityriasis versicolor

This patient has a diffuse hyperpigmented rash consisting of multiple round lesions mostly over the trunk. This is consistent with the diagnosis of **pityriasis versicolor**, a superficial cutaneous fungal infection caused by *Malassezia furfur*. The patches can vary in colour from pink, brown to pale (typically paler than the surrounding skin) and therefore explains the name 'versicolor'. The upper limbs and neck are also commonly affected. The rash is usually asymptomatic but can occasionally be itchy. Heat and moisture are risk factors for developing the rash, and the exposure to the heat seems to coincide with this patient's rash.

Guttate psoriasis is incorrect. Guttate psoriasis is a subtype of psoriasis that typically affects children and young adults following infection with *Streptococcus sp.*. The rash presents acutely with multiple small scaly and erythematous patches of skin appearing over the trunk and limbs.

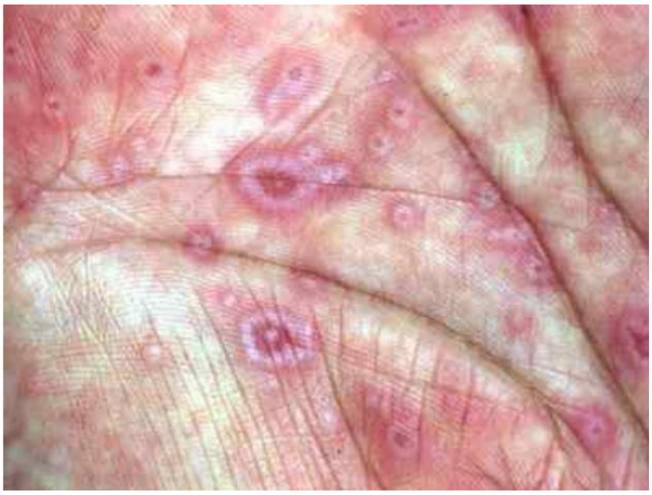
Pityriasis rosea is incorrect. Pityriasis rosea is a self-limiting rash, characterised by a large round 'herald patch', usually found on the chest, abdomen, or back. It mostly affects children and young adults and is thought to be precipitated by bacterial and viral infections (e.g. human herpes virus 6 and 7).

Tinea corporis is incorrect. Tinea corporis is a superficial fungal infection of the skin that can affect any part of the body. However, the infection often spares the hands, feet, scalp, face and groin. The lesions associated with tinea corporis are characteristic ring-shaped lesions that are not seen here.

Urticaria is incorrect. Urticaria presents as an erythematous, raised rash that is often described as being intensely itchy. This is not in keeping with this patient's presentation.

Question 55 of 82

A 55-year-old man develops a rash two days after starting a new medication. The rash is mildly pruritic and mainly affects the arms, torso and neck. The palms of his hand are shown below:



Which one of the following drugs is most likely to have been started?

A. Levetiracetam
B. Olanzapine
C. Carbamazepine
D. Fluoxetine
E. Diazepam

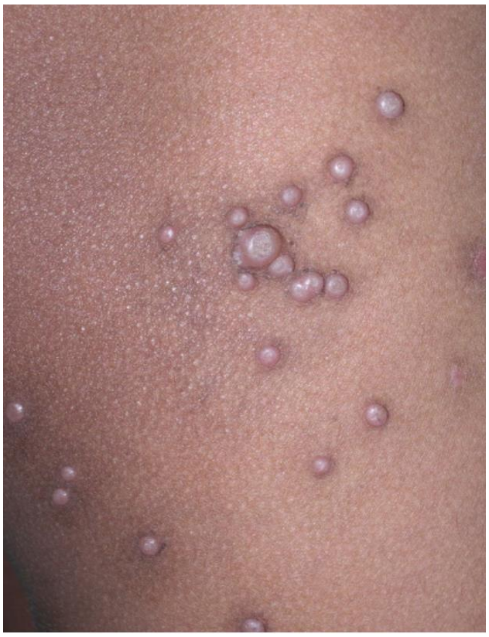
C. Carbamazepine

This patient has developed erythema multiforme which is a known complication of carbamazepine use.

Question 56 of 82

An 8-year-old boy presents with his mother, who is concerned about a rash that has developed on her son's torso. It has gradually developed over the past few weeks. He is otherwise healthy and well, with no significant medical history, nor family history.

On examination, the rash appears as shown below:



Given the likely diagnosis, what is the most appropriate management?

A. Cryotherapy
B. Piercing
C. Reassurance
D. Topical fusidic acid
E. Topical hydrocortisone

C. Reassurance

The image above depicts molluscum contagiosum in a child. The clustered lesions have the typical appearance of pearly papules with umbilicated centres. This is an incredibly common infection that is highly contagious and often spreads between children in close contact. The mother should be reassured that it is a self-limiting condition, and symptoms usually resolve within 18 months - and so reassurance is the most appropriate option.

Cryotherapy may be an option used in older children or adults, if lesions are concerning, for instance catching on clothing or in a cosmetically sensitive place. However, this is a young child, and the lesions and described as being limited to the torso.

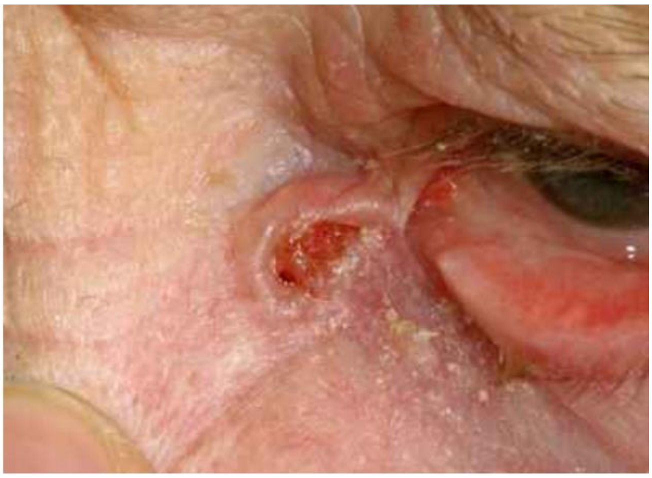
Piercing with an orange stick, or squeezing with fingernails, may be tried following a bath. This should, however, be limited to a few lesions at one time. This is not generally recommended if lesions are not problematic - in a straightforward case such as this, reassurance should be given and the lesions left alone.

Topical fusidic acid would be used if there were any signs of secondary infection - oedema, erythema, or crusting. The photo above does not demonstrate any of these signs and so an antibiotic is not necessary.

If there are any signs of inflammation, or any itching, a mild topical steroid such as hydrocortisone may be used. Again, in this photo, the lesions do not appear inflamed, nor is any description given of problematic itching.

Question 57 of 82

Please look at the skin lesion shown below:



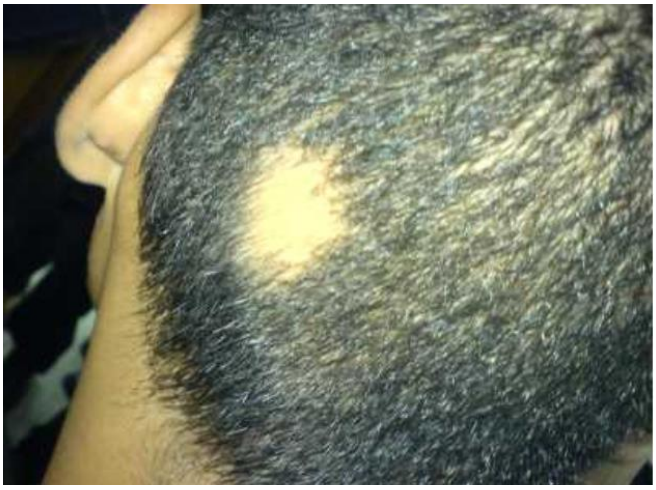
What is the most likely diagnosis?

A. Infected chalazion
B. Actinic keratosis
C. Indurated dacryocystitis
D. Keratoacanthoma
E. Basal cell carcinoma

E. Basal cell carcinoma

Question 58 of 82

A 23-year-old man presents as he is concerned about recent hair loss. Examination reveals the following:



What is the most likely diagnosis?

A. Telogen effluvium
B. Alopecia areata
C. Tinea capitis
D. Male-pattern baldness
E. Discoid lupus erythematous

B. Alopecia areata

Question 59 of 82

A 73-year-old woman attended dermatology clinic for review of a skin lesion on her right cheek. The patient had recently returned to the United Kingdom having been resident in southern Spain for the previous 20 years. She described the development of a lump just under her right eye over approximately the past six months. She also recalled having a area of slightly dry and sore skin in a similar location prior to the emergence of the lesion, although she had not sought medical attention for this. The patient denied having any symptoms from the lesion, although was very concerned and upset about the unsightly appearance it caused.

The patient's medical history included rheumatoid arthritis, type 2 diabetes mellitus, hypertension and intermittent episodes of gout. She had no previous dermatological condition. Her regular medications were methotrexate 15 mg weekly, metformin 1000 mg twice daily, ramipril 2.5 mg daily and naproxen 500 mg as required. She denied any previous treatments for cancer.

Examination of the patient demonstrated an approximately spherical nodule 11 mm in diameter arising from the patient's cheek 15 mm inferior to her eyelid. Crusting was noted on the surface of the nodule, but there was no hyperkeratosis, induration or ulceration.

A biopsy of the lesion is taken during clinic assessment with histology demonstrating squamous cell carcinoma.

What is the appropriate technique for treatment of the patient's lesion?

A. Electrodessiccation
B. Wide local excision with 4 mm margin of normal skin
C. Mohs micrographic surgery
D. Wide local excision with 6 mm margin of normal skin
E. Radiotherapy

C. Mohs micrographic surgery

The patient has a facial cutaneous squamous cell carcinoma in a functionally and cosmetically important region. Her history of immunosuppression with methotrexate could also lead to the tumour being considered high risk, although the increased risk of squamous cell carcinoma is most well characterised in those individuals highly immunosuppressed to prevent transplant rejection.

Mohs micrographic surgery is a specialised technique reserved for use in high risk tumours or those in functionally or cosmetically important areas. The technique involves histological examination of the tumour during the procedure to ensure adequate margins and complete tumour clearance. The technique provides a high cure rate and maximum tissue conservation.

In less sensitive areas of the body, wide local excision is the treatment of choice. Low risk tumours with diameter < 2 mm require margins of 4 mm, with 6 mm margins being required in high-risk or larger tumours.

Radiotherapy is an alternative treatment option for individuals not suitable for surgery but has a higher recurrence rate than surgical excision. Electrodessiccation (curettage and cautery) is occasionally used for low risk cutaneous squamous cell carcinomas on the extremities or trunk.

Question 60 of 82

A 67-year-old man presents with a five week history of skin rash. He states that the rash erupted suddenly and progressed over five weeks.

His past medical history includes type 2 diabetes mellitus. He is obese with a BMI of 39kg/m² and drinks alcohol to excess. An ultrasound scan four years ago confirmed cirrhosis.

On examination, there are over 60 raised, oval lesions with tan pigmentation over his back and chest, ranging from 5mm to 25mm diameter, and have a warty texture and stuck-on appearance.

What would be the most appropriate next step for this patient?

A. Cryotherapy
B. Curettage
C. Full malignancy screen
D. Imiquimod
E. Reassurance and discharge

C. Full malignancy screen

The abrupt appearance of multiple seborrhoeic keratoses suggests underlying cancer (LeserTrelat sign)

The question gives a classic description of seborrhoeic keratoses.

These lesions are common and are benign although the rapid development of large numbers of them is cause for concern. This is termed the Leser-Trelat sign and is most commonly associated with gastrointestinal adenocarcinomas and hepatic cancers.

Full malignancy screen: a full malignancy screen is correct given the development of the LeserTrelat sign. Bloods including cancer markers as well as endoscopy / further imaging would be recommended to investigate for underlying malignancy.

Cryotherapy: cryotherapy and curettage are options for improvement of cosmetic appearance, but seborrhoeic keratoses usually do not require any specific treatment.

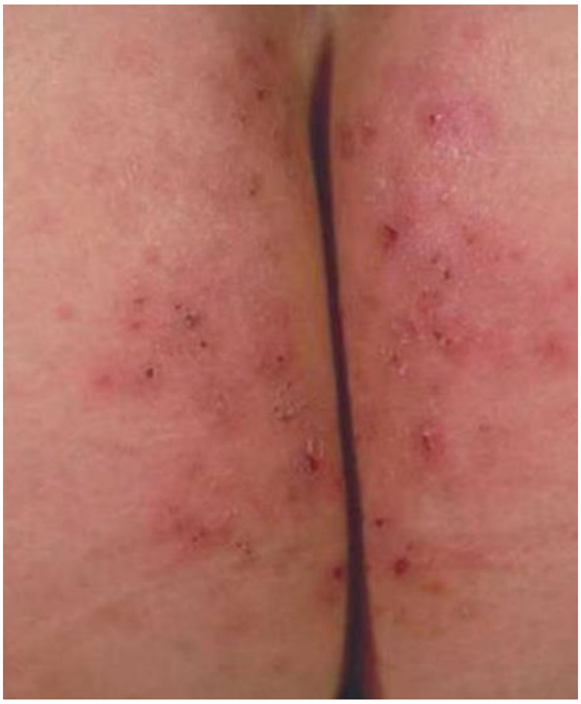
Imiquimod: Imiquimod is used for actinic keratoses rather than seborrhoeic keratoses.

Curettage: is an option for the treatment of seborrhoeic keratoses but with such high numbers would be unsuitable. Also, given the 'Leser-Trelat' sign, this patient requires investigation for underlying malignancy.

Reassurance and discharge: this is not appropriate in this case as the Leser-Trelat is associated with malignancy. If there were fewer lesions, or they had developed over a much longer time scale, it may be appropriate to reassure your patient.

Question 61 of 82

A 27-year-old man with a history of depression and coeliac disease presents with an itchy rash on his buttocks:



What is the most likely diagnosis?

A. Linear IgA dermatosis
B. Neurotic excoriations
C. Scabies
D. Dermatitis herpetiformis
E. SSRI-associated dermatitis

D. Dermatitis herpetiformis

The correct answer is **Dermatitis herpetiformis**. Dermatitis herpetiformis is a chronic, intensely pruritic skin disease characterised by grouped erythematous papules, vesicles and bullae. It is associated with coeliac disease, which the patient has a history of. The rash typically involves extensor surfaces such as the buttocks, elbows and knees. The diagnosis can be confirmed by skin biopsy showing granular IgA deposits at the dermo-epidermal junction.

Linear IgA dermatosis is an uncommon autoimmune blistering disorder that presents with tense blisters on normal or erythematous skin. It is not specifically linked to coeliac disease and while it can affect any body site, it favours the trunk and lower extremities rather than specifically the buttocks.

Neurotic excoriations refer to self-inflicted skin lesions produced by repetitive scratching. They are often seen in patients with psychiatric conditions including depression but tend to be more widespread and do not have the characteristic appearance of dermatitis herpetiformis.

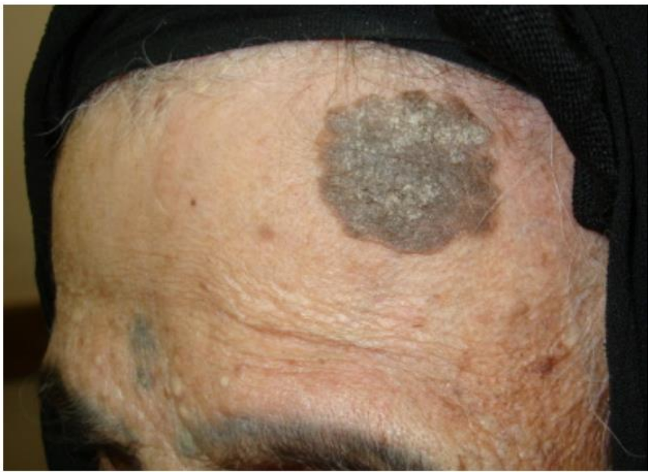
Scabies could present with an itching rash on the buttocks, however, scabies tends to cause more generalised itch especially in web spaces of fingers, wrists and ankles. The clinical picture would also usually include burrows or nodules which are not described here.

Lastly, **SSRI-associated dermatitis** refers to skin reactions related to selective serotonin reuptake inhibitors (SSRIs), a class of drugs commonly used in treating depression. While SSRIs can cause various skin reactions including rashes and pruritus, these tend to be more diffuse rather than localised solely on the buttocks as in this case scenario.

Therefore based on the clinical presentation and the patient's history of coeliac disease, Dermatitis herpetiformis represents the most likely diagnosis.

Question 62 of 82

A 68-year-old woman presents with the lesion below on the forehead. She explains that it has been present for several years and slowly growing.



It causes her no symptoms and she is otherwise well in herself. She has no significant past medical history except for hypertension, for which she takes ramipril.

Given the likely diagnosis, what is the most appropriate management?

A. Conventional surgery
B. Mohs surgery
C. Reassurance
D. Topical fluorouracil
E. Topical imiquimod

C. Reassurance

The correct answer is reassurance. This lesion has the characteristic appearance of seborrhoeic keratosis. This is a benign epidermal lesion seen in older people. They vary significantly in appearance - some have a more raised, 'stuck-on' appearance akin to a barnacle stuck on the skin, whereas some appear flat. The one above appears somewhat flatter. The uniformity of its colour and regular, well-demarcated border point toward seborrhoeic keratosis rather than malignant melanoma. For an asymptomatic patient, only reassurance is needed. If any itch, a topical corticosteroid may be used.

Conventional surgery is unwarranted. This would be used for a malignant lesion such as a melanoma, which would appear more irregular and have patchy colouring.

Mohs surgery is a technique used to remove malignant lesions from cosmetically-sensitive areas such as the face. This is often used for basal cell carcinomas of the face, which would not appear like the photo above - these are typically small, round, protruding lesions with pearly rolled edges and telangiectasia.

Topical fluorouracil may be used in the treatment of squamous cell carcinoma. These present as scaly, crusted, irregular lesions that often ulcerate and grow over weeks to months. For those in situ (known as Bowen's disease), topical treatments such as fluorouracil are a suitable initial option.

Topical imiquimod is an alternative to fluorouracil, also used in the treatment of squamous cell carcinoma in situ. It plays no role in the management of seborrhoeic keratosis, which is the most likely diagnosis given the picture above.

Question 63 of 82

A 21-year-old university student presents with an itchy skin rash. The rash came on at the start of the new school year around his groin, and a few weeks later he developed an intense itch all over his body.

On examination, he had an erythematous rash in the distribution described with wheals and papules and a mild scale. He has excoriated areas all over his body.

His flatmate complains of a similar rash on his hands.

Given the likely diagnosis, what would be the treatment of choice?

A. Malathion 0.5% to all skin for 12 hours, including the scalp, repeated after 14 days
B. Malathion 0.5% to the scalp, and permethrin 5% to the rest of the skin, for 12 hoursrepeated after 7 days
C. Permethrin 5% to all skin for 12 hours, including the scalp, repeated after 14 days
D. Permethrin 5% to all skin for 12 hours, including the scalp, repeated after 7 days
E. Permethrin 5% to all skin for 24 hours, excluding the scalp, repeated after 7 days

D. Permethrin 5% to all skin for 12 hours, including the scalp, repeated after 7 days

Scabies - permethrin treatment: all skin including scalp + leave for 12 hours + retreat in 7 days

The likely diagnosis here is scabies. The delayed itching is due to the delayed type IV hypersensitivity reaction to the mites and eggs. The rash is commonly found on the hands, flexor aspects of the wrists, groin, and around the genitals. It is spread by skin contact and so is common in households.

Permethrin 5% to all skin for 12 hours, including the scalp, repeated after 7 days is the correct answer. Permethrin should be applied to all the skin including the scalp and left for 12 hours before washing off. Patients should be advised to make sure their skin is clean and dry before applying and to ensure coverage in difficult-to-reach areas and skin creases. This process should then be repeated after 7 days.

Malathion 0.5% to all skin for 12 hours, including the scalp, repeated after 14 days is incorrect. Malathion 0.5% would be a second-line treatment option.

Permethrin 5% to all skin for 12 hours, including the scalp, repeated after 14 days is incorrect. According to the BNF, the treatment should be reapplied after 7 days.

Permethrin 5% to all skin for 24 hours, excluding the scalp, repeated after 7 days is incorrect. According to the BNF, the treatment should be washed off after 12 hours, the treatment area should also include the scalp.

Malathion 0.5% to the scalp, and permethrin 5% to the rest of the skin, for 12 hours repeated after 7 days is incorrect. Combination therapy like this is not recommended. If permethrin 5% was not successful in clearing the infestation, malathion 0.5% could be tried.

Question 64 of 82

A 50-year-old woman presents with a painful burning rash one week after starting chemotherapy. She describes a tingling sensation that started three days previously, which then progressed to swelling and redness of both her hands. She now finds that is impacting on what she able to do at home. Over the last day, she has noticed a blister as well. She has otherwise been suffering from fatigue and nausea since starting chemotherapy. She has a past medical history of left breast cancer managed with wide local excision and axillary node clearance following adjuvant radiotherapy and chemotherapy with docetaxel, as well as endocrine therapy.

What is the most appropriate management?

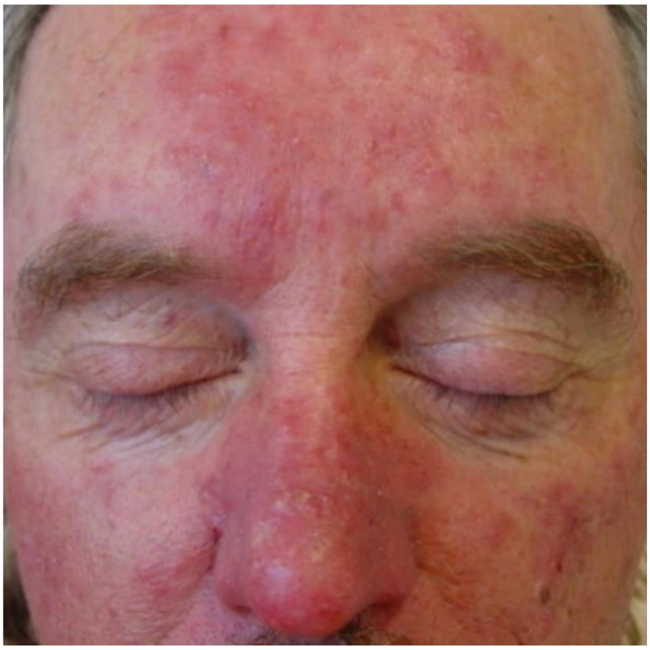
A. Topical hydrocortisone
B. Prednisolone
C. Flucloxacillin
D. Stop endocrine treatment
E. Dose reduction of docetaxel

E. Dose reduction of docetaxel

The answer is dose reduction of docetaxel. This history describes palmar-plantar erythrodysesthesia which is severe enough to limit daily activities. Whilst all cases can benefit from supportive management with emollients, cooling therapy and skin protection, severe reactions which cause an impact on the quality of life will usually be managed with dose reduction in chemotherapy. Steroids are not helpful in this condition, and there is no evidence of infection and therefore antibiotics are not appropriate. This explains why answers A, B and C are incorrect. Her endocrine treatment would not cause this problem and modifying it would be of no benefit as well, which is why D is incorrect.

Question 65 of 82

A 54-year-old man presents with a facial rash of several months. He explains that, initially, it started with redness across the face, before progressing to its current appearance as shown below:



He also mentions that it worsens in sunlight. He is otherwise fit and well, with no significant past medical history, nor any regular medications.

Given the likely diagnosis, which of the following is the most appropriate treatment?

A. Topical adapalene
B. Topical hydrocortisone
C. Topical ketoconazole
D. Topical ivermectin
E. Topical tacrolimus

D. Topical ivermectin

The correct answer is topical ivermectin - the diagnosis here is that of rosacea. The photo shows significant erythema across the forehead, nose and cheeks. Many papules and, to a lesser extent, pustules can also be seen across these same areas. The rosacea in this photo is not severe - rhinophyma of the nose would also be expected. The patient does appear to have some erythema and papules overlying the nose, which may be the start of rhinophyma. In severe rosacea, the rhinophyma would be more obvious than what is seen here. From the options above, topical ivermectin is the most appropriate treatment option in this case.

Topical adapalene is more commonly associated with the treatment of mild acne vulgaris, rather than rosacea. Typically, comedones would be present in acne vulgaris, colloquially referred to as 'whiteheads' and 'blackheads' depending on their appearance, which are not seen in this photo. Adapalene has been described in the literature as a potential alternative to topical metronidazole, although it is not recommended in guidelines for this purpose.

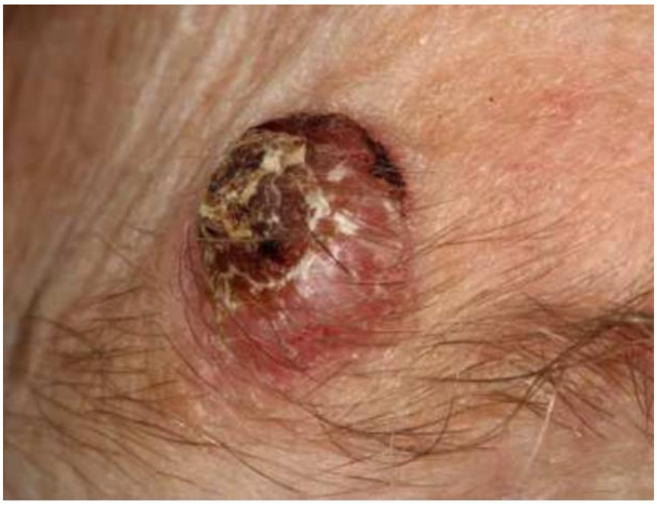
Hydrocortisone would be used for the treatment of facial eczema, not rosacea. Eczema would present with erythema and dryness, but not papules and pustules as seen in this photo. A mild corticosteroid may help to alleviate the appearance of erythema temporarily, but would not address the underlying issue.

Topical ketoconazole is an antifungal and would be used to treat seborrhoeic dermatitis rather than rosacea. This would present with erythema, but greasy, flaky scale rather than papules and pustules.

Tacrolimus can be used topically for steroid-resistant facial eczema, rather than stepping up to more potent steroids for the face. It is not routinely used in the management of rosacea.

Question 66 of 82

A 62-year-old man asks you to look at a lesion on his face:



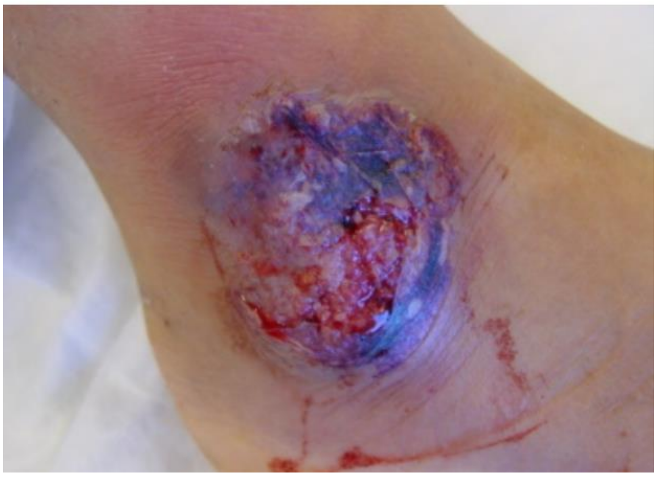
What is the most likely diagnosis?

A. Keratoacanthoma
B. Seborrhoeic keratoses
C. Actinic keratosis
D. Basal cell carcinoma
E. Pyoderma gangrenosum

Not answered

Question 67 of 82

A 67-year-old woman attends with a skin lesion. She described falling whilst walking in the Scottish Highlands and sustaining a small cut on her leg. This initially seemed to heal, but the woman then noted a blood-filled blister which has since burst and left the painful lesion shown below:



What is the most likely diagnosis?

A. Abscess
B. Cutaneous vasculitis
C. Pyoderma gangrenosum
D. Sweet's syndrome
E. Venous insufficiency ulcer

C. Pyoderma gangrenosum

The photo shows a typical pyoderma gangrenosum lesion with a deep ulcer and surrounding violaceous pigmentation.

Pyoderma gangrenosum is the correct answer. The diagnosis is often made on the characteristic appearance and history.

Cutaneous vasculitis is incorrect. This is a differential to consider, but is rare and is usually more widespread. In this case, there are also no systemic symptoms that you might expect with vasculitis.

Abscess is incorrect. This would be a fluctuant swelling, often with overlying cellulitis and would usually be associated with systemic symptoms.

Sweet's syndrome is incorrect. It is also known as acute febrile neutrophilic dermatosis and is a rare condition. It usually causes rashes on the upper body and face. It is an important differential but is not the most likely diagnosis here given the distribution of the rash and its rarity.

Venous insufficiency ulcer is incorrect. With venous insufficiency, you would expect to see associated skin changes and longer duration. This patient has no history of peripheral vascular disease which makes venous insufficiency ulcer even less likely.

Question 68 of 82

A 38-year-old man presents with a sudden onset rash. He is otherwise well in himself and has no notable past medical history. You see from his notes he has had a recent tonsillitis for which he received amoxicillin. On examination, there are multiple papules on his trunk and proximal extremities. There is a fine scale on several of these lesions.

What is the most likely diagnosis?

A. Drug eruption
B. Pityriasis rosea
C. Nummular dermatitis
D. Guttate psoriasis
E. Treponema pallidum

D. Guttate psoriasis

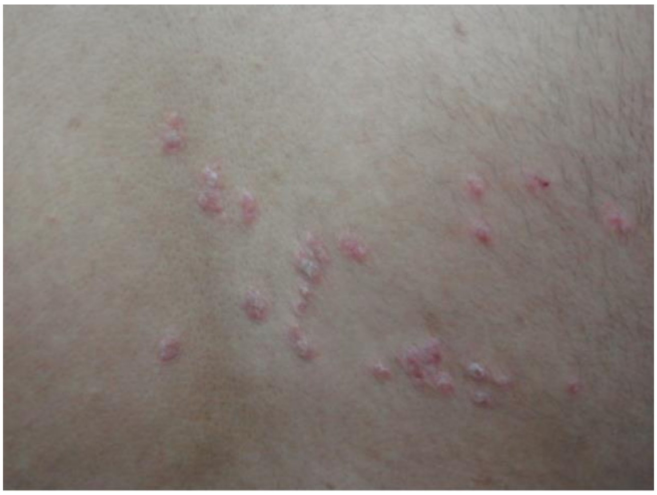
Acute onset of tear-drop scaly papules on trunk and limbs → ?guttate psoriasis

Guttate psoriasis usually presents in children and adults younger than 30 years of age. It can occur as the first presentation of psoriasis or as an acute exacerbation of plaque psoriasis, particularly after acute streptococcal infection (usually of the throat) or viral infection.

Lesions are small, round or oval (2 mm to 1 cm in diameter) scaly papules. They may be pink or red, but this can vary depending on the person's skin colour. Lesions occur all over the body, usually in large numbers and particularly on the trunk and proximal limbs (although the distal limbs can also be involved). Guttate psoriasis can occur on the face, ears, and scalp, but is rarely seen on the soles of the feet.

Question 69 of 82

A 19-year-old man presents with the following rash. He feels well in himself and has no significant past medical history. The patient does mention a sore throat that occurred several weeks ago and resolved itself after a few days.



Given the appearance above, what is the most likely diagnosis?

A. Guttate psoriasis
B. Lichen planus
C. Mycosis fungoides
D. Pityriasis rosea
E. Pityriasis versicolor

A. Guttate psoriasis

Acute onset of tear-drop scaly papules on trunk and limbs → ?guttate psoriasis

The photo above is typical of guttate psoriasis. Small, red, scaly plaques are visible. Some of the lesions in this photo have a 'tear-drop' appearance which is typical of guttate psoriasis. It classically follows a streptococcal throat infection several weeks prior. Most cases resolve spontaneously within 2-3 months. Standard psoriasis treatment may be used if needed to help resolve symptoms and appearance.

Lichen planus appears different from the photo above, with flat-topped, shiny, violaceous, polygonal papules, typically clustered around the wrists and ankles. They are overlaid by fine lines called Wickham striae, not the generalised scales seen here.

Mycosis fungoides is the most common type of primary cutaneous T-cell lymphoma. Rather than small, tear-drop lesions as seen here, it presents with poorly-defined, larger, more widespread patches, often with atrophic skin. It often occurs around sun-exposed sites.

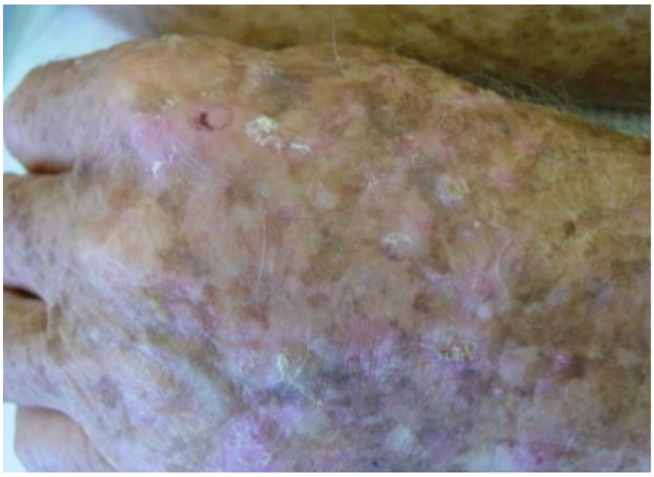
Pityriasis rosea is a reasonable differential - many patients report a preceding respiratory tract infection, but with regards to exam questions, this is not common. There is no mention of a herald patch in the question, which precedes the main pityriasis rosea rash by 1-2 weeks. The distribution of lesions typically follows a 'fir-tree' pattern, whereas, in the photo above, the distribution is relatively random.

Pityriasis versicolor is a fungal infection. Rather than the guttate lesions seen here, it leads to patchy, hypopigmented areas.

Question 70 of 82

A 67-year-old man presents to his general practitioner with skin lesions over his arms. They are not causing any itchiness or discomfort and have been developing over the past 6 months. He is otherwise well and his only past medical history of note includes hypertension and osteoarthritis.

The man reports he spends his time between the United Kingdom and a holiday home in Spain. He drinks approximately 10 units of alcohol per week and has a 20 pack-year smoking history.



What is the diagnosis?

A. Actinic keratoses
B. Basal cell carcinoma
C. Seborrhoeic dermatitis
D. Seborrhoeic keratosis
E. Squamous cell carcinoma

A. Actinic keratoses

The picture in this question depicts actinic keratoses which is the correct answer here. Sufferers often have several scaly/crusty lesions on sun-exposed areas such as the arms or head. As this man spends significant time in Spain, he is at an increased risk for developing these lesions.

Basal cell carcinomas classically present as a single pearly lump. There are several lesions in this instance and these are not consistent with this appearance. Basal cell carcinoma is therefore an incorrect answer.

Seborrhoeic dermatitis is a form of dermatitis triggered by Malassezia yeasts. This typically causes erythematous regions over the scalp, face, chest, and body creases. The distribution of the rash here as well as the scaly and crusty appearance is consistent with a diagnosis of actinic keratoses rather than seborrhoeic dermatitis.

Seborrhoeic keratoses are brown papules or plaques with a waxy appearance of unclear aetiology. The red and pink appearances of these lesions here are not consistent with this pathology.

Squamous cell carcinoma (SCC) will usually present as a solitary lesion and can arise from preexisting actinic keratoses. An SCC will typically be larger than a lesion in actinic keratoses and may become tender or ulcerated. As these features are not present here, actinic keratoses is the correct answer.

Question 71 of 82

A 24-year-old man who is normally fit and well presents with a new skin rash. He has no personal or family history of skin conditions. It started a couple of weeks ago with a lesion he noted on the hip, which is a patch approximately 4cm in diameter, mildly erythematous, with some mild scaling around the edge. He awoke this morning to find multiple smaller similar lesions, mainly on his back.

The lesions are asymptomatic.

How should this condition be managed?

A. Topical antifungal eg. ketoconazole shampoo
B. Mild topical steroid
C. No treatment required
D. Refer for phototherapy
E. Oral steroids

C. No treatment required

This is a typical history of pityriasis rosea, a self-limiting skin condition which most often occurs in teenagers and young people. The cause is unclear but thought to be viral in nature. An initial solitary 'herald patch' is followed some days to weeks later by multiple smaller lesions to the trunk. It is usually asymptomatic, in which case no treatment is required, but may cause a mild itch, in which case topical steroids and oral antihistamines could be considered. Referral to dermatology for phototherapy may be considered in extensive disease (some limited evidence of benefit).

Question 72 of 82

A 67-year-old man presents with a facial rash, which is more noticeable in the summer months. He describes flushing following sun exposure in the garden and eating spicy foods.

On examination, he has an erythematous, papular rash around his cheeks and forehead. There are visible pustules around his forehead and cheeks. There are no comedones and the rash is isolated to the face.

Which of the following would be the most appropriate first-line treatment?

A. Topical hydrocortisone
B. Topical ivermectin
C. Oral tetracycline
D. Azelaic acid
E. Emollient cream

B. Topical ivermectin

Rosacea: topical ivermectin is first-line for patients mild papules and/or pustules

This rash is typical of rosacea. This can present as flushing, erythema, telangiectasia and rhinophyma (enlarged nose).

For mild symptoms, topical therapy such as ivermectin gel is first-line. Azelaic acid can also be used but is usually not as well tolerated, especially for patients with sensitive skin.

For moderate or severe symptoms, an oral tetracycline or erythromycin can be used.

Question 73 of 82

A 25-year-old woman attends dermatology clinic for review of eczema. She has been suffering from eczema since she was young and has found the recent weather has been making her skin extremely dry and she finds that despite emollient application four times each day her skin is not improving. She also has asthma but this is well controlled and she rarely needs her inhaler. Apart from lotion emollients she is not using any creams currently. In the past she has needed topical steroids to manage her eczema when becomes very irritated. On examination the skin on over her flexural folds is very dry but does not appear irritated.

What can be advised to help manage the dry skin?

A. Advise to wear wool
B. Advise strong topical steroid use
C. Advise to change to cream rather than lotion emollient
D. Prescribe topical tacrolimus

E. Advise to change to a urea containing emollient

E. Advise to change to a urea containing emollient ×

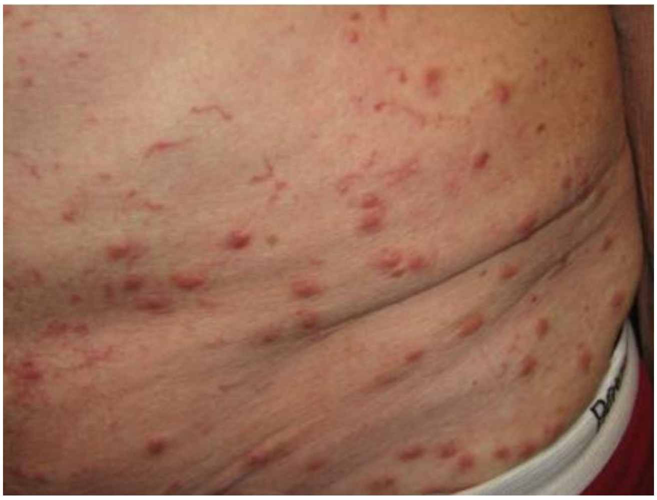
Adding urea to emollients increases moisturising effect, but can be inflammatory

Advise to change to a urea containing emollient Asking her to change to a urea containing emollient can help control dryness and itch as this can provide a greater moisturizing effect. Using urea containing emollients can increase inflammation and so in a acute inflammatory flare this would be inappropriate and such patients should be treated with topical steroids.

Changing to cream emollients would be appropriate for weeping skin. Tacrolimus can be used by dermatologists for refractory eczema.

Question 74 of 82

A 41-year-old man presents with an itchy rash over his arms and abdomen. It has got gradually worse over the past three days.



What is the most likely diagnosis?

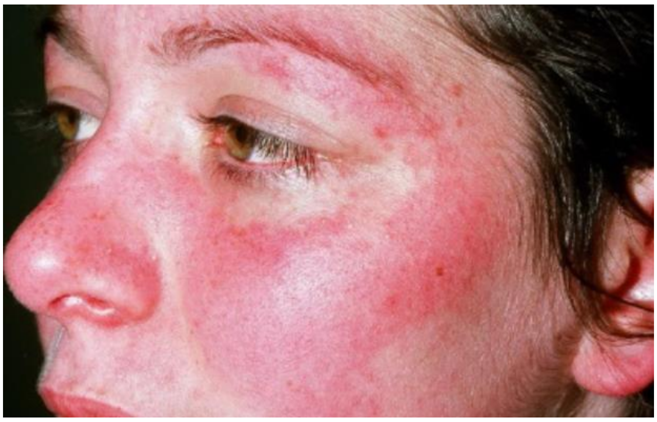
A. Scabies
B. Pityriasis rosea
C. Erythema multiforme
D. Urticaria
E. Guttate psoriasis

A. Scabies

The linear burrows of the scabies mite are clearly seen on this image.

Question 75 of 82

A 34-year-old woman presents with a rash on her face. She has been feeling generally unwell for around three months with lethargy and has a variety of joint pains



What is the most likely diagnosis?

A. Impetigo
B. Perioral dermatitis
C. Sarcoidosis
D. Acne rosacea
E. Systemic lupus erythematosus

E. Systemic lupus erythematosus

The correct answer is **Systemic lupus erythematosus**. This is a chronic autoimmune disease that can affect any system in the body, including the skin, joints, kidneys, heart and lungs. The classic malar or 'butterfly' rash seen in the image, along with systemic symptoms such as fatigue and arthralgia (joint pain), are characteristic of this condition. The rash typically spares the nasolabial folds. It's important to note that not all patients with lupus will present with this rash.

Impetigo is an incorrect answer. Impetigo is a highly contagious skin infection usually caused by *Staphylococcus aureus* or *Streptococcus pyogenes*. It generally presents as honey-coloured crusted lesions on exposed areas of skin and is more common in children. It would not explain this patient's systemic symptoms.

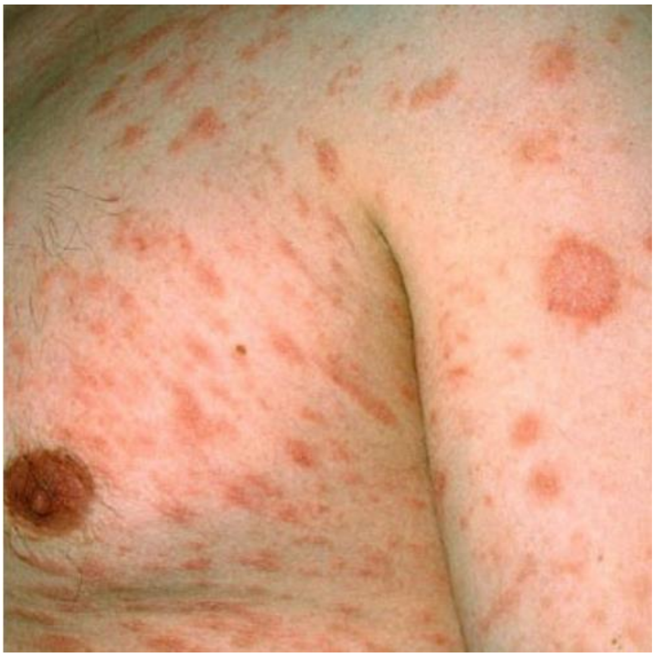
Perioral dermatitis, another incorrect option, typically presents as small red papules around the mouth and sometimes around the eyes or nose. The eruptions may be accompanied by mild itching or burning sensation but do not cause systemic symptoms like lethargy and joint pain.

Sarcoidosis is also an incorrect answer. While sarcoidosis can affect multiple organs and present with systemic symptoms similar to those described, its cutaneous manifestations are diverse but often include erythema nodosum (red tender nodules usually located on the shins) or specific lesions such as plaques, nodules or scars which look different from the presented image.

Finally, **Acne rosacea** is another incorrect choice. While it does present with facial redness and sometimes pustules similar to acne vulgaris, it primarily affects middle-aged adults and tends to be focused in the central face - cheeks, nose, forehead and chin. Rosacea does not cause systemic symptoms like fatigue or joint pains.

Question 76 of 82

A 29-year-old man presents with a rash on his trunk. Around two weeks ago he describes developing a patch of 'eczema' on his torso the size of 50 pence piece. Around a week later a number of smaller, red patches started to appear as well:



He feels systemically well and has no history of note. What is the most likely diagnosis?

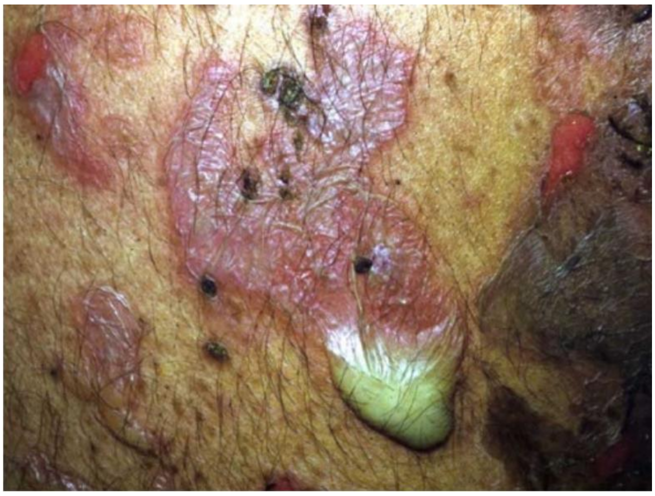
A. Erythema multiforme
B. Lichen planus
C. Pityriasis rosea
D. Guttate psoriasis
E. Tinea corporis

C. Pityriasis rosea

The earlier lesion represents a classical herald patch. Guttate psoriasis is generally has a more homogeneous, 'teardrop' appearance with a more pronounced scale. This image demonstrates the 'fir tree' appearance often seen in patients with pityriasis rosea.

Question 77 of 82

A 74-year-old man develops a painful blistering skin rash on his abdomen. He also complains of a sore throat, pain on swallowing and ulceration of the mouth:



What is the most appropriate treatment?

A. Oral prednisolone
B. Supportive treatment only
C. Intravenous flucloxacillin
D. Oral dapsone
E. Intravenous aciclovir

A. Oral prednisolone

Systemic glucocorticoids are the first-line treatment in pemphigus vulgaris

This patient has developed pemphigus vulgaris which is treated in the first instance by corticosteroids.

Question 78 of 82

A 29-year-old man present to the emergency department with a painful rash and fever. He was recently treated by his GP for a chest infection with oral amoxicillin. He found that his cough was improving but then noticed a painful rash. This was followed by a

fever, joint pain and feeling generally unwell. On examination, he has a maculopapular rash with target lesions, mucosal erosions and blistering lesions including within his oral cavity. His chest is clear on examination.

What timing of exposure is most consistent with Stevens-Johnson syndrome?

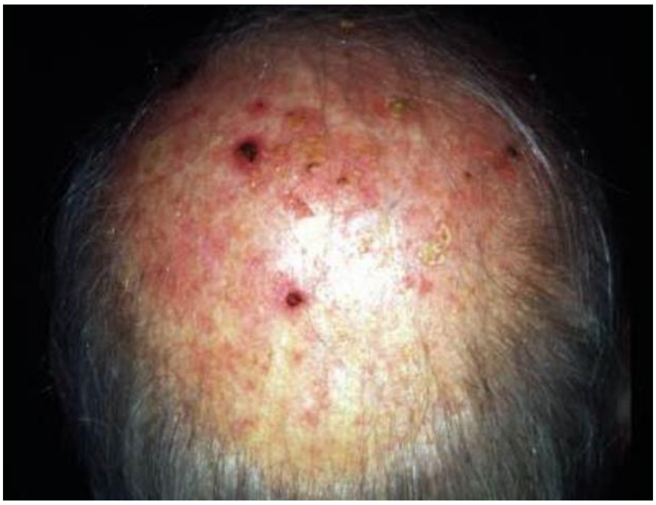
A. Within minutes
B. Within 24 hours
C. Within 3-5 days
D. Within 1-4 weeks
E. Within 4-8 weeks

D. Within 1-4 weeks

The correct answer is within 1-4 weeks. This patient has taken amoxicillin and subsequently developed a severe reaction. The description of a maculopapular rash with blistering lesions, oral involvement, fever and joint pain is all in keeping with Stevens-Johnson syndrome (SJS). SJS can occur within 1-4 weeks days of first drug exposure, and on average occurs at 14 days. In some cases, secondary exposure can lead to an earlier reaction within 48 hours but is more unusual. Another possibility is that it is secondary to Mycoplasma or Streptococcal infection. Drug reaction with eosinophilia and systemic symptoms usually occur within 2-6 weeks, whilst acute generalised exanthematous pustulosis occurs within 3-5 days.

Question 79 of 82

You are asked to look at some skin lesions on a patient who has been admitted with an infective exacerbation of COPD.



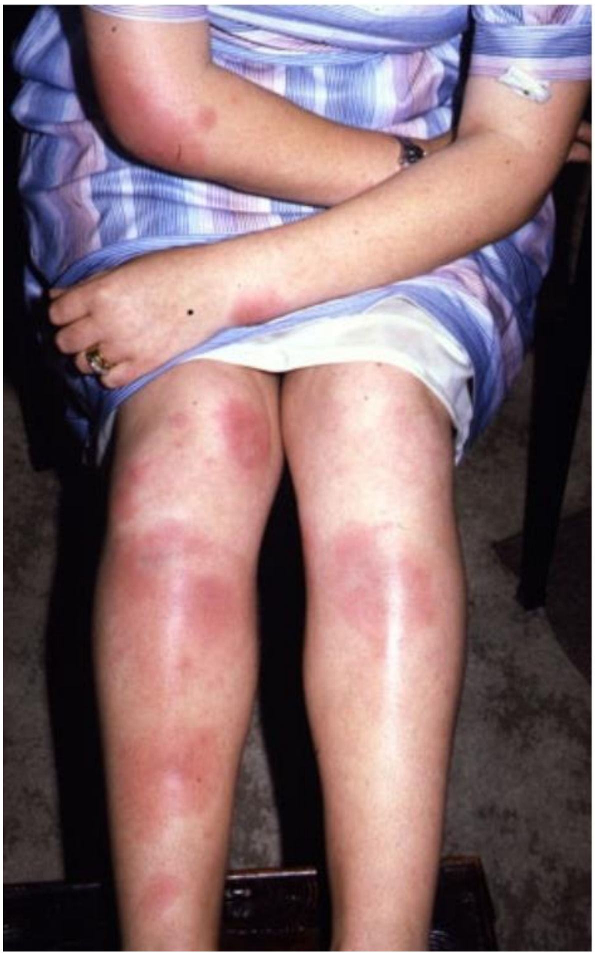
These skin lesions have been present for the past year. What is the most likely diagnosis?

A. Multiple basal cell carcinomas
B. Squamous cell carcinoma
C. Actinic keratoses
D. Seborrhoeic dermatitis
E. Seborrhoeic keratoses

C. Actinic keratoses

Question 80 of 82

A 62-year-old woman presents with painful 'bruises' on her shins and forearms.



She cannot remember knocking herself. What is the most likely diagnosis?

A. Idiopathic thrombocytopenic purpura
B. Erythema ab igne
C. Thrombotic thrombocytopenic purpura
D. Erythema nodosum
E. Cellulitis

D. Erythema nodosum

The most likely diagnosis for this 62-year-old woman presenting with painful 'bruises' on her shins and forearms is **Erythema nodosum**. Erythema nodosum is a type of panniculitis, or inflammation of the subcutaneous fat. It presents as tender, erythematous nodules typically located on the anterior shins but can also appear on the forearms. The condition may occur spontaneously or be associated with systemic diseases such as sarcoidosis, inflammatory bowel disease or streptococcal infection.

Idiopathic thrombocytopenic purpura (ITP) is an autoimmune disorder characterised by low platelet count leading to easy bruising and excessive bleeding. However, the bruises in ITP are usually not painful which makes it less likely in this case.

Erythema ab igne is a skin condition caused by prolonged exposure to heat or infrared radiation. It presents as mottled, erythematous patches that can become permanent if exposure continues. Given that the patient does not report any relevant exposures and the lesions are painful rather than asymptomatic, this diagnosis would be less likely.

Thrombotic thrombocytopenic purpura (TTP) is a rare blood disorder that causes blood clots to form in small blood vessels throughout the body. This can lead to serious problems if left untreated such as kidney failure and stroke. The bruises seen in TTP are similar to those seen in ITP - they are usually not painful and often accompanied by other symptoms like fatigue, fever, confusion which have not been reported in this case.

Lastly, **Cellulitis** is an infection of the deeper layers of skin and underlying tissue. It causes redness and swelling on affected areas of skin which are warm to touch due to inflammation caused by bacteria (most commonly Group A Streptococcus or Staphylococcus aureus). Cellulitis typically affects one side of the body, unlike our case where both shins and forearms are involved hence making it less likely.

Question 81 of 82

A 24 year-old gentleman is referred for your opinion from the Emergency Department. He presented with his first tonic-clonic seizure. A CT scan of his head shows a contrast enhancing lesion in the left frontal lobe. On taking a history you elicit that he has been having recurrent nosebleeds and dark stools for the past 12 months.

What is the likely unifying diagnosis?

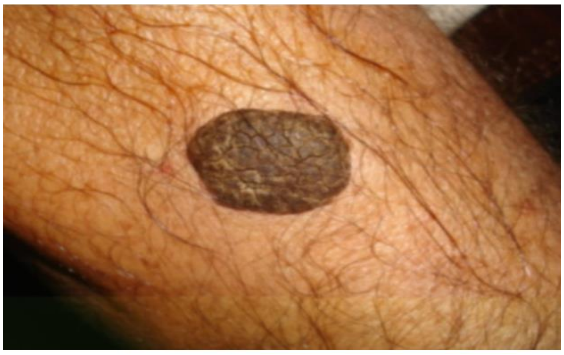
A. Peutz Jeghers syndrome
B. Hereditary haemorrhagic telangiectasia
C. Tuberous sclerosis
D. Von Willebrand disease
E. Neurofibromatosis - type 2

B. Hereditary haemorrhagic telangiectasia

This young gentleman is likely to have a diagnosis of hereditary haemorrhagic telangiectasia (HHT). The contrast enhancing lesion on the CT scan represents an arterio-venous malformation. The history of recurrent nosebleeds and melena is typical of HHT and are secondary to telangiectasia on the nasal and gastrointestinal mucosa.

Question 82 of 82

A 70-year-old man presents to the dermatology clinic with a lesion on his lower shin. He noticed the lesion 3 weeks ago after going on a resort holiday in Turkey and wearing shorts for the first time in several months. He denies pain or itch but does think it is getting bigger in size. His past medical history includes ischaemic heart disease and chronic obstructive pulmonary disease. His lesion is examined, as shown below:



What is the most likely diagnosis?

A. Basal cell carcinoma
B. Malignant melanoma
C. Seborrhoeic keratoses
D. Solar lentigo
E. Squamous cell carcinoma

C. Seborrhoeic keratoses

Seborrhoeic keratoses is correct. This patient has a well-circumscribed pigmented lesion on his shin that appears flat on the skin surface. It also has the appearance of a wart-like texture. This is in keeping with a diagnosis of seborrhoeic keratoses, a benign lesion associated with increasing age, particularly beyond 60 years. Seborrhoeic keratoses often have a 'stuck-on' appearance as seen with this patient. Treatment is not necessary for the lesions. However, curettage or cryotherapy can be offered for complete removal.

Basal cell carcinoma is incorrect. Basal cell carcinoma (BCC) is a locally-invasive cutaneous malignancy that is typically a slow-growing plaque with rolled edges and a smooth surface. They are usually pink or skin-coloured and, therefore, do not represent this patient's lesion.

Malignant melanoma is incorrect. Melanoma is a serious differential diagnosis to consider, particularly for pigmented lesions. However, characteristic features of malignant melanoma include asymmetry, irregular borders, bleeding, change in colour, increase in diameter and evolution. In contrast, this patient's lesion is well-circumscribed with no evidence of hyperpigmentation, bleeding, crusting or irregular borders.

Solar lentigo is incorrect. Solar lentigo is a benign patch of darkened skin secondary to a local proliferation of melanocytes. This process is triggered by UV radiation, giving solar lentigo the alternative name of 'age spots'. Seborrhoeic keratoses may arise from solar lentigo. However, unlike seborrhoeic keratoses, the skin is not thickened in solar lentigo, only darkened.

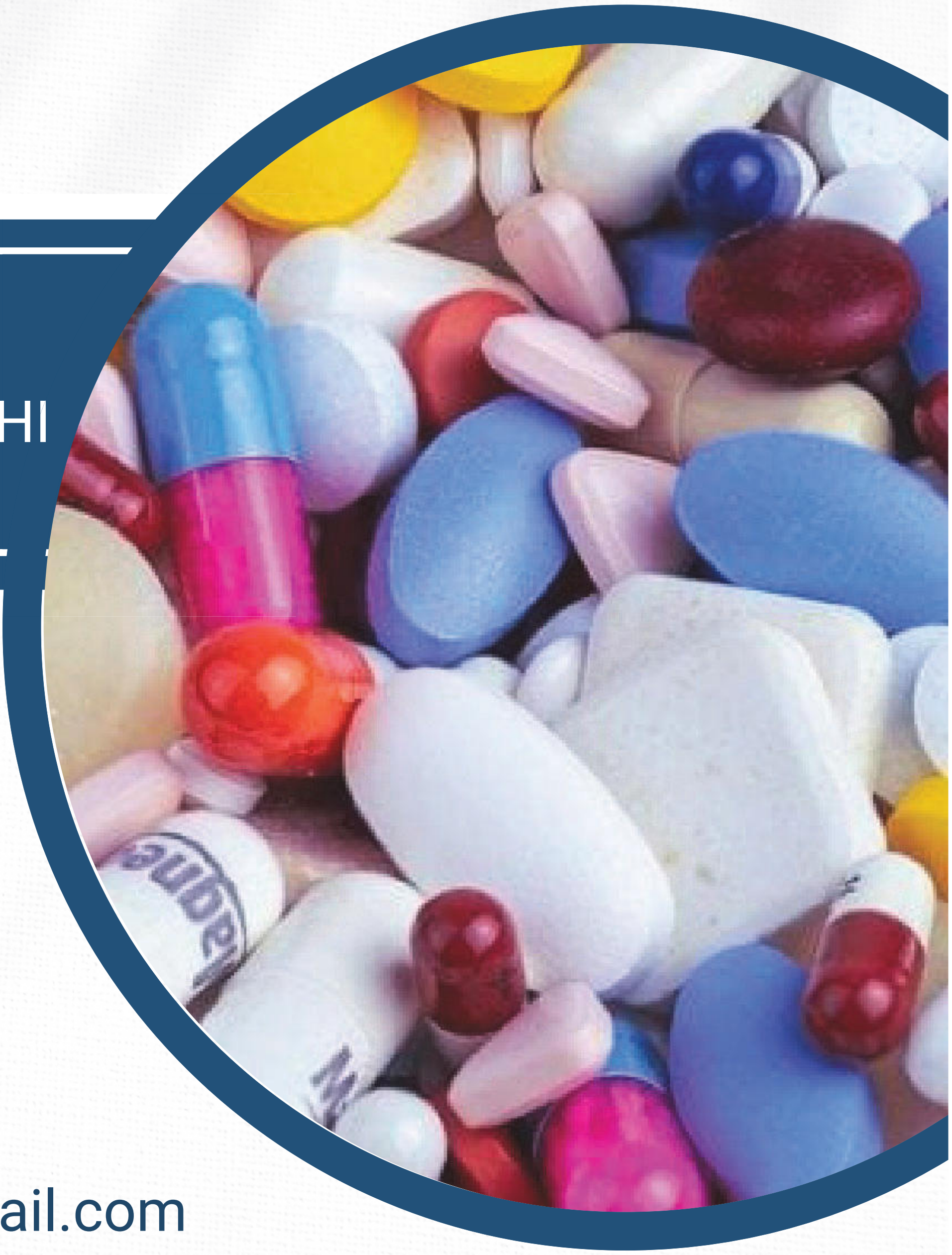
Squamous cell carcinoma is incorrect. Squamous cell carcinoma (SCC) is an invasive cutaneous malignancy that presents as an enlarging, crusted lesion that may ulcerate and are often tender. SCC typically progresses from actinic keratoses. They are non-melanocytic lesions, making SCC incorrect.

Alrajhi Jad

PASSMEDICINE

THERAPEUTICS & GERIATRICS

Internal medicine



EDITED BY
DR. JAMIL ALRAJHI

2024

773-555-493
jameelalrajhi@gmail.com

THERAPUTICS

passmedicine PART 2

2024

Question 1 of 138

A peri-arrest call is put out for a 71-year-old woman on the high dependency unit. An arterial line was being inserted and upon administration of some local anaesthetic, a sudden reduction in consciousness was noted. The patient has now started to have a seizure.

Her vital signs are 91% on 15 litres of oxygen, respiratory rate of 7/min, heart rate of 54/min, blood pressure of 72/54mmHg, the patient is unresponsive and has a temperature of 35.9ºC.

As well as emergency management, what would be the most effective treatment to give in this instance?

A. A tropine
B. Flumazenil
C. Fomepizole
D. Lipid emulsion
E. Sugammadex

A. Lipid emulsion

Local anesthetic toxicity can be treated with IV 20% lipid emulsion

This woman is suffering from local anaesthetic toxicity. The most common symptoms initially are confusion, drowsiness, and dizziness, however, this can progress to seizures, respiratory arrest, and coma. Treatment includes life support measures and benzodiazepines to control seizures. The most appropriate reversal agent is intravenous lipid emulsion which is, therefore, the correct answer.

Atropine is effective at treating bradycardia however is not the reversal agent for local anaesthetic toxicity. Whilst this woman is bradycardic, this is very likely to be refractory to treatment solely with lipid emulsion without co-administration of atropine.

Flumazenil is the reversal agent for benzodiazepine overdoses however has no role in local anaesthetic toxicity. In this instance, benzodiazepines are indicated to control the seizures and flumazenil would only act to further lower the seizure threshold.

Fomepizole is the reversal agent for ethylene glycol or methanol which can cause reduced consciousness but has no role in local anaesthetic toxicity which is the diagnosis here.

Sugammadex is the reversal agent for muscle relaxants such as rocuronium. Muscle relaxants are not administered during the insertion of an arterial line and therefore sugammadex is not indicated here.

Question 2 of 138

A 24-year-old man with a history of depression and self-harm is brought to the emergency department by a friend 2 hours after an impulsive overdose of ferrous sulphate. He weighs 65kg and has ingested 65 x 200mg ferrous sulphate tablets. This is equivalent to 4225mg of elemental iron (65mg/kg).

On assessment, he denies any abdominal pain, diarrhoea or vomiting.

Observations are as follows:

- Temperature 36.5
- Respiratory rate 18/min
- Saturations 98% on air
- Heart rate 82bpm
- Blood pressure 136/74 mmHg

Venous blood results show:

Hb	133 g/l	Na +	139 mmol/l	Bilirubin	8 µmol/l
Platelets	287 * 10 /l 9	K +	4.1 mmol/l	ALP	89 u/l
WBC	8.3 * 10 /l 9	Urea	3.4 mmol/l	ALT	34 u/l
Neuts	6.2 * 10 /l 9	Creatinine	72 µmol/l	Albumin	40 g/l
Lymphs	1.9 * 10 /l 9	Serum iron	70 umol/l		
Eosin	0.2 * 10 /l 9	pH	7.40		

Abdominal x-ray demonstrates the presence of iron in the stomach.

What is the appropriate initial medical management

A. Whole bowel irrigation
B. Activated charcoal
C. Emergency endoscopy for removal of iron from the stomach
D. Observe and repeat iron levels 4-6 hours after ingestion
E. Administer desferrioxamine

A. Whole bowel irrigation

Question 3 of 138

A 31-year-old gentleman, with known ulcerative colitis, presents with a three-day history of profuse bloody diarrhoea. He reports opening his bowels up to 12 times each day and describes passing both bloody stool and frank blood.

On examination he is pale. His heart rate is 95 beats per minute, respiratory rate 16 breaths per minute, blood pressure 128/78 mmHg. His chest is clear to auscultation and his abdomen is soft but diffusely tender. The patient is ambulant and despite his diarrhoea appears relatively well.

The patient is diagnosed with a flare of his ulcerative colitis. Intravenous access is obtained and fluids and intravenous steroids given.

Admission bloods show:

Hb	102 g/l
Platelets	556 * 10 /l9
WBC	17.5 * 10 /l9
ESR	78 mm/h

Regarding his venous thromboembolism prophylaxis, what should happen during this admission? ?

A. Compression stockings only

B. No prophylaxis due to active bleeding
C. Intermittent pneumatic compression devices
D. Prophylactic dose low molecular weight heparin and compression stockings
E. Treatment dose low molecular weight heparin and compression stockings

D. Prophylactic dose low molecular weight heparin and compression stockings

The severity of a flare of ulcerative colitis can be categorised used the Truelove and Witt criteria:

SEVERITY	MILD	MODERATE	SEVERE
Bowel movements per day	fewer than 4	4-6	>6 and features of systemic upset
Blood in stools	Small amounts	Between mild and severe	Visible blood
Pyrexia (temp >37.8ºC)	No	No	Yes
Pulse >90 bpm	No	No	Yes
Anaemia	No	No	Yes
ESR (mm/hr)	30 or below	30 or below	Above 30

Based on these criteria, this patient can be defined as suffering an acute severe flare of ulcerative colitis which is a medical emergency. Intensive treatment with intravenous steroids and early surgical intervention has reduced the mortality from acute severe ulcerative colitis to 2.9% however the colectomy rate of 30% in acute flares has not changed for 40 years.

Whilst this patient is complaining of passing blood per rectum an acute flare of ulcerative colitis is an extremely prothrombotic state and as a result, the British Society of Gastroenterology recommends that all patients should receive subcutaneous heparin to reduce the risk of thromboembolism. Intermittent pneumatic compression devices are a form of venous thromboembolism prevention that has evidence in acute stroke so is not the correct answer here. Whilst the patient is in a prothrombotic state, there is no indication for treatment dose low molecular weight heparin and this would increase the patient's risk of excessive blood loss.

Question 4 of 138

A 67 year old man is admitted to the Emergency Department following a deliberate overdose of an unknown drug. On assessment he is unwell and vomiting although he is maintaining an airway. Oxygen saturations are 100% on 15L/min supplemental oxygen and his respiratory rate is 32/min. His chest is clear and heart sounds are rapid. An ECG shows sinus tachycardia with a rate of 135bpm, a broad QRS complex and a QTc 520msec. His skin is flushed and hot. He complains of ringing in his ears and visual blurring as well as abdominal pain. Tympanic temperature is 37.5°C.

Blood results show:

Haemoglobin	101g/L	Sodium	131mmol/L	pH	7.11
White cells	7.8x10^9/L	Potassium	2.9mmol/L	pCO2	2.4kPa
Platelets	97x10^9/L	Urea	9.3mmol/L	pO2	78.4kPa
INR	1.3	Creatinine	198µmol/L	HCO3-	6.3mmol/L
APTT	33secs	Glucose	2.8mmol	BE	-19.7
Chloride	94mmol/L	Lactate	2.4mmol/L		

During assessment he has a generalised tonic-clonic seizure.

Which of the following drugs is he most likely to have overdosed on

A. Ibuprofen
B. Isocarboxazid
C. Paracetamol
D. Quinine
E. Venlafaxine

D. Quinine

Quinine toxicity (cinchonism) presents with myriad **ECG changes**, hypotension, metabolic acidosis, **hypoglycaemia** and classically tinnitus, flushing and visual disturbances. Flash pulmonary oedema may occur

Paracetamol toxicity presents with early hepatotoxicity and coagulopathy, and later renal failure and encephalopathy which are features lacking in this clinical scenario.

Ibuprofen, a non-steroidal anti-inflammatory, is relatively safe in overdose although massive ingestion may cause gastrointestinal ulceration and haemorrhage as well as electrolyte imbalances, coagulopathy and prominent cerebellar signs. This is not commented upon in the clinical vignette.

Venlafaxine is a selective serotonin and noradrenaline reuptake inhibitor and presents with symptoms of serotonin and sympathetic overdrive in poisoning, including agitation, sweating, hyperpyrexia, clonus, tachycardia and hypertension. ECG abnormalities similar to the above may occur, as may hypoglycaemia but venlafaxine is not the most likely agent.

Isocarboxazid is a monoamine oxidase inhibitor and in overdose may also cause symptoms of the serotonin syndrome although sympathomimetic features tend to predominate, including hyperglycaemia.

Question 5 of 138

A 28-year-old female with a history of cerebral palsy is admitted for an elective intrathecal pump refill. The procedure unfortunately was unsuccessful and the pump could not be re-sited. Later on in the evening the patient complains of severe pain in her lower limbs and feels generally unwell.

Blood results are as follows:

Hb	145 g/l	Na^+	138 mmol/l
Platelets	525 * 10 /l 9	K^+	5.6 mmol/l
WBC	14.1 * 10 /l 9	Urea	12.8 mmol/l
Neuts	8.4 * 10 /l 9	Creatinine	156 µmol/l
Creatine kinase	8445 Units/litre (normal range 24-170)	CRP	44 mg/l

What drug is the patient withdrawing from?

A. Lidocaine
B. Baclofen
C. Pethidine
D. Clonidine
E. Ketorolac

B. Baclofen

Intrathecal baclofen withdrawal syndrome is associated with severe spasticity, rhabdomyolysis, acute renal failure and multisystem organ failure

Intrathecal baclofen can be used to treat spasticity. It can be administered continuously via a pump. It is believed to work by agonising GABA receptors.

Discontinuation of baclofen can be associated with a withdrawal syndrome which resembles benzodiazepine withdrawal and alcohol withdrawal. Intrathecal baclofen withdrawal syndrome is associated with severe spasticity, rhabdomyolysis, acute renal failure and multisystem organ failure.

Question 6 of 138

A 76-year-old man presented with shortness of breath to the emergency department. On the basis of a recent knee replacement operation and unremarkable chest x-ray a CT pulmonary angiogram was performed and demonstrated a segmental pulmonary embolus with evidence of mild right heart strain. The patient was haemodynamically stable and required only minimal supplemental oxygen therapy.

The patient had known chronic kidney disease stage IV secondary to type 2 diabetes and hypertension. Treatment was therefore initiated with an intravenous unfractionated heparin infusion.

The patient's condition was stable over the following week with warfarin loading cautiously started at day 6 of admission. Routine blood tests at this point indicated a new abnormality in full blood count leading to further investigations as detailed below.

Haemoglobin	14.5 g / dL
White blood cells	8.6 x 10>3 / microlitre
Neutrophils	4.5 x 10>3 / microlitre
Lymphocytes	2.1 x 10>3 / microlitre
Platelets	67 x 10>3 / microlitre
Mean cell volume	85 fL
Mean cell haemoglobin	30.1 pg
B12	252 pmol / L
Folate	20 nmol / L

Heparin induced thrombocytopenia antibodies: positive (high titre)

Following cessation of IV heparin infusion,

what is the appropriate management of the patient's thrombocytopenia?

A. Bivalirudin
B. Enoxaparin
C. Warfarin
D. Tirofiban
E. Platelet transfusion

A. Bivalirudin

This patient has a confirmed diagnosis of heparin induced thrombocytopenia (HIT) based on the suggestive clinical picture and positive HIT antibody screen. Decision tools such as the Warkentin probability scale can be useful by providing a pre-test probability score of HIT based on the clinical picture. In this case, the timing of thrombocytopenia with onset between 5-10 days after initial exposure to heparin is highly suggestive.

In suspected or confirmed cases of HIT, heparin anticoagulants must be withheld immediately. Treatment with a non-heparin anticoagulant such as bivalirudin should be started to reduce the risk of HIT-related thrombosis. Vitamin K antagonists such as warfarin such be withheld or reversed as they do not prevent HITassociated thrombosis and increase the risk of venous gangrene. Platelet transfusion would not be indicated in this patient.

Question 7 of 138

A 24-year-old man is brought to the emergency department having developed acute confusion and fever. The family tell you he was well on going to bed last night then become poorly over several hours this morning.

He has a background of depression for which he takes sertraline. His mother tells you he has recently been trying some over-the-counter remedies for his depression and has also been complaining of lower back pain, for which he has started new painkillers. A set of observations are available.

Respiratory rate 30/min
Oxygen saturation 97% on air
Heart rate 120/min
Blood pressure 184/106mmHg
Temperature 39.1ºC

On examination, you find him to be agitated and disorientated. Neurological assessment reveals generalised rigidity and brisk reflexes. His pupils are dilated and you note marked myoclonus at both ankles. An ECG shows sinus tachycardia with a normal QRS duration.

What treatment is indicated?

A. Cyproheptadine
B. Dantrolene
C. Glucagon
D. N-acetylcysteine
E. Sodium bicarbonate

A. Cyproheptadine

Treatment of severe serotonin syndrome- cyproheptadine (5-HT2 receptor antagonist)

The correct answer is **cyproheptadine**. This is a 5-HT2 receptor antagonist used in the treatment of serotonin syndrome. Key features include rigidity, tachycardia, hyperpyrexia and hypertension. In this case, the rapid onset, dilated pupils and brisk reflexes support this as the diagnosis. The over-the-counter remedy and new painkiller may represent St. John's wort or tramadol, both of which may interact with SSRIs such as sertraline to induce this syndrome.

Dantrolene is incorrect. This is the treatment for neuroleptic malignant syndrome. This syndrome is associated with antipsychotics rather than anti-depressants. It also generally is of slower onset than serotonin syndrome and presents with normal pupils and diminished reflexes.

Glucagon is incorrect. This is a treatment used in the management of beta-blocker overdose. There is no suggestion this patient has ingested these in the case described. The presentation is also not in keeping with beta-blocker toxicity, particularly tachycardia.

N-acetylcysteine is incorrect. This is the antidote for paracetamol overdose. Although this may be the painkiller he is taking for his back pain, it would not account for the clinical features described. Paracetamol overdose generally causes hepatotoxicity.

Sodium bicarbonate is incorrect. This can be used in the management of tricyclic antidepressant overdose. The case does not describe ingestion of these, and significant toxicity is associated with a widening of the QRS complex leading to ventricular arrhythmias. In this case, we are told the QRS is normal, which would go against significant TCA toxicity.

Question 8 of 138

A 17 year old girl is brought to the Emergency Department following a prolonged seizure lasting seven minutes. She is known to suffer from epilepsy and has been stable on lamotrigine for eight years, having on average only one generalised tonic clonic seizure every eight months. Her mother describes an increase in her seizure frequency over the last four weeks; she has has three generalised tonic-clonic seizures during this period, each lasting around three minutes. There have been no changes to stress levels, sleep pattern or diet. She confirms she has however started a new medication for dysmenorrhoea.

Which is the most likely to explain this increase in frequency?

A. Medroxyprogesterone (Depo-Provera)
B. Mefenamic acid
C. Ibuprofen
D. Aspirin
E. Naproxen

B. Mefenamic acid

Taking a full medication history is an essential aspect of any history; it can be particularly important in epilepsy, not only to gauge medication compliance and anti-epileptic therapy, but to determine any precipitating factors.

Analgesics that are known to induce seizures or worsen seizure control include fentanyl, mefenamic acid, and tramadol (among others). Other drugs that are known to induce seizures include amitriptyline, aminophylline, isotretinoin and haloperidol.

It is important to be aware of the interaction between contraception and antiepileptic medication; contraceptive failure, teratogenicity and reduced seizure control are potential effects.

P450 enzyme inducers will decrease the drug level and therefore increase the failure rate of oestrogen and progesterone containing contraceptives.

- Carbamazepine
- Oxcarbazepine
- Phenytoin
- Phenobarbitone
- Primidone
- Topiramate

Lamotrigine is not a P450 inducer. Studies have shown a slight decrease in levonorgestrel levels, although the clinical significance is undetermined. Levels of lamotrigine are reduced by oral contraceptives containing ethinylestradiol, increased seizure frequency may therefore occur. There is no evidence that progestogen-only methods affect lamotrigine levels.

Question 9 of 138

A 34-year-old man is seen in the emergency department at 5 am on Saturday morning in a state of extreme agitation. He is alert from the end of the bed, tremulous and diaphoretic. His respiratory rate is 26 breaths per minute, oxygen saturation 97% on air, heart rate 132 beats per minute, blood pressure 185/103mmHg and temperature 38.2ºC. There is bilateral air entry on auscultation of the chest with vesicular breath sounds. His peripheries are clammy, hot to touch and his pulse is fast and bounding. His heart sounds are normal with no added murmurs. His JVP is not raised. He is talking to you but is confused. Pupils are equal and reactive to light but markedly dilated. There is no lateralising neurology.

The nurse cannulates the patient and takes blood. The initial investigation results are as follows:

ECG	sinus tachycardia, 1mm T wave inversions in V2-V6
Urine dipstick	+ protein +++ haemoglobin

Venous blood gas:

pH	7.32	7.31-7.41
pO 2	5.35 kPa	4.0-5.3 kPa
pCO 2	4.73 kPa	5.5-6.8 kPa
Lac	3.2 mmol/L	<2.0 mmol/L
Glucose	6.4 mmol/L	4.0-7.8 mmol/L
HCO3	24 mmol/L	23-29 mmol/L
BE	-3	-2 - +2

What would be the next most appropriate management step?

A. Aspirin, clopidogrel, fondaparinux and sublingual glycerine trinitrate
B. CT aortogram
C. IV benzodiazepines
D. IV labetalol
E. IV 8.4% sodium bicarbonate

C. IV benzodiazepines

Treatment of cocaine toxicity is largely supportive with judicious usage of benzodiazepines

This man displays signs of cocaine toxicity with dilated pupils, hypertension, tachycardia, anxiety, likely myoglobinuria secondary to rhabdomyolysis and myocardial strain. The management would involve IV benzodiazepines. Nitroglycerine can also be useful in the face of coronary vasospasm.

It is important to bear in mind that β-blockers are contraindicated in cocaine toxicity due to the risks of the unopposed α-agonist activity. For this reason, IV labetalol would not be the next most appropriate management step.

Aspirin, clopidogrel, fondaparinux and sublingual glycerine trinitrate would be the first line if you suspected a non-ST elevated myocardial infarction. In this case, the main concern is cocaine toxicity which is causing signs of rate-dependent ischemia on the ECG. First line management would be benzodiazepines which would help to resolve this issue.

A CT aortogram would be useful if you suspected aortic dissection. There is no strong evidence that this is occurring and cocaine toxicity is the more likely diagnosis in this case.

IV 8.4% sodium bicarbonate is useful for overdoses of tricyclic antidepressant but it is not the next most appropriate management step in the case of cocaine toxicity.

Question 10 of 138

A 55-year-old man presents to the Emergency Department after taking an overdose. He can not tell you which of his regular tablets he has taken an overdose of. His past medical history includes Crohn's disease, gastroesophageal reflux disease, hypertension, gout, depression and paroxysmal atrial fibrillation.

He is complaining of chest pain and shortness of breath. Observations - respiratory rate 26/min, saturations 94% on air, heart rate 50/min, blood pressure 75/40 mmHg, temperature 37.2ºC.

His ECG shows atrial fibrillation with a broad QRS. Which drug is the most likely culprit?

A. Flecainide
B. Amitriptyline
C. Allopurinol
D. Azathioprine
E. Diltiazem

A. Flecainide

Flecainide is the most likely culprit here due to the chest pain, signs of heart failure and prolonged QRS. Diltiazem would be also be possible, but in overdose would likely show a more marked bradycardia with heart block.

Flecainide is a Vaughan Williams Class Ic anti-arrhythmic drug, associated with PR, QRS, and QTc prolongation on the electrocardiogram and development of life-threatening cardiac toxicity in overdose.

The cornerstone of treatment is fluid resuscitation and the administration of magnesium and sodium bicarbonate. Intravenous fat emulsion has also been trialed with some success.

Note - flecainide should NOT be used in patients who have a structurally abnormal heart due to the risk of causing arrhythmias, including torsades de pointes.

Question 11 of 138

A 27 year old well-nourished male electroplater is admitted to hospital with abdominal pain and neurological symptoms. He tells you that he has had worsening symptoms for the past three months with severe central abdominal pain, diarrhoea, vomiting and painful burning sensation in both feet. He also describes increasing clumsiness with loss of manual dexterity and tripping over his feet frequently. He describes dimming of his vision over the past two weeks with a sepia tinge. On examination, there is mild voluntary guarding of the abdomen but no discrete masses and no organomegaly. The cardiorespiratory examination is normal. Examination of the neurological system reveals distal weakness with MRC power grade 4/5 in all limbs but normal proximal power. There is a mild tremor present with his arms outstretched and trunk ataxia. Sensory examination discloses painful paraesthesia in the hands and feet with hyperalgesia. There is a loss of proprioception. The cranial nerves are notable in that a left sixth nerve palsy is present with accompanying diplopia. Visual acuity is reduced to 6/30 bilaterally and eye movements are painful. He has a bilateral ptosis. He is noted to have minimal body hair, including hairless arms and legs, lateral third of the eyebrow and temporal and crown baldness which he tells you occurred in the past month. He also has scaling of the palms and soles, tender glossitis and transverse white lines on all his nails. He is cyclothymic during assessment.

What is the most appropriate management for this patient?

A. High dose IV hydrocortisone 200mg three times daily
B. High dose IV vitamin B complex (Pabrinex I+II) and folinic acid
C. Intravenous immunoglobulins
D. Oral Prussian Blue 10g twice daily
E. Procaine penicillin (G) 2 million unit daily IM with oral probenicid

D. Oral Prussian Blue 10g twice daily

Thallium poisoning is a rare cause of painful polyneuropathy, mood change and alopecia. Treatment is chelation therapy with oral Prussian Blue

The above clinical descriptor is classical for thallium poisoning. The triad of fluctuating mood, with or without confusion, painful distal paraesthesia and alopecia is virtually pathognomonic of toxicity with this heavy metal.

Thallium is a highly toxic metal; in its univalent Tl+ ion it is readily soluble in water and easily enters the body across the skin or gastrointestinal tract using potassium influx channels. Thallium fumes may also be generated on heating this metal and these may be inhaled when using it during many electroplating manufacturing processes. In the UK, the Health and Safety Executive stipulates that worker occupational exposure limits to thallium in any form must not exceed 0.1mg/m3 in an 8 hour period without adequate ventilation due to the health risks.

Thallium avidly binds to sulphur ligands within cellular proteins, disrupting many intracellular metabolic processes. In massive exposure, death from neuromuscular respiratory failure is rapid, but in more prolonged exposure insidious symptoms of neuropathy, abdominal pain, gastrointestinal bleeding, optic neuritis, confusion and weakness occur. Skin changes such as palm and sole scaling, stomatitis, glossitis, keratitis, eczema and classical alopecia and Mee's lines on the nails typically occur within two weeks of continual exposure. Treatment is with Prussian Blue which is administered orally at a dose of 250-300mg/kg/day (approximately 10g twice daily for an adult) and this complexes with thallium and is excreted in the faeces.

Thallium poisoning is often diagnosed late or not at all due to a low index of suspicion; indeed it is often misdiagnosed as Guillain-Barre syndrome due to the glove and stocking distribution of sensory symptoms and patients may receive intravenous steroids or immunoglobulins unnecessarily. In Guillain-Barre syndrome (GBS), classically there is ascending paralysis that is not seen in thallium poisoning and typically sensory symptoms in GBS are of numbness not hyperalgesia.

Thallium poisoning has also been mistaken for neurosyphilis and severe vitamin B deficiency. While neurosyphilis shares symptoms of confusion, fluctuating mood and palmar lesions as well as neuropathy, abdominal symptoms are usually not present and the diagnostic alopecia is absent in syphilis. Similarly, conditions such as Pellagra (niacin deficiency) and beriberi (thiamine deficiency) may share similar features with thallium poisoning such as alopecia, skin changes and mood disturbances, a painful neuropathy is less common; indeed sensory loss is more common in the extremities. Also in these conditions, the prime pathological aetiology is dietary deficiency and the history gives no suggestion this is an issue in this case.

Question 12 of 138

A 72-year-old male was admitted with 72 hours of progressive ascending motor weakness associated with palpitation and back pain. A diagnosis of Guillain-Barre syndrome was made and he was started on intravenous immunoglobulin (IVIg) with intensive monitoring on the neurology ward. You are asked to review the bloods of the patient on the fourth of five days of IVIg treatment. You note his sodium to have changes as follows: Na 145 → 141 → 134 → 130 → 126. The patient reports 'feeling better after the drip', denies any cough, shortness of breath, diarrhoea, nausea or vomiting. On examination, he appears comfortable in bed, JVP 2/3 cm above the angle of Louis, has warm peripheries and capillary refill time of 2 seconds. His chest is clear on auscultation, cardiovascular examination unremarkable and neurological examination unchanged from admission.

What is your management plan?

A. No action
B. Intravenous fluid
C. Fluid restrict to 1.5 litres
D. Contact endocrinology team regarding possible syndrome of inappropriate anti-diuretichormone (SIADH)
E. Contact intensive care unit regarding escalation and intubation

A. No action

The patient has received four days of intravenous immunoglobulin (IVIg), which is a protein and hence likely to distort the measurement of sodium, producing a pseudohyponatraemia. This is also observed in patients with high serum concentrations of triglycerides, cholesterol and in those with high serum protein such as myeloma patients. This patient is clinically euvolaemic. No action is required.

Question 13 of 138

A 63-year-old man is commenced on anti-tuberculosis therapy, after presenting to the hospital with a history of several months of weight loss, shortness of breath and a productive cough which was sometimes accompanied by haemoptysis.

A chest x-ray had revealed apical consolidation and sputum was positive for acid-fast bacilli, following which a diagnosis of tuberculosis was then made. His only past medical history includes a diagnosis of asthma for which he takes PRN salbutamol and Seretide (25 micrograms of salmeterol and 125 micrograms of fluticasone propionate) inhaler twice daily.

A week after being diagnosed, he is admitted to the acute medical unit with shortness of breath and bilateral expiratory wheeze. His observations reveal a temperature of 37.1ºC, respiratory rate of 27 breaths per minute and oxygen saturations of 94% on room air. Oxygen is commenced via a face mask. A chest x-ray reveals the same apical consolidation as before, with no new features.

What is the most appropriate next step in her treatment?

A. Continue the tuberculosis medication at current dose and increase the steroid dose
B. Continue the tuberculosis medication at current dose and decrease the steroid dose
C. Reduce the tuberculosis medication and increase the steroid dose
D. Stop the tuberculosis medication and increase the steroid dose
E. Continue the tuberculosis medication at current dose and keep the steroid dose the same

A. Continue the tuberculosis medication at current dose and increase the steroid dose

Rifampicin increases the metabolism of corticosteroids, thus patients who are on long-term steroids (as in this case) should receive a higher dose of corticosteroids when starting tuberculosis therapy.

Question 14 of 138

A 27-year-old man presents to his GP with generalised itchiness. This has been ongoing for some weeks and he has tried a number of creams over the counter. The GP provided a steroid cream and chlorphenamine tablets which helped initially but he is now finding he cannot sleep due to the itching. He has become tired and has been late to work as a fitness instructor several times. He denies alcohol use and is an ex smoker. His GP decides to perform some blood tests.

On examination, this well built gentleman looks well with scleral icterus present. There are numerous scratch marks across his torso and he has mild gynaecomastia. His abdomen is soft with no palpable masses. There are no spider naevi. His ankles are soft with no oedema. He has no rashes.

He mother has ulcerative colitis with liver complications and he has no past medical history.

Hb	180 g/l	Na +	145 mmol/l	Bilirubin 45 µmol/l
Platelets	459 * 10 /l 9	K +	3.0 mmol/l	ALP 189u/l
WBC	9.8 * 10 /l 9	Urea	4.5 mmol/l	ALT 61 u/l
Neuts	7.2 * 10 /l 9	Creatinine	95 µmol/l	γGT 213 u/l
Lymphs	1.2 * 10 /l 9	Albumin	45 g/l	
Anti smooth muscle antibody	negative	Anti liver kidney antibody	negative	

What is the likeliest cause of his deranged bloods?

A. Primary biliary cirrhosis
B. Alcoholic fatty liver disease
C. Primary sclerosing cholangitis
D. Anabolic steroid
E. Wilson's Disease

D. Anabolic steroid

Anabolic steroids are a cause of cholestasis

This gentleman presents with a symptomatic LFT rise. The ALP is raised out of proportion to the ALT making this a cholestatic picture which rules out AFLD and Wilson's disease. He has a family history for autoimmune disease but his antibodies are negative making PBC less likely. He is a well built gentleman and works in the fitness industry. Anabolic steroid use is highly prevalent especially with easy access through online sales. This gives an isolated cholestasis but can also give a polycythaemia through erythropoeisis and causes hypernatraemia and hypokalaemia. This should be discussed with the gentleman.

Question 15 of 138

A 29-year-old man is brought to the emergency department after the sudden onset of severe leftsided chest pain.

His observations are taken as follows: temperature 37.2ºC, blood pressure 165/92 mmHg, pulse 129/min and regular, and respiratory rate 20/min. On examination, he is anxious, diaphoretic and agitated. His nasal mucosa is atrophic, and both pupils are dilated. On auscultation, heart sounds are regular with no murmurs or rubs, and the lungs are clear.

An initial ECG shows ST-segment elevation in leads V4-V6. A chest x-ray is normal. His laboratory studies show:

Reference range		
Haemoglobin	165 g/L	Men: 135-180 g/L Women: 115-160 g/L
White blood cells	8.5 * 10 /l 9	4.0-11.0 * 10 /L9
Platelets	310 * 10 /l 9	150-400 * 10 /L9
Sodium	139 mmol/L	135-145 mmol/L

Potassium	4.5 mmol/L	3.5-5.0 mmol/L
Urea	3.5 mmol/L	2.0-7 mmol/L
Creatinine	94 umol/L	55-120 umol/L
Troponin T	32 ng/L	<14 ng/L

Which of the following treatment options must be avoided in this patient?

A. Aspirin
B. Diazepam
C. Diltiazem
D. Labetalol
E. Nitroglycerin

D. Labetalol

Cocaine toxicity - avoid beta-blockers

This patient is presenting with acute cocaine toxicity complicated by myocardial ischaemia. Hints pointing towards cocaine toxicity are excessive sympathetic stimulation, including tachycardia, dilated pupils and sweating. Cocaine toxicity can cause coronary artery vasospasm, giving rise to myocardial ischaemia. Troponin T levels are raised in myocardial ischaemia. The treatment of myocardial ischaemia in patients with and without cocaine intoxication is similar other than one important distinction: **patients with cocaine intoxication are not given beta-blockers**. The rationale behind these guidelines is that beta-blockage can lead to unopposed alpha-adrenergic stimulation and worsening coronary vasoconstriction.

Aspirin is routinely used to treat patients with myocardial infarction, especially those with cocaine intoxication. Cocaine can induce thrombus formation by activating platelets, stimulating their aggregation, and increasing thromboxane production. Patients with aortic dissection, another common complication of cocaine intoxication, should not be given aspirin. This patient's chest xray is normal, and his presentation does not point towards aortic dissection, making aspirin a recommended drug in this case. Aortic dissection presents with severe chest pain radiating to the back or between the shoulder blades. The chest x-rays in such cases often show a widened mediastinum.

Patients with cocaine intoxication often present with psychomotor agitation. In such patients, like this one, IV diazepam is given until the agitation is controlled.

Calcium-channel blockers (diltiazem and verapamil) are used as adjunctive therapy in patients who continue to have ischaemic symptoms despite using nitroglycerin. It is not contraindicated in patients with acute cocaine intoxication.

Sublingual nitroglycerin is one of the first drugs administered to patients presenting with ischaemic chest pain. Both nitrates and calcium channel blockers are beneficial for cocaineinduced coronary artery vasoconstriction.

Question 16 of 138

A 28-year-old woman was admitted to the Emergency Department drowsy and unwell after a suspected suicide attempt at home. The patient was under the care of the community psychiatry and had been receiving treatment for schizoaffective disorder. There was no other known past medical history. The paramedics bringing the patient to hospital also had brought the medications they found in the patient's home including quetiapine, levomepromazine, zopiclone and oxazepam. The paramedics reported that they had not seen evidence of empty medication packets or blister packs at the patient's home.

Initial assessment of the patient was unremarkable except for reduced consciousness level. Basic observations at presentation are listed below.

Blood pressure: 150/79 mmHg Heart rate: 89 bpm Respiratory rate: 20 / min O2 saturations (15 L O2):100 % Glasgow coma score: M5 V3 E2 Blood glucose: 7.0 mmol / L Temperature: 36.8ºC

Results from an arterial blood sample (15 L O2) were as follows.

pH	7.19
PaCO2	3.3 kPa
PaO2	21.2 kPa
Bicarbonate	8.9 mmol / L (reference 20.0-26.0)

Sodium	142 mmol / L
Potassium	3.6 mmol / L
Calcium	2.13 mmol / L (reference 2.20-2.60)
Chloride	110 mmol / L (reference 99-108)
Urea	5.2 mmol / L
Creatinine	110 micromol / L
Lactate	26 mmol / L
Plasma osmolality	380 mmol / Kg (reference 280-295)
Haemoglobin	12.0 g / dL

Based on the above blood results, what is the most likely cause of the patient's reduced consciousness level?

A. Ethanol intoxication
B. Benzodiazepine overdose
C. Ethylene glycol intoxication
D. Quetiapine overdose
E. Chronic renal failure

C. Ethylene glycol intoxication

The patient is unwell with a severe lactic acidosis. However, there is no clinical evidence for conditions that may cause this state (such as shock, hypoxia, sepsis or lactate-inducing drugs). Calculation of the anion gap and osmolality gap provide further clues to the likely cause.

Anion gap = Sodium - (Chloride + Bicarbonate) = 23.1 mmol / L (reference 5-11)

Osmolality gap = Measured osmolality - ((2*Sodium) + Urea + glucose)) = 83.8 mmol / Kg (reference < 6)

A severe lactic acidosis may cause a small osmolality gap (< 10 mmol / Kg) but is not consistent with the high value in this case). Possible causes of a high osmolality gap include pseudohyponatraemia (in extreme hyperproteinaemia or hyperlipidaemia), chronic renal failure or excess of toxic alcohols or glycols.

However, in the case of ethanol intoxication or pseudohyponatraemia the osmolality gap is not associated with a metabolic acidosis. Given that the blood results do not suggest evidence of chronic renal failure, the most likely cause is ethylene glycol intoxication. Testing for serum levels of methanol and ethylene glycol would confirm the diagnosis. Appropriate treatment is with ethanol and haemodialysis.

There is no evidence from the collateral history that the patient had taken an overdose of her prescription medications and this would not explain the metabolic abnormalities.

Question 17 of 138

A 17-year-old girl is brought to the Emergency Department by ambulance after her drink was 'spiked' at a party. Her friends recall her complaining that she felt like she was having an 'out of body experience' and that everything she touched seemed artificial. Over the next hour, she became increasingly distressed, slurring her words and appearing off-balance when walking.

Her past medical history is unremarkable. Her friends state that she does not usually partake in recreational drug use and had only consumed 2 or 3 alcoholic drinks during the course of the evening.

Examination reveals a slim young female with a temperature of 37.3, a pulse of 113bpm and a blood pressure of 137/76mmHg. Her pupils measure 5mm in diameter and she is GCS 14/15 due to persistent confusion. Her chest is clear and her heart sounds are unremarkable. Her abdomen is soft and non-tender. Neurological examination reveals bi-directional nystagmus and truncal ataxia with no focal weakness. Her deep tendon reflexes are brisk and symmetrical.

Which drug is she most likely to have been exposed to?

A. Ecstasy
B. Methoxetamine
C. Cocaine
D. Gamma-hydroxybutyric acid (GHB)
E. Nexus (2C-B)

B. Methoxetamine

Both ecstasy and cocaine could cause the tachycardia, hypertension, mydriasis and marginally elevated temperature described in the above scenario. The 'dead giveaway' however, is the presence of dissociative symptoms in combination with an acute cerebellar syndrome, pointing strongly towards methoxetamine intoxication.

Methoxetamine is an analogue of ketamine; a dissociative anaesthetic that acts as a NMDA receptor antagonist. Its effects are typical of the adverse effects of ketamine and include tachycardia, hypertension, mydriasis, confusion and agitation. Nystagmus, dysarthria, and ataxia can be severe and persist for several days.

GHB is a relatively short-acting CNS depressant that causes euphoria followed by coma. Patients can often be roused by external stimuli before returning to a comatose state. Rapid, spontaneous recovery can occur and patients have been known to extubate themselves.

Nexus (2C-B) is a synthetic phenylethylamine with psychoactive and stimulant effects. It is typically snorted or ingested with the former typically causing intense nasal pain. Its effects on the CNS include euphoria, synaesthesia, hallucinations and increased visual and tactile sensation, and probably occur due to antagonism of the 5HT2 receptor. Nexus (2C-B) is also an alpha-1 receptor agonist and its use can lead to ischaemic complications.

Question 18 of 138

A 35-year-old woman is referred to oncology. She has recently been diagnosed with metastatic breast cancer, which is HER2 positive. She has no other past medical history. She does not smoke or drink alcohol. She is an artist and lives with her husband and two young children.

She is commenced on trastuzumab.

The concurrent administration of which medication or medications should be avoided?

A. Carboplatin and paclitaxel
B. Docetaxel and cyclophosphamide
C. Doxorubicin and cyclophosphamide
D. Pertuzumab
E. Pertuzumab and hyaluronidase-zzxf

C. Doxorubicin and cyclophosphamide

The combination of trastuzumab + anthracyclines (both often used for breast cancer) is particularly cardiotoxic

Doxorubicin and cyclophosphamide is the correct answer. This combination is particularly cardiotoxic and concurrent administration of trastuzumab and anthracyclines (doxorubicin) is not recommended.

Carboplatin and paclitaxel are not correct. This is a possible combination of chemotherapeutic agents that could be used in conjunction with trastuzumab to treated metastatic HER2 positive breast cancer.

Docetaxel and cyclophosphamide is not the right answer. This is a possible combination of chemotherapeutic agents that could be used in conjunction with trastuzumab to treated metastatic HER2 positive breast cancer.

Pertuzumab is incorrect. Research shows that adding pertuzumab to trastuzumab and chemotherapy as part of first-line therapy for HER2-positive metastatic breast cancer improves mortality.

Pertuzumab and hyaluronidase-zzxf is incorrect. This can be combined with trastuzumab to be administered as a single medication combination and is used in the treatment of metastatic HER2 positive breast cancer.

Question 19 of 138

A 19-year-old woman is referred to the acute medical team after presenting to the emergency department. The patient reports taking an overdose of paracetamol that evening, following an argument with her mother. She immediately regretted taking the overdose and asked her mother to take her to the hospital. She is now very upset and anxious that she has caused herself serious harm and is requesting treatment.

The patient took just under two packets of 500 mg paracetamol tablets (an estimated 28 tablets), 40 minutes prior to presentation to hospital. She denied any history of mental or physical health problems, and had never taken an overdose before. She took no regular medications and did not drink alcohol.

Examination revealed an alert but anxious young woman with observations within normal physiological parameters, and no abnormal physical signs.

Haemoglobin	128 g / dL
Mean cell volume	95.0 fl
White cell count	8.4 x 10>3 / microlitre
Platelets	198 x 10>3 / microlitre
Urea	4.3 mmol / L
Creatinine	67 micromol / L
Sodium	137 mmol / L
Potassium	4.3 mmol / L
Albumin	45 g / L (reference 35-50)
Alkaline phosphatase	56 U / L (reference 35-100)
ALT	34 U / L (reference 3-36)
Bilirubin	15 micromol / L (reference < 26)
International normalised ratio	1.1 (reference 0.8-1.2)

What is the most appropriate management of the patient's paracetamol overdose?

A. Treatment with activated charcoal if 4-hour paracetamol level > 100 mg / L
B. Immediate treatment with acetylcysteine
C. Haemodialysis
D. Immediate treatment with methionine

E. Immediate treatment with activated charcoal

E. Immediate treatment with activated charcoal

The patient has taken a large (> 10 g) paracetamol overdose and then presented to the hospital very promptly (< 2 hours). For such patients, activated charcoal should be considered as a first-line therapy, if the risk of aspiration is low. In practice, this means that the patient is either fully compliant with treatment (as in this example) or is intubated.

No randomised controlled trials have examined the role of activated charcoal in paracetamol overdose. However, a cohort study of patients who ingested > 10 g paracetamol, found that treatment with charcoal within two hours was associated with reduced need for acetylcysteine. If this link is assumed to be causal, then number needed to treat to avoid further treatment was 7. In addition, two randomised controlled trials have shown that activated charcoal is very safe.

The use of activated charcoal also increases elimination of anti-epileptic and contraceptive medications, and precautions should be taken against the short-term risk of treatment failure.

Treatment decisions regarding paracetamol poisoning antidote (acetylcysteine or methionine) should be guided by the 4-hour paracetamol level and reference to paracetamol poisoning nomogram. There is no benefit to treatment prior to the 4-hour paracetamol level result; hepatotoxicity following paracetamol toxicity does not occur until glutathione stores are depleted, around 8 hours after the overdose.

Haemodialysis removes only a small proportion of ingested paracetamol, as well as removing acetylcysteine. Therefore, it is not a therapeutic option in paracetamol overdose.

Question 20 of 138

A 78-year-old man with a history of atrial fibrillation, diabetes, hypertension and peripheral neuropathy was referred to the anticoagulation clinic by his General Practitioner due to difficulty controlling the international normalised ratio (INR). He is currently on 12 mg of warfarin and the dose had to be repeatedly increased as his INR has been in the subtherapeutic range in the last month.

Which one of the following medication may be responsible for the above?

A. Omeprazole
B. Carbamazepine
C. Cimetidine
D. Valproate
E. Ciprofloxacin

B. Carbamazepine

Carbamazepine is a P450 enzyme inductor

Carbamazepine is a hepatic enzyme inducer and may reduce the effectiveness of warfarin, whereas the others are enzyme inhibitors.

Others to remember are:

P450 enzyme inducers	P450 enzyme inhibitors
Phenytoin	Amiodarone
Rifampicin	Ketoconazole
Alcohol	Erythromycin
Isoniazid	
Sulphonamides	
Allopurinol	

Question 21 of 138

A 62-year-old man presents to the emergency department after drinking a small amount of methanol in an attempt to end his life. His observations are as follows: heart rate 110 beats per minute, blood pressure 130/85 mmHg, respiratory rate 22 breaths per minute, temperature 37.5ºC, and SpO2 95% on air. A fundoscopic examination is normal.

Blood results are as follows:

Hb	120 g/L	Male: (135-180)Female: (115 - 160)
Platelets	165 * 10^9/L	(150 - 400)
WBC	14.8 * 10^9/L	(4.0 - 11.0)
Na^+	135 mmol/L	(135 - 145)
K^+	4.2 mmol/L	(3.5 - 5.0)
Urea	2.8 mmol/L	(2.0 - 7.0)
Creatinine	68 µmol/L	(55 - 120)
CRP	22 mg/L	(< 5)
Glucose	4.2 mmol/L	(3.9 - 5.6)
Serum osmolarity	305 mOsm/kg	(275 - 295)

You start the patient on fomepizole. What additional treatment is required?

A. Activated charcoal
B. Ethanol
C. Folic acid
D. Folinic acid
E. Haemodialysis

D. Folinic acid

Folinic acid can be used in methanol poisoning to reduce ophthalmological complications

The patient has clinical and biochemical features of methanol toxicity.

The osmolar gap = osmolality (measured) - osmolarity (calculated). A normal osmolar gap should be < 10.

The calculated osmolarity in this case = (2 x [Na+]) + [glucose] + [urea]) = 277 mOsm/kg. Therefore the osmolar gap = 305 - 277 = 28 mOsm/kg.

The patient thus has a high osmolar gap which indicates the presence of an abnormal solute present in significant amounts.

Causes of a high osmolar gap include:

- Mannitol
- Methanol
- Ethylene glycol

Folinic acid is correct. The clinical and biochemical features support a diagnosis of methanol poisoning. For patients with methanol intoxication, early administration of fomepizole will minimise the further metabolism of methanol and the development of clinical and metabolic complications. The addition of folinic acid can help reduce the accumulation of toxic byproducts in the eyes thereby reducing the associated ophthalmological complications.

Activated charcoal is incorrect. Activated charcoal is of no use since it does not adsorb significant quantities of methanol.

Ethanol is incorrect. Ethanol results in competitive inhibition of methanol thereby slowing down formic acid formation. Fomepizole is the preferred antidote since, unlike ethanol, it does not require regular monitoring of blood concentrations or cause inebriation.

Folic acid is incorrect. It is folinic acid rather than folic acid which has the best evidence base to reduce the ophthalmological complications of methanol.

Haemodialysis is incorrect. Haemodialysis has a role in severe or refractory cases. Indicators of severe disease include a methanol concentration greater than 500 mg/L, visual disturbance, features of CNS toxicity, severe metabolic acidosis and renal failure.

Question 22 of 138

A 57-year-old man attends neurology clinic. He has recently been diagnosed with Parkinson's disease and has agreed to start treatment with cabergoline as monotherapy. He has had a full examination, lung function tests, routine blood tests, chest X-ray and an echocardiogram.

Apart from a clinical review, what investigation is most important to arrange regularly to monitor for complications?

A. Chest X-ray
B. FBC
C. U&E
D. Echocardiogram
E. LFT

D. Echocardiogram

The correct answer is echocardiogram. Cabergoline is a dopaminergic drug which can be used to treat Parkinson's disease. Dopaminergic drugs are associated with fibrotic reactions including pulmonary, retroperitoneal and pericardial fibrosis, but cabergoline also assocaited with development of new valvulopathy. The BNF recommends establishing a baseline set of investigations and then to monitor clinically for dyspnoea, persistent cough, chest pain, cardiac failure and abdominal pain or tenderness.

Question 23 of 138

A 52-year-old man is brought into the emergency department resuscitation room. He was found collapsed by his wife 40 minutes ago. He was last seen 3 hours prior. His wife is present, who informs you his past medical history consists of depression, osteoarthritis, sciatica and ischaemic heart disease.

His Glasgow coma scale was 13/15. Nystagmus was noted.

Observations: respiratory rate 28 breath/minute, oxygen saturations 100% on 15 litres of oxygen, heart rate 112 beats/minute, blood pressure 142/92mmHg. Electrocardiogram (ECG) is sinus tachycardia, but otherwise normal.

An arterial blood gas is done and bloods are sent to the lab.

Hb	143 g/L	Male: (135-180)Female: (115 - 160)
Platelets	383 * 10 /L $_9$	(150 - 400)
WBC	13.2 * 10 /L $_9$	(4.0 - 11.0)
Na +	140 mmol/L	(135 - 145)
K +	5 mmol/L	(3.5 - 5.0)
Urea	9 mmol/L	(2.0 - 7.0)
Creatinine	135 µmol/L	(55 - 120)
CRP	14 mg/L	(< 5)
Glucose	6 mmol/L	(4-7)
Osmolality	322mOsm/kg	(275 - 295)
pH	7.31	(7.35-7.45)
pO2	26 kPa	(10-14)
pCO2	2.9 kPa	(4.5-6)
HCO3 -	12 mmol/L	(22-28)
Cl -	95 mmol/L	(95-105)

What is the most likely cause of this man's presentation?

A. Amitriptyline
B. Citalopram
C. Codeine
D. Ethylene glycol
E. Mirtazapine

D. Ethylene glycol

Metabolic acidosis with a high anion gap and high osmolar gap is seen in ethylene glycol poisoning

This question involves toxicology and utilising the anion gap and osmolar gap helps reach the diagnosis. The presentation of reduced GCS is non-specific and can occur in many toxidromes. From his observations, we can see that he has a high respiratory rate, despite normal saturations.

Assessing his arterial gas, he has a partially compensated metabolic acidosis. His high respiratory rate is helping to blow off CO2 to compensate. The anion gap can be calculated as 33 or 38 (depending on whether you include potassium in your calculation). This is high.

When osmolality is stated in a question regarding toxicology it is often worth calculating the osmolar gap - the difference between measured and calculated osmolality. Calculated osmolality is 2Na + glucose + urea. The normal range of the osmolar gap is less than 10. In this case, it's 27.

Common causes of a high osmolar gap in toxicology are ethylene glycol, ethanol and methanol. Out of these three, only ethylene glycol is an option and so this is the most likely answer.

Paracetamol and selective serotonin reuptake inhibitors may cause high anion gap metabolic acidosis indirectly by causing acute kidney injury, but are not typically associated with a high osmolar gap.

Amitriptyline is a tricyclic. Within 1-2 hours of onset of toxicity they tend to cause hypotension and tachycardia, classically broad-complex. This progresses to seizures and reduced level of consciousness. This patient is not hypotensive and the ECG is not broad-complex so this is less likely.

Codeine is an opioid and so you expect to find respiratory depression if this was the case in this patient.

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Question 24 of 138

You are in the general medical clinic. A 37-year-old male has been referred by his GP with dyspnoea. His symptoms started about 5 months ago with dyspnea mostly on exertion which is progressively getting worse. He also has a dry cough for last 2 months. There is no orthopnoea or paroxysmal nocturnal dyspnoea. There is also no history of chest pain. His past history includes Hodgkin's lymphoma which was successfully treated 2 years ago with ABVD regimen. He is currently not taking any medications. He works in a printing press and does not smoke. On examination, there is no raised JVP or ankle oedema. Auscultation revealed normal heart sounds and bilateral fine crackles. Results of his investigations are as follows:

Hb	125 g/l	Na +	139 mmol/l	Bilirubin	12 µmol/l
Platelets	188 * 10 /l $_9$	K +	4.1 mmol/l	ALP	95 u/l

WBC	6.7 * 10 /l 9	Urea	4.3 mmol/l	ALT	15 u/l
Neuts	3.5 * 10 /l 9	Creatinine	86 µmol/l	γGT	45 u/l
Lymphs	2.2 * 10 /l 9	ESR	25 mm/hr	Albumin	34 g/l
Eosin	0.3 * 10 /l9				

Chest x-ray normal

What is the next best investigation for evaluation of this patient?

A. Bronchoalveolar lavage (BAL)
B. Mantoux test
C. Echocardiogram
D. Chest ultrasound
E. Pulmonary function tests

E. Pulmonary function tests

Bleomycin may cause pulmonary fibrosis

History is suggestive of interstitial lung disease most likely secondary to bleomycin and pulmonary function tests will aid in diagnosis by showing a restrictive pattern. Bleomycin is part of ABVD

regimen and known to cause pulmonary fibrosis. That's why baseline pulmonary function testing is important in patients receiving bleomycin.

BAL is an invasive procedure with non-specific findings in cases of drug-induced interstitial lung disease. Non-invasive investigations like pulmonary function tests and high-resolution CT (HRCT) are carried out first before proceeding for invasive tests.

Mantoux test can help exclude tuberculosis but tuberculosis is not the most likely diagnosis in the given scenario.

An echocardiogram can be used to exclude dyspnoea due to heart failure, however, there is no history of orthopnoea or paroxysmal nocturnal dyspnoea, and no findings of raised JVP, abnormal heart sounds or ankle oedema.

Chest ultrasound is not helpful unless a pleural effusion was suspected.

Question 25 of 138

A 48-year-old farmer is found collapsed in his workshop having left a suicide note and consumed unknown weedkillers and insecticides from his store. On arrival in the Emergency department, his Glasgow coma scale is 10, pupils are pinpoint and poorly reactive. His blood pressure is 100/70 mmHg, his pulse is 88 beats per minute and regular. He moans incomprehensibly when you palpate his abdomen. You are concerned about the possibility of a paraquat overdose.

How can you screen for it?

A. Serum NADPH testing
B. Serum free fatty acid testing
C. Urine copper sulphate testing
D. Urine ketone testing
E. Urine dithionate testing

E. Urine dithionate testing

Qualitative confirmation of paraquat in the urine is easily performed in any laboratory. The main role of this test is to confirm or exclude exposure to paraquat. The presence of paraquat is confirmed by a colour change noted after the addition of a dithionite solution. The test is usually positive within six hours of a large ingestion and remains positive for several days. It's important not to wait for the results of the test before administering oral Fuller's earth.

The other options have no value in the evaluation of paraquat poisoning. Serum free fatty acids may rise where there is mitochondrial dysfunction, as may urinary ketones. Serum lactate is used in evaluation of metabolic dysfunction associated with cyanide poisoning.

Question 26 of 138

A 35-year-old man was assessed in endocrinology clinic after being referred by his GP for advice on management of the consequences of the patient's use of anabolic steroids. The patient reported that he had been using anabolic steroids intermittently for the previous 10 years to augment his weight-training regime. Having recently become aware of the potential adverse health consequences , the patient now wished to know how he could safely stop his anabolic steroid use.

The patient described his pattern of anabolic steroid use: typically, he had been taking a 'cycle' of one or more oral synthetic testosterone derivatives for between 6-12 weeks, prior to a 3-4 week break in his use of steroids. The patient stated this strategy was an attempt to minimise his risk of side effects. The patient had not received any medical supervision of his anabolic steroid use, relying instead on discussions with friends at his gym and information from online forums. The patient declined to disclose the source from which he had obtained his supply of medications.

The patient stated that he believed his anabolic steroid use had contributed to his male pattern baldness and also had caused intermittent breakouts of acne on his chest and face. The patient was not aware of any other symptoms related to his steroid use, although stated that he was concerned about possible lasting cardiac side effects. The patient had no other significant medical history and took no other regular medications. He did not consume cigarettes, alcohol or recreational drugs.

General examination of the patient revealed a muscular and lean adult male in apparent good health. A cardiovascular system examination noted a forceful but not displaced apex beat. Mild gynaecomastia was present but gastrointestinal examination was otherwise unremarkable. Using an orchidometer the patient's testicular volume was estimated as 16 ml. Although anxious about the possible health consequences of his anabolic steroid use, the patient did not seem to be significantly depressed or anxious.

Haemoglobin	173 g / dL
Sodium	148 mmol / L
Potassium	3.6 mmol / L
Alkaline phosphatase	135 U / L (reference 35-100)
ALT	32 U / L (reference 3-36)
Bilirubin	20 micromol / L (reference < 26)
Prolactin	469 mU / L (reference 80 - 400)
Luteinising hormone	0.9 IU / L (reference 1.8 - 8.6)

Follicle-stimulating hormone	1.2 mU / ml (reference 1.5 - 12.4)
Fasting LDL cholesterol	4.1 mmol / L (reference < 3.0)
Fasting HDL cholesterol	1.0 mmol / L (reference > 1.2)
HbA1C	45 mmol / mol (reference < 42)

What is the appropriate advice to the patient regarding the safe cessation of anabolic steroid use?

A. Convert patient to prescribed testosterone replacement and taper over 1 year
B. Taper anabolic steroid use over period of 6 months
C. Stop immediately, tapered withdrawal not required
D. Taper anabolic steroid use over period of 6 weeks
E. Convert patient to prescribed testosterone replacement and continue lifelong

C. Stop immediately, tapered withdrawal not required

The patient has typical side effects and examination findings associated with long-standing anabolic steroid use. These include male pattern baldness, acne, gynaecomastia and testicular atrophy (typical adult male testicular volume around 25 mL). His blood tests also demonstrate typical abnormalities including erythropoiesis, hypernatraemia, hypokalaemia, cholestatic liver function tests, deranged lipid profile, evidence of elevated serum glucose and typical hormone profile abnormalities.

Question 27 of 138

A 50 year-old man presents to the medical assessment unit with a two day history of nausea and vomiting. His notes are currently not available to you, and the patient is unable to recall his medical problems. He is currently an inpatient at the local psychiatry facility.

On examination, he appears drowsy with an ataxic gait. Neurological examination reveals normal tone with generalised reduced power. He also has a coarse tremor when his hands are outstretched.

Cardiorespiratory examination reveals is unremarkable.

ECG- sinus arrhythmia at 90 beats per minute. T wave inversion in leads V1-V3.

Observations

- Blood pressure- 150/90 mmHg
- Heart rate- 90 beats per minut
- Respiratory rate- 16 breaths per minute
- Oxygen saturations- 99% on room air
- Temperature- 37.3°C

What is the most likely cause of his symptoms?

A. Lithium toxicity
B. Alcohol withdrawal
C. Serotonin syndrome
D. Neuroleptic malignant syndrome
E. Tricyclic antidepressant overdose

A. Lithium toxicity

Lithium salts are used in the prophylaxis and treatment of bipolar disorder, refractory depression and aggressive or self-harming behaviour.

Lithium has a narrow therapeutic range (0.5-1 mmol/l). The likelihood of toxicity increases with increasing serum lithium levels. Toxic effects are seen with a concentration of >2 mmol/l.

Very early features may be non-specific, such as apathy and restlessness

Early signs of toxicity (1-2 mmol/l)

- anorexia and vomiting
- ataxia and dysarthria
- nystagmus
- blurred vision
- coarse tremor drowsiness

Late signs of toxicity (>2mmol/l)

- coma
- seizures
- acute renal failure
- death

Serotonin syndrome is a drug reaction to serotonergic agents, and ranges from mild to severe. It is characterised by a triad of autonomic hyperactivity (hyperthermia, tachycardia), neuromuscular abnormality (tremor) and mental state changes (confusion, coma).

Neuroleptic malignant syndrome is a rare reaction to neuroleptic drugs, often following initiation or increase in dose. It is characterised by recent neuroleptic use, hyperthermia, rigidity and autonomic dysfunction.

Question 28 of 138

A 28-year-old man is brought into the emergency department by paramedics. He was found collapsed by his wife just outside the house. With the paramedics he was rousable and they initially suspected he was drunk.

On transfer, he has become more drowsy but is still talking on physical stimuli. Initial venous blood gas shows a metabolic acidosis with a high anion gap. Lab blood results show:

Hb	148 g/L	Male: (135-180)Female: (115 - 160)
Platelets	345 * 10 /L 9	(150 - 400)
WBC	12.5 * 10 /L 9	(4.0 - 11.0)
Na +	135 mmol/L	(135 - 145)
K +	5.2 mmol/L	(3.5 - 5.0)
Bicarbonate	18 mmol/L	(22 - 29)
Urea	12 mmol/L	(2.0 - 7.0)
Creatinine	148 µmol/L	(55 - 120)
Glucose	6.2 mmol/L	(4-10)
Osmolality	307 mOsm/kg	(275-295)
Paracetamol levels	<5 mg/L	(<5)
Salicylate levels	<5 mg/dL	(<5)

What is the most appropriate treatment?

A. Chlorpromazine
B. Cyproheptadine
C. Ethanol
D. Fomepizole
E. Haemodialysis

D. Fomepizole

Ethylene glycol toxicity management - fomepizole. Also ethanol / haemodialysis

Reduced consciousness is a non-specific symptom of poisoning. Looking at his blood tests show kidney injury, a mild inflammatory response and a high osmolality. The blood tests allow us to calculate whether there is an osmolar gap.

Calculated osmolarity = 2Na + glucose + urea

Calculated osmolarity = 2(135) + 6.2 + 12 = 288.2

Lab osmolality = 307

Osmolar gap = Osmolality (measured) - Osmolarity (calculated)
Osmolar gap = 307 - 288.2 = 18.8 (normal is <10)

There are several causes of an osmolar gap, including ethanol, isopropyl alcohol, methanol, ethylene glycol, and mannitol. **Fomepizole** is the treatment option used in the management of methanol or ethylene glycol poisoning which is likely to be the cause in this patient.

Chlorpromazine may be used in the management of serotonin syndrome. Although serotonin syndrome can cause metabolic acidosis, it would not explain the osmolar gap.

Cyproheptadine may be used in the management of serotonin syndrome. Although serotonin syndrome can cause metabolic acidosis, it would not explain the osmolar gap.

Ethanol is incorrect, as although it can be used in methanol and ethylene glycol poisoning, fomepizole is the superior antidote so is a better treatment if available. Ethanol has many issues as a treatment, including being difficult to dose and maintain appropriate levels.

Haemodialysis has a role in refractory cases of ethylene glycol toxicity. However, in this case, 1stline management should be tried first, and therefore this answer is incorrect.

Question 29 of 138

A 75-year-old man underwent a right hemicolectomy as radical treatment for a caecal adenocarcinoma. During the immediate post-operative period the patient was noted to be significantly confused and investigations to assess for a septic source were requested. Blood tests indicated new neutrophilia and thrombocytopenia. Noting that the patient had been started on enoxaparin for venous thrombosis prophylaxis on the first day after the operation, heparin induced thrombocytopenia antibodies were requested by the surgical junior team.

A specialist medical review was requested on day 6 post-operatively to assess the patient in the light of the results of these investigations. The patient was found to be back to his cognitive baseline and was starting to mobilise on the ward with the help of physiotherapy. Clinical examination was unremarkable except for the healing abdominal surgical incision.

Aside from his recent cancer diagnosis, the patient's past-medical history included only hypercholesterolaemia treated with simvastatin. He had no known allergies or drug sensitivities.

Blood result	Pre-operatively	Day 1 post-op	Day 4 post-op	Day 6 post-op
Platelets (x 10>3 / microlitre)	175	95	125	141
Neutrophils (x 10>3 / microlitre)	5.3	14.8	12.8	8.8
C-reactive protein (mg / L)	6	142	94	25
International normalised ratio	1.0	1.2	1.1	1.1

Chest x-ray: clear lung fields aside from minor bibasal atelectasis

Urine culture: no growth

CT abdomen: minimal abdominal free fluid, no evidence of focal collection, large bowel anastomoses intact

Heparin-induced thrombocytopenia antibodies: positive (moderate titre)

What is the likely cause for the patient's thrombocytopenia?

A. Heparin induced thrombocytopenia
B. Post-operative sepsis
C. Post-operative thrombocytopenia
D. Thrombotic thrombocytopenic purpura
E. Disseminated intravascular coagulation

C. Post-operative thrombocytopenia

Heparin induced thrombocytopenia (HIT) antibodies have a high false positive rate and their interpretation needs to take into account careful consideration of the entire clinical picture. Decision tools to assess the likelihood of HIT such as the Warkentin probability scale are useful in this setting. This provides a pretest probability score to assist with interpretation of HIT antibody results based on the extent of the thrombocytopenia, the timing of onset relative to heparin administration, thrombotic events and the presence or absence of other causes of thrombocytopenia.

In this case, there are few clinical features consistent with HIT and a low pretest probability score would be calculated. The combination of this and moderate titre positive HIT antibodies is considered to exclude a HIT diagnosis.

In this patients case a post-operative thrombocytopenia is the most likely cause in the absence of evidence for other possible answers. The patients elevated inflammatory markers are secondary to surgery with no septic source identified on investigation. TTP can cause thrombocytopenia associated with delirium, although the patients clinical and haematological recovery make this an unlikely cause. DIC is excluded by the normal prothrombin time and improving clinical picture.

Question 30 of 138

A 49-year-old woman has attended the clinic with a 3-month history of heat intolerance, weight loss, and intermittent diarrhoea.

She has a past medical history of paroxysmal ventricular tachycardia and type 2 diabetes mellitus. Her regular medications include amiodarone, metformin, and linagliptin.

On clinical examination, she is mildly tremulous with prominent skin capillaries. She is tachycardia but has no audible murmurs. A neck examination does not reveal a goitre.

Thyroid function tests are requested.

Free T4	42.1	11.0 - 23.0
TSH	0.05	0.35 - 5.00

Given the likely diagnosis, what is the best management option?

A. Carbimazole
B. Levothyroxine
C. Potassium perchlorate
D. Prednisolone
E. Propylthiouracil

D. Prednisolone

In amiodarone induced thyrotoxicosis type 2 the management is with corticosteroids rather than carbimazole

The above case describes a patient with signs and symptoms commonly associated with hyperthyroidism. In combination with ongoing treatment with amiodarone, the diagnosis of amiodarone-induced thyrotoxicosis (AIT) can be made. AIT can be sub-divided into type 1 and type 2. This should not be confused with amiodarone-induced hypothyroidism which is thought to be due to the high iodine content of amiodarone.

Type 1 AIT is caused by excessive iodine from amiodarone causing increased thyroid hormone synthesis, whereas AIT type 2 is related to destructive thyroiditis. The two variations can be clinically distinguished by the presence (AIT type 1) or absence (AIT type 2) of a goitre. Given that the patient in the above scenario has no clinical findings of a goitre, AIT type 2 can be diagnosed. This is treated with corticosteroids, such as prednisolone.

Carbimazole is a well-recognised anti-thyroid medications commonly used in hyperthyroidism. It acts by reducing T3 and T4 production via thyroid peroxidase. It is an option in the management of AIT type 1.

Levothyroxine is used for thyroid hormone replacement in hypothyroidism and can be used in amiodarone-induced hypothyroidism.

Potassium perchlorate is an oxidising agent used as an anti-thyroid agent in thyrotoxicosis. It acts by competitively inhibiting iodine uptake in follicular thyroid cells. Along with carbimazole, it is a management option in type 1 AIT.

Although a treatment option in hyperthyroidism, propylthiouracil is not used in the management of amiodarone-induced thyrotoxicosis.

Question 31 of 138

A 35-year-old woman presents to the emergency department with agitation and sweating. She was recently started on paroxetine for depression. She takes St John's Wort over the counter. There is no history of substance abuse.

On examination, various muscle groups have sudden, brief involuntary twitching. She is diaphoretic, agitated and confused. There is hyperreflexia. She has a heart rate of 124 beats per minute and a temperature of 40ºC.

An ECG demonstrates sinus tachycardia.

Given the likely diagnosis, what is the appropriate treatment?

A. Bisoprolol
B. Co-amoxiclav
C. Cyproheptadine
D. Dantrolene
E. Procyclidine

C. Cyproheptadine

Treatment of severe serotonin syndrome- cyproheptadine (5-HT2 receptor antagonist)

Cyproheptadine is correct. The patient has serotonin syndrome as a consequence of the interaction between St. John's Wort and paroxetine. Her presentation is clinically severe and she should be treated with cyproheptadine, which is a 5-HT2 receptor antagonist. Milder cases can be treated with benzodiazepines alone.

Bisoprolol is incorrect. Sinus tachycardia does not tend to need treatment with cardiovascular medications and the treatment should be against the underlying cause.

Co-amoxiclav is incorrect. While the patient has a fever, the clinical history of the recent combination of an SSRI and St. John's Wort along with myoclonus and hyperreflexia suggest serotonin syndrome rather than infection.

Dantrolene is incorrect. This is the treatment for malignant hyperthermia. This is also characterized by high fever and muscle spasms but occurs post exposure to inhalational anaesthetics or depolarising muscle relaxants.

Procyclidine is incorrect. This is an anti-cholinergic drug used in the treatment of drug-induced Parkinsonism.

Question 32 of 138

A 47-year-old man has been referred to the endocrinology clinic. He has a history of hypertension and bipolar disorder. He can recall his antihypertensives, lisinopril, and amlodipine, but cannot remember which medication he is on for his bipolar disorder.

Recently he has developed polydipsia and polyuria. His GP performed some blood tests and found elevated blood glucose. His HbA1c is sown below.

HbA1C	62 mmol/mol	42 - 48

What medication is the most likely cause?

A. Aripiprazole
B. Lithium
C. Olanzapine
D. Risperidone
E. Sertraline

C. Olanzapine

Long-term use of olanzapine is a significant risk factor for the development of type 2 diabetes mellitus

Although the pathophysiology is not known, it is recognised that **olanzapine** carries a significant risk for the development of type 2 diabetes mellitus, along with other antipsychotic agents. Olanzapine has an antagonistic effect on muscarinic acetylcholine receptor 3 which has been found to be crucial in glucose-induced insulin resistance. Olanzapine also leads to hyperglycaemia through its antagonistic effects on dopamine 2 receptors (increased appetite), H1 histamine receptors (insulin resistance) and 5-HT2A serotonin receptors (insulin resistance).

As with all antipsychotic medications, **aripiprazole** can cause hyperglycaemia, however, it carries a small risk, compared to olanzapine which carries significant risk.

Long-term use of **lithium** can increase blood glucose levels. However, it carries a risk of the development of diabetes insipidus rather than diabetes mellitus.

Risperidone carries an intermediate risk of weight gain and the development of diabetes. The risk of diabetes development is higher with olanzapine.

According to the BNF, **sertraline** should be used with caution in people with known diabetes. This is due to its increased effect on insulin secretion thus increasing the risk of hypoglycaemia. Sertraline has not been shown to increase the risk of type 2 diabetes.

Question 33 of 138

A 20-year-old man is brought to the Emergency Department by the Police with agitation and aggression. He is accompanied by a friend, who says they had been out celebrating his brother's birthday and that the patient may have 'snorted something'.

He recalls the patient complaining of intense nasal pain for some time after he allegedly took the substance, but it soon settled and he seemed in good spirits; talking enthusiastically about seeing vivid lights and waves emanating from the club's speakers. As the night went on, however, he became increasing disturbed; shouting at other clubgoers and complaining about hearing threatening voices.

On examination, his temperature is 39.2ºC. His pulse is 117bpm and his blood pressure is 181/98mmHg. He is difficult to examine due to intermittent aggression, and he has lashed out on several occasions.

In order to ensure his own safety, a decision to sedate the patient is made. The patient is successfully restrained and cannulated but suffers a tonic-clonic seizure shortly afterward. He is given 4mg IV lorazepam, but then suffers a cardiac arrest from which he cannot be resuscitated.

Which drug is most likely to be responsible?

A. Lysergic acid diethylamide (LSD)
B. Nexus (2CB)
C. Spice
D. Gamma-hydroxybutyric acid (GHB)
E. Methoxetamine

B. Nexus (2CB)

Nexus is a designer drug belonging to the 2C family; a group of ring-substituted phenethylamines similar in structure to 3,4-methylenedioxy-N-methylamphetamine (MDMA). 2C refers to the presence of two carbon atoms between the amine group and the benzene ring in the chemical structure.

Common features of 2C intoxication include agitation, aggression, and hallucinations, often complicated by the presence of serotonergic or sympathomimetic toxidromes. It is possible to increase the hallucinogenic effects of these drugs by placing substituents on the aromatic ring at positions 2, 4, or 5. Synthesis of 2C drugs by amateur chemists can, therefore, lead to the inadvertent formation of dangerously potent agents.

2Cs are ingested orally or by insufflation, with the latter being extremely painful. At low doses, 2Cs have stimulating effects and cause increased visual, auditory and tactile sensations. Hallucinations occur at moderate doses, whilst high doses can cause frightening hallucinations, hypertension, tachycardia, hyperthermia, seizures and excited delirium.

Patients with excited delirium often exhibit aggression, hyperactivity, hyperthermia and, as in this case, unexpected cardiac arrest. It is thought to relate to excessive dopamine release in the mesolimbic pathways and has been linked to a number of 2C-related deaths.

LSD is also a hallucinogen, but it would not cause the hypertension or hyperthermia evident in the vignette. Fatal overdose is rare.

GHB is a CNS depressant that causes euphoria followed by coma. It has a short duration of action and toxicity is often maximal at presentation.

Methoxetamine is a ketamine derivative that causes dissociative symptoms, tachycardia, hypertension, confusion, and occasionally an acute cerebellar syndrome. It does not typically cause hallucinations.

Spice is a synthetic cannabinoid. Like marijuana, spice is usually smoked or ingested orally. Intoxication results in elevated mood, relaxation, and altered perception. Spice, however, is more likely than marijuana to cause hypertension, tachycardia, seizures and acute psychosis.

Question 34 of 138

A 74-year-old man presents increasing ankle swelling but is otherwise well. The swelling is uncomfortable and impacting on his quality of life. He takes amlodipine 10mg once daily (OD) and losartan 75mg OD. His blood pressure is 128/73 mmHg in clinic. His examination reveals pitting oedema in the ankles but nil else. He had a recent chest radiograph which was normal and an echocardiogram that showed good systolic function.

What action would you take?

A. Add furosemide 20mg once daily
B. Add spironolactone 25mg once daily
C. Fluid restrict to 1.5 litres a day
D. Reduce amlodipine to 5mg once daily
E. Prescribe compression stockings

D. Reduce amlodipine to 5mg once daily

This is likely a medication side effect given his normal echocardiogram. Amlodipine is heavily associated with ankle swelling. Therefore its dose should be reduced and if the blood pressure rises a further agent can be added.

There is no indication for diuretic therapy at this stage since there is no heart failure and the blood pressure is controlled.

Question 35 of 138

A 19-year-old female is admitted to the ward with reduced conscious level. Her mother found her at home lying on her bedroom floor. She has a past medical history of anxiety, depression, and hypothyroidism. On examination her heart rate is 110 beats per minute, blood pressure 124/80 mmHg, and respiratory rate is 36 breaths per minute.

Blood results are as follows:

Hb	122 g/l	Na +	134 mmol/l
Platelets	488 * 10 /l 9	K +	5.6 mmol/l
WBC	14.2 * 10 /l 9	Urea	10.8 mmol/l
Neuts	11.8 * 10 /l 9	Creatinine	158 µmol/l
Lymphs	1.8 * 10 /l 9	CRP	42 mg/l
Chloride	105 mmol/l		

An arterial blood gas is as follows:

PaO2	10.1 kPa
PCO2	2.2 kPa
pH	7.28
HCO3	12 mmol/l

What is the most likely diagnosis?

A. Addisonian crisis
B. Intracerebral hemorrhage
C. Morphine overdose
D. Encephalitis
E. Salicylate overdose

E. Salicylate overdose

Salicylate overdose can cause a mixed primary respiratory alkalosis and metabolic acidosis

The patient clearly has a metabolic acidosis as demonstrated by the low pH and low bicarbonate. With a primary metabolic acidosis we would expect the body to compensate by generating a respiratory alkalosis. Indeed this patient has hyperventilation and a low PCO2 indicating respiratory alkalosis. However it is important to remember that there is a limit for respiratory compensation. Even though respiratory compensation occurs relatively quickly, it can take up to twelve hours to reach maximal capacity.

The maximal respiratory compensation for metabolic disturbance can be calculated with:

pCO2 (maximal response in kPa) = (0.2 * HCO3) + 1

If the patient's pCO2 lies within 0.5 kPa of this calculated value, then the respiratory response is appropriate. If the pCO2 is higher then either the compensation is in a very early stage or the patient has a superimposed respiratory acidosis. In this case we would expect a maximal compensation of 3.4 kPa. The pCO2 value is actually less than this indicating we also have a superadded primary metabolic alkalosis.

When dealing with a metabolic acidosis it is also important to work out the anion gap which is very useful for identifying the cause. In this case the anion gap = (Na + K) - (Cl +HCO3) = 22.6. This is a high anion gap metabolic acidosis (HAGMA). Causes of HAGMA include ketoacidosis, uraemia, lactic acidosis and toxins (e.g. salicylates, ethylene glycol and methanol).

The combination of HAGMA with a superadded primary respiratory alkalosis makes salicylate overdose the most likely diagnosis. Salicylates toxic effects on the brainstem result in hyperventilation and respiratory alkalosis.

Encephalitis can also affect the brainstem resulting in abnormal respiratory patterns and respiratory acid base balance disorders. However we would not expect there to be a severe HAGMA as evident in this case.

Morphine overdose typically causes respiratory depression resulting in a respiratory acidosis.

An Addisonian crisis typically causes a normal anion gap metabolic acidosis (NAGMA) and would not be associated with a primary respiratory alkalosis.

Question 36 of 138

You receive a request for a second opinion from a GP who has performed a general physical check on one of their new patients. He is a 54-year-old man who is from Thailand. He had been complaining of low mood, fatigue and sensitivity to the cold. His body mass index is 31 kg/m^2. The following is a list of investigations performed by the GP.

Na^+	141 mmol/l
K^+	4.8 mmol/l
Urea	9.8 mmol/l
Creatinine	142 µmol/l
CRP	4 mg/l

Bilirubin	14 µmol/l
ALP	86 u/l
ALT	27 u/l
Calcium	2.89 mmol/l
Albumin	39 g/l
TSH	24.0 mU/l
Free T4	0.8 pmol/l
Free T3	0.4 pmol/l

ECG: Heart rate 68, sinus rhythm, QRS width 128ms, flattened T waves V1 to V6

The patient has told the GP that he takes one medication regularly but is unable to give the name.

Which medication is most likely to cause the following abnormalities?

A. Bendroflumethiazide
B. Amiodarone
C. Lithium
D. Carbimazole
E. Propylthiouracil

C. Lithium

The answer is Lithium. Long-term treatments with Lithium causes hypothyroidism in up to a third of patients with middle-aged females being the most likely demographic to get this complication. In addition, Lithium therapy is known to cause hypercalcaemia and hyperparathyroidism. Longterm lithium therapy is well known to cause renal impairment and weight gain on Lithium is common too. Cardiac side effects are rare on lithium but the most common ECG changes are flattened T waves and a widened QRS complex, in particular, a modified Brugada pattern may be seen.

Options D and E can cause hypothyroidism but are not known to cause any of the other abnormalities seen. Option A can cause weight gain and hypercalcaemia but is not known to cause any of the other abnormalities seen. Option B can cause hypothyroidism and similar ECG changes as seen here but is not known to cause any of the other abnormalities.

Question 37 of 138

A 23-year-old female student presents to the emergency department with a headache which has developed since starting her third year at university studying economics. She has also been noticing nausea and breathlessness. She had spent the summer travelling to Argentina and teaching English, after which she returned to the UK and moved into shared student accommodation in preparation for her next term. She had been well during her travels except for four days of diarrhoea shortly after she arrived. She has a past medical history of chlamydia which was treated by a local GUM clinic. She does not smoke and has minimal alcohol intake. On examination, she appears tired but otherwise well. Auscultation of her chest is normal. A urinary pregnancy test is positive.

Blood tests:

Hb	127 g/l
Platelets	336 * 10 /l9
WBC	9.4 * 10 /l9
Na^+	137 mmol/l
K^+	4.2 mmol/l
Urea	3.7 mmol/l
Creatinine	63 µmol/l

Arterial blood gas:

pH	7.41 g/l
pCO_2	3.6 mmHg
pO_2	9.4 mmHg
Na^+	137 mmol/l
FCOHb	22%

She is started on 15L of oxygen via a non-rebreather mask. How should she be further managed?

A. Wean off oxygen guided by standard pulse oximetry saturation levels
B. Add IV mannitol
C. Maintain high-flow oxygen until asymptomatic or carbon monoxide levels are <10%
D. Refer urgently for hyperbaric oxygen treatment
E. Arrange urgently for non-invasive ventilation

D. Refer urgently for hyperbaric oxygen treatment

This is a case of carbon monoxide poisoning in a pregnant patient and therefore needs urgent consideration of hyperbaric oxygen treatment. The danger of carbon monoxide poisoning to the foetus is significant as carbon monoxide has greater affinity to foetal heamaglobin and the placenta is unable to substantially compensate for reduced oxygen delivery. Whilst mild to moderate poisoning has not been shown to cause any adverse outcomes, severe maternal poisoning has been shown to cause neurobehavioural deficits when treated with normobaric oxygen alone.

Standard pulse oximetry would be unable to assess oxygenation levels in carbon monoxide poisoning. Whilst if this patient was not pregnant, there would be no indication for hyperbaric oxygen treatment and maintaining high flow oxygen until asymptomatic or carbon monoxide levels would be less than 10% would then be appropriate. Non-invasive ventilation has no role in carbon monoxide poisoning, and mannitol is only indicated in cerebral oedema.

Question 38 of 138

A 52-year-old male was brought to the emergency department by his wife after being found confused and unsteady whilst walking. He had been well that morning but his wife had found him confused about 2 hours prior to presenting in the emergency department. He had a background of alcohol excess and bipolar disorder, for which he was on lithium. His wife reported the patient had recently been low in mood, triggered by his recent job loss.

On examination, the patient was confused with a flushed complexion, slurred speech, horizontal nystagmus, and a marked ataxic gait. Observations were as follows:

oxygen saturations 97% on room air

heart rate 65 bpm

blood pressure 129/84mmHg temperature 37.2ºC

Blood results were as follows:

Creatinine	110 µmol/L	(55 - 120 µmol/L)
Urea	7 mmol/L	(2.0 - 7.0 mmol/L)
Na +	140 mmol/L	(135 - 145 mmol/L)
K +	4 mmol/L	(3.5 - 5.0 mmol/L)
Cl -	100 mmol/L	(96 - 106 mmol/L)
Bicarbonate	17 mmol/L	(22 - 29 mmol/L)
pH	7.24	(7.35 - 7.45)
Anion gap	27 mmol/L	(<16 mmol/L)
Glucose	5.0 mmol/L	(4.0 - 11.1 mmol/L)
Lactate	2.5 mmol/L	(<2.3 mmol/L)
Ethanol	7.2 mmol/L	(<0.1 mmol/L)
Osmolality	359 mOsm/kg	(285 - 295 mOsm/kg)
Osmolar gap	59 mOsm/kg	(<10 mOsm/kg)
Salicylate	negative	
Paracetamol	negative	
Lithium	pending	

What is the most likely cause of the patient's presentation?

A. Lithium overdose
B. Ethanol toxicity
C. Ethylene glycol toxicity
D. Wernicke's encephalopathy
E. Cyanide toxicity

C. Ethylene glycol toxicity

Metabolic acidosis with a high anion gap and high osmolar gap is seen in ethylene glycol poisoning

This question is looking at the calculation of both the anion gap and osmolar gap.

This patient presents with features of intoxication. He is acidotic and has a raised anion gap as follows: (Na+K) - (HCO3+Cl) = 144 - 117 = 27mmol/L.

A normal anion gap is <16 mmol/L.

He also has a raised osmolar gap. This is calculated using the following equation: Osmolar gap = measured osmolality - calculated osmolality.

- Measured osmolality = 359 mOsm/kg.
- Calculated osmolality = 2Na + glucose + urea = 288 + 7 + 5 = 300 mOsm/kg.
- Osmolar gap = 359 - 300 = 59 mOsm/kg.

A normal osmolar gap = <10 mOsm/kg.

We can add the ethanol concentration provided into our calculation of osmolality, to make it even more representative of the true osmolality. Despite this, we would still only have a calculated value of 307.2 mOsm/kg. This indicates that there is an unmeasured osmole present in the blood, causing a high osmolality, which is not explained by the presence of ethanol.

It is evident that this patient has consumed alcohol given the mildly raised serum ethanol concentration. Whilst ethanol toxicity can cause this presentation, the level is not high enough to account for such a severe presentation (according to Toxbase, symptoms of moderate toxicity such as ataxia and confusion typically occur when ethanol levels are 21.7 - 43.4 mmol/L). Furthermore, the high osmolar gap indicates the presence of another unmeasured osmole in the blood.

There are many causes of a high anion gap metabolic acidosis (please see the mnemonic CATMUDPILES below for a helpful way to remember these!). One such cause is a high concentration of serum lactate. This patient has a mildly raised lactate which could be caused by cyanide poisoning (cyanide toxicity is typically associated with signs such as 'cherry red' skin and confusion). However, whilst lactate can cause a high anion gap, it does not cause a high osmolar gap. Furthermore, cyanide poisoning severe enough to cause this presentation would typically be associated with a much higher lactate concentration (a lactate level of >7mmol/L is typically associated with cyanide poisoning). Lactate levels are often falsely elevated in ethylene glycol poisoning.

Lithium overdose is not associated with a raised osmolar gap and typically presents with nausea, tremor, and muscle weakness (early signs).

Wernicke's encephalopathy can present with confusion, ataxia, nystagmus, and ophthalmoplegia, particularly in those with a history of alcohol excess. However, this is due to a deficiency in thiamine and does not explain the metabolic abnormalities present.

Interestingly, the treatment for ethylene glycol poising is fomepizole or ethanol. There have been cases where the co-ingestion of ethylene glycol and ethanol has provided some protective benefit, but ethanol levels much higher than 7mmol/L are normally required.

Some causes of a raised anion gap metabolic acidosis can be remembered by the mnemonic CATMUDPILES (from lifeinthefastlane.com): -

- Cyanide, carbon monoxide –
- Alcoholic ketoacidosis –
- Toluene
- Methanol, metformin –
- Uraemia
- Diabetic ketoacidosis
- Phenformin, pyroglutamic acid, paraldehyde, propylene glycol, paracetamol –
- Iron, isoniazid
- Lactate (numerous causes) –
- Ethanol, ethylene glycol –
- Salicylates

Some causes of a high osmolar gap are also provided below (from lifeinthefastlane.com): -

- Mannitol –
- Methanol
- Ethylene glycol –

- Sorbitol
- Polyethylene glycol (IV lorazepam)
- Propylene glycol (IV lorazepam, diazepam, and phenytoin) –
- Glycine (TURP syndrome) –
- Maltose (IV IG - Intragram)

Question 39 of 138

A 57-year-old male is admitted to hospital with an acute kidney injury (AKI). His past medical history includes polycystic kidney disease for which he received a renal transplant 4 years ago, and type 2 diabetes mellitus. His drug history includes tacrolimus, mycophenolate mofetil, amlodipine and simvastatin. Tacrolimus toxicity is suspected to have caused his AKI.

He informs you that he has only recently been discharged, where he was started on rifampicin and erythromycin for Legionnaires' disease. He was also started on pioglitazone after a diabetic team review.

Which of his other medications is the most likely culprit of the tacrolimus toxicity?

A. Pioglitazone
B. Erythromycin
C. Simvastatin
D. Rifampicin
E. Mycophenolate mofetil

B. Erythromycin

Despite his complicated medical and drug history, this is a relatively simple question about drug interactions.

Erythromycin is a potent inhibitor of the cytochrome P450 system, which means it can have a rapid effect on levels of other drugs metabolised by this system, e.g. tacrolimus, warfarin.

Clarithromycin, fluconazole and clotrimazole are also inhibitors.

Rifampicin is an enzyme inducer, which would cause decreased tacrolimus concentrations, potentially leading to symptoms of organ rejection.

Question 40 of 138

A 23-year-old woman is reviewed following admission for paracetamol overdose. She ingested 25 grams of paracetamol and was started on N-acetylcysteine as her paracetamol level was 812 micromol/L eight hours following ingestion. She feels well, denying any abdominal pain or other symptoms.

At what point should N-acetylcysteine be stopped?

A. When INR <1.3 and ALT less than 2x upper limit
B. When abdominal pain resolved
C. When pH is >7.35
D. When paracetamol levels become undetectable
E. After 48 hours

A. When INR <1.3 and ALT less than 2x upper limit

The correct answer is when INR <1.3 and ALT less than 2x upper limit. Note that the primary measure of synthetic liver function is clotting which is why it is so closely monitored in paracetamol overdose and helps indicate when the liver is improving. pH is a useful measure for criteria for a liver transplant but is not helpful in most cases as it generally remains normal despite severe liver dysfunction. Abdominal pain is often present but not always and is therefore not a very sensitive marker. Paracetamol levels do not reflect the extent of liver damage and they can be very difficult to interpret in staggered overdoses. Depending on the extent of liver damage, treatment may need to be longer than 48hrs and in some cases shorter.

Question 41 of 138

A 62-year-old man is currently being treated with linezolid for MRSA bacteraemia.

Which of the following medications may need to be discontinued whilst this is being administered?

A. Metformin
B. Citalopram
C. Alendronic acid
D. Candesartan
E. Atorvastatin

B. Citalopram

Linezolid was originally discovered as a psychotropic agent with antidepressant effects through mild reversible nonselective inhibition of monoamine oxidase (MAO), before it was found to have antibiotic efficacy against drug-resistant gram-positive cocci. In combination with serotonin agonists there is a risk of serotonin syndrome. Therefore, when using linezolid it must be considered to discontinue SSRIs.

There are no known listed interactions between alendronic acid, candesartan and atorvastatin.

Linezolid may potentially enhance the hypoglycaemic effect of metformin. Therefore careful capillary glucose monitoring should occur in a patient on both medications, but this is not necessarily a reason to discontinue the drug.

Question 42 of 138

A 72-year-old lady is admitted with a tonic-clonic seizure. She was initially treated with two doses of 10mg diazepam however she failed to respond. She was then prescribed a loading dose of intravenous phenytoin and then prescribed maintenance dose phenytoin. Past medical history included a previous stroke, gallstones and hypertension. Drug history included clopidogrel, amlodipine, simvastatin. She recovered well and was discharged 6 days later.

Three weeks after her hospital admission she visits her GP accompanied by her husband complaining of feeling generally unwell and a rash. On examination she has a wide spread morbilliform eruption on most of her body and some mild right upper quadrant pain. The GP could palpate cervical and inguinal lymph nodes. Observations heart rate 110 regular, blood pressure 120/80, saturations 97% on air and temperature 38.2. The GP sends the patient to hospital for further tests. Bloods show:

Hb	110 g/l
WCC	14 * 10 /l9
Neutrophils	7.2 * 10 /l9
Eosinophils	2.3 * 10 /l9
Platelets	81 * 10 /l9
Na +	141 mmol/l

K +	4.3 mmol/l
Creatinine	101 µmol/l
Urea	6.8 mmol/l
Bilirubin	22 µmol/l
Alk phos	80 u/l
ALT	150 u/l
Albumin	32 g/l
INR	1.1

What is the most likely diagnosis?

A. Lymphoma
B. DRESS syndrome
C. Cholecystitis
D. EBV
E. Meningococcal septicaemia

B. DRESS syndrome

This patient has DRESS syndrome (drug reaction with eosinophilia and systemic symptoms) after starting phenytoin.

Question 43 of 138

You are asked to review a 75-year-old lady on the surgical ward. She has malignant carcinoma of the colon and had a large bowel resection seven days ago. Despite being on dalteparin since admission, she had developed a right-sided deep venous thrombosis (DVT). She tells you she had a similar problem ten years ago and had to take 'blood-thinning injections' for several months.

On examination, she looks well with no signs of respiratory distress. Her oxygen saturations are 99% on room air and on auscultation she has vesicular breath sounds throughout both lung fields.

Her pre-operative blood tests are as following:

Hb	119 g/l	Na +	135 mmol/l	Bilirubin	12 mol/l
Platelets	457 * 10 /l 9	K +	4.5 mmol/l	ALP	111 u/l
WBC	6.8 * 10 /l 9	Urea	4.9 mmol/l	ALT	45 u/l
Neuts	4.0 * 10 /l 9	Creatinine	81 µmol/l	γGT	30 u/l
Lymphs	0.9 * 10 /l 9	Albumin	34 g/l		
Eosin	0.0 * 10 /l9				

Her blood results today are as follows:

Hb	105 g/l	Na +	133 mmol/l	Prothrombin time	10.7 s
Platelets	77* 10 /l 9	K +	4.1 mmol/l	APTT	27.5s
WBC	7.6 * 10 /l 9	Urea	5.7 mmol/l	APTT ratio	45 u/l
Neuts	4.9 * 10 /l 9	Creatinine	98 µmol/l	D-Dimer	>1000 ng/ml
Lymphs	1.1 * 10 /l 9	Albumin	31 g/l		
Eosin	0.1 * 10 /l9				

Which of the following represents the optimal management for this patient while she remains an inpatient?

A. Warfarin
B. Bivalirudin
C. IV heparin infusion
D. Plasma exchange
E. Rivaroxaban

B. Bivalirudin

This lady has heparin-induced thrombocytopenia (HIT). She needs anticoagulation but should ideally be switched to a non-heparin drug for now. Bivalirudin is a direct thrombin inhibitor and is not associated with HIT.

Plasma exchange would be the treatment of choice for thrombotic thrombocytopenic purpura, which is not the case here.

Warfarin causes a rapid fall in protein C levels, risking complications such as skin necrosis, so is not used acutely.

Question 44 of 138

A 35-year-old man presents to the emergency department complaining of chest pain. He has no significant past medical history and does not take any regularly prescribed medications. He has been awake for twenty-four hours having ingested a large quantity of cocaine.

His observations are as follows: heart rate 111 beats per minute, respiratory rate 21/minute, blood pressure 180/100 mmHg, oxygen saturation 97% on room air and temperature 37ºC.

On examination, he is diaphoretic and anxious. Chest auscultation is normal. The heart sounds are normal with no added sounds or murmurs. The jugular venous pulse is not raised and there is no peripheral oedema. Abdominal and neurological examinations are normal.

ECG demonstrates sinus tachycardia with T wave inversion in leads V5-V6, I and AVL.

What medication should be avoided in this circumstance?

A. Aspirin
B. Benzodiazepines
C. Bisoprolol
D. Glyceryl trinitrate
E. Sodium nitroprusside

C. Bisoprolol

Cocaine toxicity - avoid beta-blockers

Bisoprolol is the correct answer. Beta-blockers should be avoided in cocaine-induced chest pain. The rationale is that beta-antagonism may result in unopposed alpha-mediated coronary vasospasm.

Aspirin is incorrect. This patient has an acute coronary syndrome and in addition to cocaine specific treatment, he will need antiplatelet treatment and coronary angiography.

Benzodiazepines are incorrect. This class of medication is used early in the treatment of cocaineinduced chest pain.

Glyceryl trinitrate is incorrect. This class of medication is used in the management of cocaineinduced chest pain.

Sodium nitroprusside is incorrect. The use of this may be indicated in those patients suffering from cocaine toxicity and persistent hypertension.

Question 45 of 138

A 68-year-old man is admitted with a two day exacerbation of COPD. He arrives unwell and is started on steroids, nebulisers, aminophylline, ciprofloxacin and non invasive ventilation. He improves well and is weaned off the ventilation by his second day but starts to complain of palpitations and chest pain on the third day. He is usually well taking fostair twice a day. He used to smoke and drinks 25 units of alcohol a week.

You review him and find he has a pulse of 134 beats per minute that is irregular. He is clammy and his blood pressure is 96/76mmHg. He has a chest that is wheezy throughout and is saturating at 91% with 2 litres of nasal cannulae oxygen. He is visibly trembling and feels nauseous. A cardiac monitor is attached showing an irregular narrow complex tachycardia.

Na +	145 mmol/l
K +	3.2 mmol/l
Urea	4.5 mmol/l
Creatinine	76 µmol/l
Theophylline level	28 mcg/ml (normal range 10-20)
ECG	irregular narrow complex tachycardia with no visible p-waves
Chest x-ray	hyperexpanded film, no consolidation seen

What is the likely cause of his drug toxicity?

A. Ciprofloxacin
B. Hypokalaemia
C. Anxiety
D. Hidden pneumothorax
E. Alcohol withdrawal

A. Ciprofloxacin

Quinolones -> inhibit liver enzymes -> risk of theophylline toxicity in COPD/asthma exacerbation

This gentleman has developed new fast atrial fibrillation despite his initial improvement. The hypokalaemia probably contributes but it is clear he is toxic with aminophylline. Aminophylline concentrations are highly sensitive depending on the speed of liver metabolism. Ciprofloxacin is a liver enzyme inhibitor and when started alongside aminophylline, patients can be slow to metabolise and rapidly develop theophylline toxicity unless the dose is reduced. Alcohol withdrawal and anxiety are two differentials if no abnormalities showed on the investigations. A hidden pneumothorax is unlikely to be the cause here.

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A 69-year-old woman attends the emergency department with a 2-day history of lower back pain after falling down the stairs. She has been taking 1g of paracetamol every 1-2 hours to control the pain but says this has not helped her symptoms. Over the last 8 hours, she now has developed right upper quadrant pain and nausea. Her last dose of paracetamol was 45 minutes ago.

Laboratory investigations:

Hb	123 g/L	(115 - 160)
Na +	137 mmol/L	(135 - 145)
K +	4.1 mmol/L	(3.5 - 5.0)
Urea	5.4 mmol/L	(2.0 - 7.0)
Creatinine	77 µmol/L	(55 - 120)
Bilirubin	16 µmol/L	(3 - 17)
ALP	99 u/L	(30 - 100)
ALT	42 u/L	(3 - 40)
Paracetamol levels	35 mg/L	(10-30)

What is the most initial step in the management of this patient?

A. Activated charcoal
B. Initiate N-acetylcysteine
C. No treatment indicated
D. Referral to liaison psychiatry
E. Repeat paracetamol levels at 4 hours from last ingestion

B. Initiate N-acetylcysteine

This patient has taken a staggered overdose of paracetamol, having taken 1g every 1-2 hours for back pain. Regardless of the paracetamol level and where it corresponds on the treatment graph, patients should be immediately treated with **N-acetylcysteine** to prevent liver toxicity.

Activated charcoal is incorrect. This can be administered within the 1st hour of paracetamol ingestion, to reduce absorption. However, in the event of taking a staggered overdose, NICE guidelines recommend commencing N-acetylcysteine regardless of the paracetamol levels and time following ingestion.

No treatment indicated is incorrect. Not treating a staggered paracetamol overdose is dangerous and could lead to long-term hepatic complications or death from acute liver failure.

Referral to liaison psychiatry is incorrect. Whilst patients who have presented with a drug overdose should be considered for referral to liaison psychiatry, this patient has no other presenting signs to suggest an intentional overdose. Nevertheless, the most important initial step is to administer treatment with N-acetylcysteine prior to referring to liaison psychiatry.

Repeat paracetamol levels at 4 hours from last ingestion is incorrect. In most cases of paracetamol overdose, guidelines state that paracetamol levels should be taken 4 hours from the last ingestion. If the paracetamol levels are then over the treatment line, then treatment with Nacetylcysteine can be commenced. However, the exception to the rule includes a large overdose (>150mg/kg) or a staggered overdose of paracetamol.

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A 28-year-old lady is brought in by her mother having taken an overdose of an unknown medication. She has recently broken up with her boyfriend and has been fired from her job for turning up late to work persistently. She was found in her room by her mother with a small bottle of vodka which was empty and some burnt medication packaging. She has no history of mental health disease, although did self-harm (arm cutting) twice whilst a teenager. She has no medical history other than being treated for malaria during a recent trip to west Africa. The patient is tearful, regrets the overdose and is currently complaining of nausea, epigastric discomfort, slightly blurred vision and a ringing in her ears.

On examination, her heart rate is 115 beats/min, her blood pressure 98/48 mmHg and she appears flushed. Her respiratory rate is 18 breaths per minute and her oxygen saturations 98% breathing room air. Her Glasgow coma score is 15/15 and her pupils are equal and reactive. Her blood results are as follows.

Na +	135 mmol/l
K +	4.3 mmol/l
Urea	5.1 mmol/l
Creatinine	86 µmol/l
Bilirubin	18 µmol/l
ALP	65 u/l
ALT	56 u/l

What is the most likely toxin?

A. Quinine overdose
B. Ibuprofen overdose

C. Alcohol intoxication
D. Paracetamol overdose
E. Malarone overdose

A. Quinine overdose

Cinchonism is the syndrome described (blurred vision, tinnitus, nausea...) and can be secondary to quinine or quinidine ingestion as an overdose, but also less commonly with normal dosing. Although uncommon in the western world use of quinine as a drug in overdose is not uncommon in malaria endemic areas such as south-east Asia. The tinnitus may be associated with transient loss of high frequency hearing loss. The blurred vision can progress to complete blindness, which in some cases can be permanent. None of the other toxins would explain this toxidrome.

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A 65-year-old woman attends the emergency department complaining of three weeks of gradual visual loss. She has a past medical history of rheumatoid arthritis, type 2 diabetes, and pulmonary tuberculosis. Her medications include methotrexate, hydroxychloroquine, metformin, rifampicin, ethambutol hydrochloride, pyrazinamide, isoniazid and pyridoxine.

On examination, visual acuity is 20/50 in both eyes. There are central scotomas bilaterally on visual field testing. The macula and fovea are normal.

Blood tests:

ESR 88 mm/hr	(<20)
Hba1c	54 mmol/mol (<48)

What is the likely cause of her presentation?

A. Ethambutol treatment
B. Giant cell arteritis
C. Hydroxychloroquine treatment
D. Multiple sclerosis
E. Vitreous haemorrhage

A. Ethambutol treatment

Optic neuritis is common in patients taking ethambutol

Ethambutol treatment is correct. The patient presents with insidious onset painless loss of central vision. This is typical of optic neuropathy associated with ethambutol treatment. Eventually, the optic nerve may become visually abnormal but it may be normal early in the course of the disease. The raised ESR is likely due to either tuberculosis or rheumatoid.

Giant cell arteritis is incorrect. This can cause visual loss and a raised ESR. However, visual loss in giant cell arteritis (GCA) is typically unilateral and sudden rather than bilateral and progressive.

Hydroxychloroquine treatment is incorrect. This can cause painless bilateral loss of vision. However, we would expect macular oedema, which is absent here. Hydroxychloroquine (HCQ) retinopathy typically occurs when patients are treated with HCQ for extended periods of time i.e. > 5 years.

Multiple sclerosis is incorrect. This can cause optic neuritis. However, this is more likely to be painful and unilateral rather than painless and bilateral.

Vitreous haemorrhage is incorrect. This can occur as a complication of diabetes. However, it would typically be unilateral and cause sudden painless loss of vision. Additionally, blood may be visible on fundoscopic examination.

Question 49 of 138

A 45-year-old woman presents to the emergency department with bilateral hand tremor, unsteady gait, and blurry vision. Her past medical history is significant for bipolar disorder, which has been under control for the last year on lithium. Recently, her general practitioner started her on amlodipine for essential hypertension. Additionally, she started taking paracetamol and oral ibuprofen daily over the last week for knee pain related to a recent fall.

On examination, a coarse tremor is noted in both hands. There is also nystagmus, dysdiadochokinesia, and past pointing.

What is the best next step in managing this patient?

A. Increase lithium dose
B. Start haemodialysis
C. Stop amlodipine
D. Stop ibuprofen
E. Stop paracetamol

D. Stop ibuprofen

Lithium toxicity can be precipitated by NSAIDs

The patient presents with classic symptoms of lithium toxicity related to recent use of ibuprofen. **Increasing the dose of lithium** would cause further deterioration of her symptoms. Lithium should be held, and lithium levels should be measured to guide further management.

Management of lithium toxicity is mainly supportive. It is essential to discontinue lithium and the drug that precipitated lithium toxicity (e.g., ibuprofen, bendroflumethiazide) and consider administration of intravenous fluids based on the patient's fluid balance status. **Haemodialysis** may be required in the context of severe symptoms, kidney dysfunction, and significantly raised lithium levels, but it is not indicated as a first-line treatment for this patient.

Amlodipine does not interact with lithium and, as such, it can be continued for the management of essential hypertension.

This patient presents with classic features of lithium toxicity. This has been precipitated by ibuprofen ingestion. Non-steroidal anti-inflammatory drugs and thiazide-type diuretics can increase lithium levels and, as such, should be avoided in patients on lithium. In this case, **ibuprofen should be discontinued** in the first instance.

Paracetamol does not interact with lithium, and as such, it can be continued for the management of essential hypertension.

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A 24-year-old man presents to the accident and emergency department with his girlfriend after collapsing in a nightclub at 0400hrs. It is now 0600hrs On examination he is V on the AVPU scale with dry mucous membranes, he has flushed skin and feels very warm to the touch. There is mildly increased muscle tone, myoclonic jerks and hyperreflexia globally but worse in the lower limbs. Pupils are dilated but respond to light equally and bilaterally. He is disorientated and agitated during the examination, thrashing about in the bed and occasionally lashing out at people

Heart rate 126/min, blood pressure 90/60 mmHg, respiratory rate 18/min, temperature = 40.3°C, sats 97% on 10l of oxygen.

Na +	136 mmol/l

K +	3.9 mmol/l
Urea	2.3 mmol/l
Creatinine	77 µmol/l
Glucose	4.6 mmol/l
Creatinine kinase	12,000 U/l

Bilirubin	25 µmol/l
ALP	185 u/l
ALT	125 u/l
γGT	144 u/l
Albumin	40 g/l

CT Head: No abnormality detected. Lumbar puncture reveals:

Opening pressure	15 cmCSF
Appearance	Clear
Glucose	3.5 mmol/l
Protein	0.3 g/l

White cells	4 / mm^3

He has a past medical history of schizoaffective disorder. His girlfriend shows you his regular medications, the prescription has been unchanged for 1 year. Olanzapine 15mg and Sertraline 150mg. His girlfriend tells you that he took an unknown white powder whilst in the club

What is the most appropriate treatment plan?

A. IV Ceftriaxone
B. Stop Olanzapine and give dantrolene
C. Stop sertraline and give IV lorazepam and consider cyproheptadine
D. Give IV naloxone
E. Stop olanzapine and sertraline and supportive therapy

C. Stop sertraline and give IV lorazepam and consider cyproheptadine

The diagnosis is MDMA intoxication which has led to serotonin syndrome and rhabdomyolysis. The treatment for serotonin syndrome is stopping any serotonergic agents, using benzodiazepines for agitation and consideration of the use of serotonin antagonists such as cyproheptadine if there is severe autonomic disturbance. MDMA intoxication can lead to serotonin syndrome, especially if used in conjunction with any drug which increased serotonin transport across the synapses eg SSRIs such as sertraline.

Serotonin syndrome presents with agitation, reduced consciousness, flushing of the skin, hyperthermia, hyperreflexia, myoclonus (worse in the lower limbs), tachycardia, hypertension or labile BP. Most cases of serotonin syndrome present within 6 hours of any change in medication or dose i.e in this case after the MDMA.

Neuroleptic malignant syndrome would present in a similar way and would be treated by option B.However NMS usually evolves over 1-3 days with extreme muscle rigidity being a characteristic feature. NMS is less likely when on a lower potency antipsychotic such as Olanzapine and less likely when the dose has been stable for a long period of time.

Heroin overdose would not present with these features. Dilated pupils essentially rule this out and heroin is not usually a white powder which makes option D less likely.

Although meningitis can present with fever and reduced consciousness the normal LP rules out this diagnosis and therefore option A is an inappropriate treatment

Option E is inappropriate as this gentleman is severely unwell with autonomic instability, severe hyperthermia and significant rhabdomyolysis with renal impairment. Active management other than supportive treatment would be recommended for this individual if the diagnosis were NMS or serotonin syndrome

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A 65-year-old woman attends the clinic with shortness of breath. This has progressively worsened over the past 3 months. She also complains of swollen ankles. She has a past medical history of breast cancer which was treated with docetaxel, cyclophosphamide, trastuzumab, and radiotherapy. She also has rheumatoid arthritis for which she takes methotrexate.

On examination, you note bilateral pitting oedema to the mid calves and coarse crepitations to the mid zones of the lungs. Observations are as follows: heart rate 92 beats per minute, respiratory rate 18 breaths per minute, blood pressure 125/75 mmHg, SpO2 94% on air, and temperature 37.2ºC.

An echo is performed:

Echo	Biventricular failure; left ventricle ejection fraction 28%; no evidence of pericardial thickening

What is the most likely implicated therapeutic?

A. Cyclophosphamide
B. Docetaxel
C. Methotrexate
D. Radiotherapy
E. Trastuzumab

E. Trastuzumab

Trastuzumab (Herceptin) - cardiac toxicity is common

Trastuzumab is correct. Trastuzumab (Herceptin) is a monoclonal antibody directed against the HER2/neu receptor. Cardiotoxicity is a well-recognised complication and is more common when anthracyclines have also been used.

Cyclophosphamide is incorrect. Although higher doses of this drug may produce significant cardiac toxicity (including fatal hemorrhagic myocarditis), the incidence is much less so compared to trastuzumab.

Docetaxel is incorrect. Cardiac failure is a rare side effect of this agent.

Methotrexate is incorrect. Several clinical trials have shown that methotrexate is associated with improved endothelial function, slower atherosclerosis progression, decreased risk of major cardiovascular adverse events, and benefits on overall survival.

Radiotherapy is incorrect. Constrictive pericarditis is a late complication of radiation therapy. Patients typically present with signs and symptoms of heart failure, similar to other causes of constrictive pericarditis. The absence of pericardial thickening on echo makes this a less likely diagnosis.

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A 65-year-old female presents to her GP with a four days history of non-itchy rashes on her face, neck and bilateral upper limbs. Her chest and abdomen are not affected. She does regular gardening. Her past medical history includes myocardial infarction and asthma. Further history reveals that last week she was seen in the cardiology outpatient clinic for arrhythmias and was started on a new medication.

Which of the following drug is most likely casuing the patient's symptom?

- A. Digoxin
- B. Dabigatran
- C. Verapamil
- D. Amiodarone
- E. Flecainide

D. Amiodarone

The distribution of the rash suggests phototoxicity. Among the list of the medications, only amiodarone and flecainide are assocaited with phototoxicity. However, flecainide is contraindicated in patients with history of myocardial infarction.

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A 22-year-old male is 'blue-lighted' to the Emergency Department having been found collapsed at a party. On arrival, his GCS is 3/15 and he is immediately intubated and ventilated by the Emergency Physicians.

On examination he is afebrile. He is mildly bradycardic at 53bpm and his blood pressure is 109/69mmHg. His pupils measure 2mm bilaterally. IV access is obtained and 400 micrograms of naloxone are administered without effect. The on-call radiologist is contacted and a CT head scan is arranged.

One hour later the patient extubates himself without warning. He is referred to medicine as his GCS is still 13/15. His CT demonstrates no acute intracranial pathology. By the time you arrive in the Emergency Department his GCS has improved to 15/15 and he is demanding to go home.

Which drug is most likely to be implicated?

- A. Diazepam
- B. Methoxetamine
- C. Gamma-hydroxybutyric acid (GHB)
- D. Heroin
- E. Methamphetamine

C. Gamma-hydroxybutyric acid (GHB)

GHB or 'Grievous Bodily Harm' is a colourless, odourless, bitter-tasting substance that acts as a CNS depressant. It is abused for its ability to induce euphoria, amnesia, and hypnosis. It is popular amongst party-goers and achieved notoriety for its use as a date-rape drug.

GHB toxicity occurs secondary to its effects as a CNS depressant. Patients typically present with coma, respiratory depression, mild bradycardia, and vomiting. The key feature of GHB toxicity is its short recovery time. Patients typically recover in 6 hours and toxicity is often maximal on presentation. Recovery often occurs spontaneously in the Emergency Department and patients have been known to self-extubate.

Heroin and diazepam could both produce a similar toxidrome. Recovery is generally more gradual, and one would expect naloxone to reverse a heroin overdose.

Methamphetamine is a stimulant. Clinical features include agitation, tachycardia, hypertension, mydriasis, and hyperthermia.

Methoxetamine is an analogue of the dissociative anaesthetics ketamine and phencyclidine (PCP). In addition to dissociative phenomena, it also causes tachycardia, hypertension, confusion, and mydriasis. Patients may also present with an acute cerebellar syndrome.

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A 35-year-old man is brought to the emergency department. He was a bystander at a house fire, who called the fire and ambulance services. The female resident of the property had escaped the fire but was found collapsed in the front garden. The bystander started cardio-pulmonary resuscitation including mouth-to-mouth breathing until the ambulance crew arrived.

Whilst the emergency services were dealing with the fire and unconscious resident, the 35-yearold man starts to feel 'light-headed'. The ambulance crew bring him to the emergency department as a precaution.

He is quickly reviewed by the emergency team, who refer this gentleman because of ongoing symptoms and feel he needs a longer period of observation in the medical assessment unit. They handover there was no clinical features to suggest inhalation injury from the fire.

Upon your review, the patient has deteriorated. He is confused and unable to provide any history. You are able to perform a limited examination which reveals a temperature of 37.5ºC, heart rate 38 beats/min, blood pressure 80/60 mmHg, respiratory rate 10/min and oxygen saturation of 98% on room air. Examination of the chest reveals equal vesicular breath sounds, no added sounds and dual heart sounds with no murmurs. Neurological assessment reveals confusion, a reduced level of consciousness and dilated pupils bilaterally.

You take an urgent arterial blood gas and prescribe a bolus of crystalloid fluid. A 12 lead ECG shows sinus bradycardia. Whilst awaiting the results of the arterial blood gas, the patient develops generalised convulsions.

You and the emergency team work together to stabilise the patient - he is intubated, placed on mechanical ventilation and vasopressors are commenced to support his blood pressure. During this time, you are presented with the results of the arterial blood gas:

pH	7.11
PaO2	100mmHg
SaO2	96% on room air
PaCo2	28mmHg
Bicarbonate	7 mmol/L
Chloride	105 mmol/L
Lactate	8 mmol/L
Carboxyhaemoglobin	0.7%
Na+	138 mmol/l
K+	4.5 mmol/l

The full blood count and biochemistry results are still awaited.

Which of the following is the most appropriate management for this patient?

- A. Urgent computed tomography (CT) imaging of the brain
- B. Atropine boluses, followed by atropine infusion
- C. Pralidoxime
- D. Benztropine
- E. Sodium thiosulfate

E. Sodium thiosulfate

Whist carbon monoxide poisoning is most associated with exposure to house/industrial fires, cyanide toxicity from burning of synthetic materials should also be considered

This man has cyanide poisoning. Performing mouth to mouth resuscitation has led to the oral transmission of cyanide molecules from the resident. The absorption of cyanide is slower via the oral route as compared to inhalation, which explains why this patient had a relatively 'delayed' presentation of cyanide poisoning. Also, exposure to small amounts of cyanide can lead to a longer duration of symptoms before cardiopulmonary arrest. The antidote for cyanide poisoning is intravenous sodium thiosulfate (+/- hydroxocobalamin).

Fire is the most common source of cyanide exposure in industrialised countries. Mouth-to-mouth resuscitation is contraindicated in cyanide poisoning because of risk to the provider of CPR.

The other differential diagnosis, in this case, is carbon monoxide inhalation. However, seizures are common in cyanide poisoning but are rare in carbon monoxide poisoning. Cyanide poisoning also causes mydriasis. whereas carbon monoxide does not affect the pupils. Administration of 100% oxygen is indicated for both carbon monoxide and cyanide poisoning.

In normal human physiology, detoxification of cyanide occurs via rhodanese, an enzyme found in many tissues but particularly in the liver and muscle. Rhodanese converts cyanide to thiocyanate, a water-soluble molecule excreted in the urine.

In poisoning, rhodanese is saturated and unable to remove the surplus cyanide molecules. Sodium thiosulfate serves as a sulfur donor in the reaction catalyzed by rhodanese. Hence administration of sodium thiosulfate increases cyanide to thiocyanate conversion. Hydroxocobalamin (vitamin B12) has also been approved for the treatment of cyanide poisoning.

Although rare, you should think of cyanide poisoning in any patient with unexplained severe lactic acidosis, coma, bradycardia, and normal oxygenation.

Option 1 - treating the poisoning should take priority over investigating the reduced level of consciousness/seizures with brain imaging

Option 2 - this is part of the treatment algorithm for organophosphate poisoning

Option 3 - this is also part of the treatment algorithm for organophosphate poisoning

Option 4 - this is part of the treatment algorithm for dystonic reactions that may be secondary to anti-psychotic overdose

Option 5 - the correct answer

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A 33-year-old woman with bipolar affective disorder attends as she is struggling to conceive. She also complains of severe fatigue, palpitations, and a worsening acneiform facial rash.

On review, you note that she has been taking olanzapine for over ten years.

Which of the following is she at increased risk due to long-term use of this medication?

A. Acne
B. Agranulocytosis
C. Polycystic ovarian syndrome
D. Prolonged QTc
E. Type 2 diabetes mellitus

E. Type 2 diabetes mellitus

Long-term use of olanzapine is a significant risk factor for the development of type 2 diabetes mellitus

Type 2 diabetes mellitus is correct. Long-term olanzapine use is a significant risk factor for developing type 2 diabetes.

Acne is incorrect. There is no evidence to show a link between the development of acne and olanzapine use.

Polycystic ovarian syndrome is incorrect. There is no evidence to suggest an increased risk of polycystic ovarian syndrome as a result of olanzapine use. Traditional neuroleptics can lead to raised levels of prolactin which causes relative infertility. Olanzapine has only minimal effect on prolactin levels.

Agranulocytosis is incorrect. Some antipsychotic medications including clozapine, tetracyclic antidepressants, and phenothiazines have been shown to cause agranulocytosis, but this is not the case with olanzapine. There are a small handful of case reports showing blood dyscrasias with olanzapine and so monitoring full blood counts is still recommended.

Prolonged QTc is not seen with olanzapine use but can be seen with other neuroleptic medications such as chlorpromazine, fluoxetine, haloperidol, and amitriptyline.

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A 45 woman is admitted with fever, night sweats and weight loss. She has a past medical history of type 1 diabetes. At a recent retinal screen there were microaneurysms and dot and blot haemorrhages noted on the periphery of both her discs. She has never noted any visual problems.

A sputum sample is positive for acid fast bacilli and she is subsequently started on Rifampicin, Isoniazid, Pyrazinamide, ethambutol and pyridoxine.

On the same admission she is diagnosed with HIV. Her CD4 count is 140 cells/µL. She is started cotrimoxazole and a few weeks later starts antiretroviral therapy with Efavirenz, emtricitabine and tenofovir.

She makes a good recovery and is discharged from hospital. A few months later she complains of increasingly blurry vision. Fundoscopy shows pale optic discs, microaneurysms and blot haemorrhages.

What is the most likely cause for her visual symptoms?

A. CMV retinitis
B. Ethambutol
C. Efavirenz
D. Diabetic retinopathy
E. Pyrizinamide

B.Ethambutol

Ethambutol is the correct answer here and is well known to cause optic neuritis. Diabetic retinopathy is unlikely to cause such an acute deterioration considering her recent retinal screen. CMV retinitis is unlikely to cause this picture and efavirenz and pyrazinamide do not have ocular side effects.

Question 57 of 138

A 46-year-old male was transferred to the Intensive Care Unit (ICU) as an emergency admission. He had been scheduled to undergo an elective open cholecystectomy under general anaesthetic. He had been intubated and ventilated successfully, but a few minutes into the operation he had become tachycardic, with his heart rate rising from a resting rate of 72 bpm to a rate of 142bpm. His oxygen saturations dropped down to 92% on 15 litres of oxygen per minute, and his blood pressure rose from his baseline of 114/78 mmHg up to 162/98 mmHg. In addition, his end tidal CO2 concentration rose, despite several checks to ensure that the tracheal tube was correctly sited. His past medical history was unremarkable except for an appendicectomy under general anaesthetic aged 26 yrs old.

The operation was abandoned. Upon his arrival to ICU, he was intubated and ventilated, maintaining an oxygen saturation of 96% on 15 litres per minute of oxygen. He appeared flushed and had significant muscle rigidity. His blood pressure had risen to 174/102 mmHg, and his heart rate was 136bpm. His temperature was 38.2 C. Examination of the cardiovascular revealed vasodilated peripheries with a bounding pulse and normal heart sounds. Auscultation of the lungs revealed good air entry in all zones and a correctly cited tracheal tube. Examination of the abdomen was unremarkable. Examination of the neurological system confirmed the presence of equal and reactive pupils, with generalised muscle hypertonicity and masseter muscle spasm. A central venous catheter and arterial line were inserted. The central venous pressure was 9 cm.

An arterial blood gas sample on 15 litres of oxygen was taken:

pH	7.26
HCO3	18 mmol/l
Pa02	17 kPa
PaC02	7.6 kPa
Na+	139 mmol/l
K+	7.1 mmol/l

What is the next best immediate management step?

A. Commence IV labetalol infusion
B. Commence IV meropenem
C. Commence high dose IV hydrocortisone
D. Commence IV dantrolene
E. Arrange immediate therapeutic hypothermia

D. Commence IV dantrolene

This patient has developed malignant hyperthermia as a result of his anaesthetic. In addition to stopping the responsible anaesthetic agent, the best immediate management step involves commencing IV dantrolene. Early commencement of dantrolene has greatly reduced mortality associated with malignant hyperthermia and should be administered as soon as the diagnosis is suspected. Note that hyperthermia is a relatively late feature of malignant hyperthermia, and earlier features include hypoxia, hypercarbia, masseter muscle spasm and hypertonicity.

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A 58-year-old man presents with several months of feeling generally tired, cold and experiencing some weight gain. His past medical history includes atrial fibrillation, for which he has taken amiodarone for several years.

Blood tests are performed, which show the following:

Thyroid-stimulating hormone (TSH)	9.2 mU/L	(0.5-5.5)
Free thyroxine (T4)	6.7 pmol/L	(9.0 - 18)

Given the above, what is most appropriate?

A. Continue amiodarone and start carbimazole
B. Continue amiodarone and start levothyroxine
C. Stop amiodarone and monitor
D. Stop amiodarone and start levothyroxine
E. Stop amiodarone and start prednisolone

B. Continue amiodarone and start levothyroxine

In amiodarone induced hypothyroidism, amiodarone can be continued with levothyroxine

The scenario above demonstrates an example of amiodarone-induced hypothyroidism. This is common, with around 1 in 6 patients taking amiodarone developing some form of thyroid dysfunction. As well as **treating the deficit with levothyroxine, amiodarone can be continued** in these patients, and so this is the correct answer.

Continuing amiodarone and starting carbimazole is inappropriate. Carbimazole would be appropriate for thyrotoxicosis, not hypothyroidism. In thyrotoxicosis, amiodarone should be stopped, unlike hypothyroidism, and so continuing would be incorrect. If type 1 - excess iodineinduced thyroid hormone synthesis and a goitre present, carbimazole is an appropriate option.

Stopping amiodarone and monitoring is not the preferred management option here. Amiodarone is important with regards to controlling this patient's atrial fibrillation. Also, levothyroxine should be administered to treat hypothyroidism.

Stopping amiodarone and starting levothyroxine is not quite right. Although commencing levothyroxine is appropriate, amiodarone should be continued.

Stopping amiodarone and starting prednisolone is incorrect. This would be suitable for type 2 thyrotoxicosis. Type 2 is defined as amiodarone-related destructive thyroiditis with an absent goitre.

Question 59 of 138

A 48-year-old man is referred to the outpatient department by his GP, having experienced tremor, heat intolerance and 2 kg weight loss over the last 6 weeks. His past medical history includes having atrial fibrillation for which he takes warfarin and amiodarone. He is a non-smoker and drinks on average 10 units of alcohol per week.

Blood tests are performed and reveal:

Hb	142 g/l	
Platelets	220 * 10 /l9	
WBC	7.2 * 10 /l9	
Na +	140 mmol/l	
K +	4.2 mmol/l	
Urea	4.5 mmol/l	
Creatinine	45 µmol/l	
Thyroid stimulating hormone (TSH)	0.03 mu/l	
Free thyroxine (T4)	29 pmol/l	
Total T3 (TT3)	252 ng/dL	Normal rar

A colour flow Doppler sonography of the thyroid is performed and shows absent vascularity and gland destruction.

What is the most likely diagnosis?

A. Type 1 amiodarone-induced thyrotoxicosis
B. Type 2 amiodarone-induced thyrotoxicosis
C. Grave's disease
D. Hashimotos thyroiditis
E. De Quervains thyroiditis

B. Type 2 amiodarone-induced thyrotoxicosis

Type 2 amiodarone-induced thyrotoxicosis typically occurs in patients without underlying thyroid disease and is the result of the toxic effect of amiodarone on the thyroid follicular cells, causing a destructive thyroiditis that results in excess release of preformed T4 and T3 into the circulation. The condition has two phases a thyrotoxic phase which can last weeks to months, which is often followed by a hypothyroid phase and an eventual recovery in most patients.

Type 1 amiodarone-induced thyrotoxicosis normally occurs in patients with underlying thyroid pathology like Graves disease. In these patients, there is accelerated thyroid hormone synthesis secondary to the iodide load from the amiodarone therapy.

Question 60 of 138

A 25-year-old woman presents to the Emergency department some 90 mins after taking 50x300mg aspirin tablets. She feels nauseated, has vomited once, complains of severe tinnitus, and is hyperventilating. She has no past medical history of note and takes no regular medications. Examination reveals a blood pressure of 105/70 mmHg, pulse is 87 beats per minute and regular. Chest is clear, respiratory rate is elevated at 35/min. She has been given activated charcoal on admission. 1 litre of normal saline given intravenously over 1hr is in progress.

investigations

Hb	125 g/l
Platelets	189 * 10 /l9
WBC	10.2 * 10 /l9
Na +	145 mmol/l
K +	3.1 mmol/l
Urea	6.1 mmol/l
Creatinine	90 µmol/l
pH	7.54
Serum salicylate	4.2 mmol/l

Which of the following is the most appropriate next step?

A. IV furosemide
B. IV sodium bicarbonate
C. Oral diazepam
D. Referral for haemodialysis
E. Repeated activated charcoal

IV sodium bicarbonate

Administration of sodium bicarbonate is recommended to drive alkalinisation of urine, even where there is respiratory alkalosis in aspirin overdose. It is therefore the correct next step here.The

recommended dose If the salicylate concentration in an adult is above 3.6 mmol/L is 225 mmol sodium bicarbonate, (225 mL of 8.4% over 60 minutes or 1.5 L of 1.26% over two hours).

Forced diuresis with furosemide is not recommended as large fluid shifts may increase the risk of pulmonary oedema associated with aspirin overdose. Oral diazepam is not appropriate and would potentially just shorten the time to development of acidosis. Haemodialysis is considered when salicylate concentration is greater than 5.1 mmol/l.

Question 61 of 138

A 52-year-old gentleman presents after hearing a popping sound followed by acute onset right ankle pain. On examination he is unable to plantar flex his right ankle. He denies any recent strenuous exercise. He has never had any joint problems but does tell you he has recently been treated for a urinary tract infection.

Which one of these antibiotics is likely have contributed to his presentation?

A. Erythromycin
B. Trimethoprim
C. Nitrofurantoin
D. Ciprofloxacin
E. Co-amoxiclav

D. Ciprofloxacin

Ciprofloxacin may lead to tendinopathy

A rare side effect of ciprofloxacin is tendon inflammation +/- tendon rupture as listed in the BNF. With a history suggestive of tendon rupture remember to ask about recent antibiotic usage.

Question 62 of 138

A 37-year-old man attends endocrinology clinic for assessment of the unwanted effects of longterm anabolic steroid use. The patient had been a competitive bodybuilder, who had used anabolic steroids to enhance his training regime, but now stated that he had 'retired' from bodybuilding last week and so stopped using steroids. The patient was concerned about the longterm health consequences of his previous use.

The patient described a 10-year period of anabolic steroid use. Typically he would take a daily oral formulation of a synthetic testosterone supplemented by an intramuscular injection of a longer acting agent every few weeks. The patient would take breaks from anabolic steroid use intermittently throughout the year, to reduce unwanted effects and also to evade anti-doping testing arranged by the organisers of the competitions in which he competed. The patient stated that he had never shared or reused needles when injecting himself with anabolic steroids.

The patient reported a range of unwanted effects he had developed secondary to his anabolic steroid use. These included severe acne affecting the patient's face and chest, intermittent symptoms of gastrointestinal dysfunction, and male pattern baldness. When asked directly, the patient reported that he had suffered from erectile dysfunction and scrotal discomfort towards the end of his use of anabolic steroids. The patient did not report any other concerns, in particular, he denied symptoms associated with heart or liver disease. The patient had no other significant past medical history and disclosed being a former user of recreational drugs, including cocaine.

Examination of the patient's cardiovascular and respiratory symptoms was unremarkable except for moderate pitting oedema to the level of the mid-tibia bilaterally. Examination of the abdomen noted mild bilateral gynaecomastia but no other signs of chronic liver disease. The patient's testicular volume was estimated as 18 ml bilaterally. A brief mental state examination did not reveal any evidence of a significant mood or anxiety disorder.

HbA1C	47 mmol / mol (reference < 42)
Fasting LDL cholesterol	4.5 mmol / L (reference < 3.0)
Fasting HDL cholesterol	1.0 mmol / L (reference > 1.2)
Prolactin	501 mU / L (reference 80 - 400)
Luteinising hormone	1.4 IU / L (reference 1.8 - 8.6)
Follicle-stimulating hormone	1.2 mU / ml (reference 1.5 - 12.4)
Transthoracic echocardiogram	Normal systolic and diastolic function; no left ventricular hypertrophy; normal valvular function

Which of the patient's unwanted effects will be irreversible with cessation of steroid use?

A. Pitting oedema

B. Erectile dysfunction
C. Scrotal pain
D. Male pattern baldness
E. Gastrointestinal dysfunction

D. Male pattern baldness

Anabolic steroid use is associated with a wide variety of unwanted effects. Morbidity and mortality from cardiac diseases are increased, although the precise mechanism of this effect is unknown. In addition, serious liver disease - for example, hepatocellular carcinoma - is also associated with anabolic steroid use. There are a variety of less dangerous but troubling unwanted effects as described below, some of which will be irreversible with cessation of anabolic steroid use. Due to the potential risk to long-term health, anabolic steroid users should be strongly advised to stop their use. No tapering of the dose is required. Active or former users should be monitored lifelong for evidence of complications.

Reversible unwanted effects of anabolic steroids include:

- Increased appetite
- Gastrointestinal dysfunction
- Mood swings
- Anxiety
- Acne
- Oedema
- Libido change
- Scrotal pain
- Erectile dysfunction
- Menstrual irregularities

Irreversible unwanted effects of anabolic steroids include:

- Hirsutism
- Voice pitch changes
- Male pattern baldness
- Skin striae or keloid scarring
- Chest pain
- Clitoral hypertrophy
- Short stature due to premature fusion of growth plates

It is unclear whether other unwanted effects are reversible with cessation of anabolic steroids; for example, gynaecomastia, testicular atrophy and infertility. Some limited studies suggest that normal sperm production and fertility return following cessation of anabolic steroid use, although on a timescale ranging from 4 months to 5 years.

The patient also has a typical pattern of hormonal irregularities associated with anabolic steroid use. A deranged lipid profile and impaired glucose tolerance are also frequently observed. The patient's echocardiogram is reassuringly normal, which suggests that his leg oedema will resolve with cessation of anabolic steroid use.

Question 63 of 138

A 23-year-old male is brought to the ED via ambulance after a seizure. His friends reported an episode of jerking of all four limbs lasting 1-2 minutes associated with urinary incontinence.

He has vomited twice in the ED and is complaining of muscle aches.

On examination the patient is agitated, confused and appears to be hallucinating. Myoclonic jerks are present. The chest is clear to auscultation and oxygen saturations are 98% on room air. Heart sounds are normal, the pulse rate is 130bpm and the blood pressure is 161/84mmHg.

An ECG reveals sinus tachycardia. The pupils measure 8mm and are equal and reactive. The patient's temperature is 36.6ºC.

Blood tests reveal:

Hb	138 g/l
Platelets	362 * 10 /l9
WBC	11.2 * 10 /l9
Na +	135 mmol/l
K +	2.7 mmol/l
Urea	12.6 mmol/l
Creatinine	187 µmol/l
Bilirubin	18 µmol/l
ALP	98 u/l
ALT	53 u/l
γGT	27 u/l
Albumin	32 g/l

What is the most likely cause for this patient's presentation?

A. Toluene solvent toxicity
B. Amyl nitrate toxicity
C. Gamma hydroxybutyric acid toxicity
D. Synthetic cannabinoid toxicity
E. Heroin toxicity

D. Synthetic cannabinoid toxicity

The hallucinations, seizure, myoclonus, mydriasis, hypertension, acute kidney injury and hypokalaemia are consistent with synthetic cannabinoid toxicity.

Features of toluene toxicity can be similar however irritation to the eyes, nose and respiratory tract is often seen from inhalation in toluene abuse.

Amyl nitrate is a vasodilator so toxicity tends to result in hypotension rather than hypertension as in this case. Other features of amyl nitrate toxicity include blurred vision, xanthopsia and haemoptysis.

Features of gamma hydroxybutyric acid (GHB) toxicity include CNS and respiratory depression, hypersalivation, bradycardia and hypotension.

Heroin toxicity typically results in depression of the respiratory and central nervous systems and pin-point pupils (rather than mydriasis in this case).

Question 64 of 138

A 69-year-old man with a history of chronic obstructive pulmonary disease is admitted to the Emergency department with an exacerbation. His GP has started ciprofloxacin some 3 days earlier. Other medication of note includes high dose salmeterol fluticasone, oral theophylline and tiotropium. Over the past 24hrs he has begun to vomit and feels light-headed. Of note is that his blood pressure is 132/88 mmHg with a postural drop of 15 mmHg, and his serum potassium is 3.0 mmol/l.

Which of the following is the most appropriate intervention?

A. Stop salmeterol fluticasone
B. Stop theophylline
C. Stop tiotropium
D. Stop ciprofloxacin
E. Start oral potassium replacement

B. Stop theophylline

Stop theophylline when CYP450 inhibitors are prescribed

Theophylline is metabolised by the CYP1A2 isoenzyme. Ciprofloxacin is a moderate inhibitor of CYP1A2, which can drive accumulation of theophylline. Symptoms of toxicity include nausea, vomiting, hyperventilation and marked hypokalaemia, as is seen here. The most important intervention is withholding of the theophylline.

Metabolism of salmeterol and tiotropium is not affected by co-administration of ciprofloxacin as such there is no reason for discontinuing these medications. Starting oral potassium replacement won't affect the build up of theophylline and won't therefore prevent further symptoms of theophylline toxicity. Clozapine, olanzapine, ropinirole and duloxetine are other examples of drugs metabolised by CYP1A2, accumulation of which may be driven by ciprofloxacin.

Question 65 of 138

A 20-year-old female presents having taken 20 aspirin 500mg tablets in a suicide attempt. She has felt well since and is asymptomatic, and now regrets these actions. She has a background of borderline personality disorder for which she takes olanzapine and has never attempted suicide in the past. She was seen by her GP with a presumed respiratory tract infection one week ago and prescribed amoxicillin. Cardiovascular, respiratory and abdominal examinations are all unremarkable. Her blood gas reveals a ph 7.41, lactate 0.5mmol/L and laboratory bloods are unremarkable other than a salicylate level 4 hours post ingestion of 660mg/L.

Which management step should be taken next?

A. Intravenous (IV) Sodium Chloride 0.9%
B. Reassure and discharge
C. IV Sodium bicarbonate 1.26%
D. N-acetylcysteine infusion
E. Dialysis

C. IV Sodium bicarbonate 1.26%

Although this young lady demonstrates no obvious compromise from her overdose, guidelines suggest that salicylate levels greater than 450mg/L in adults represents moderate poisoning. Sodium bicarbonate used here alkalinises the urine which is more effective than a massive diuresis. Urine pH should be greater than 7.5, ideally 8.0-8.5.

Question 66 of 138

A 77-year-old woman is admitted to the acute medical ward with a 3-day history of right loin pain and dysuria. A diagnosis of pyelonephritis is made and she is treated with intravenous gentamicin and ceftazidime.

Gentamicin levels are taken 8 hours post-dose:

Trough level	3.1 mg/L	(1 - 2)

What is the most appropriate next step in the management of this patient?

A. Continue with dose and prescribe IV 0.9% sodium chloride
B. Delay next dose of gentamicin
C. Reduce dose of gentamicin
D. Stop gentamicin
E. Switch antibiotic to ciprofloxacin

B. Delay next dose of gentamicin

For multiple daily dose regimens of gentamicin, if the pre-dose ('trough') concentration is high, the interval between doses must be increased. If the post-dose ('peak') concentration is high, the dose must be decreased

Patients receiving gentamicin therapy should have both pre-dose (trough) and post-dose (peak) levels checked. The recommended trough levels should be 1-2 mg/L, whilst the peak levels should be between 5-10 mg/L. A raised trough level means that there should be a **delay in the next dose of gentamicin**.

Continuing with the dose and prescribing IV 0.9% sodium chloride is inappropriate as this means that the patient could be receiving a harmful dose that could cause toxicity such as renal or otic impairment.

In contrast to raised trough levels, a raised peak level means that there should be a **reduced dose of gentamicin**.

There is no need to **stop gentamicin** at this stage. Instead, a raised trough level indicates an increased drug dosage interval as per the BNF. If the drug were to no longer be indicated or was evidenced to be causing significant harm or impairment in renal function, then careful discussion regarding its continuation would be needed.

Switch antibiotic to ciprofloxacin is incorrect. Rather than changing antibiotics, a raised trough level indicates that an increased dosing interval before the next administration of gentamicin is needed. It would be inappropriate to change antibiotics as there are risks for antimicrobial resistance.

Question 67 of 138

A 19 year old girl is brought to the Emergency Department by a friend. She is a first year university student and her friends are concerned her behaviour has altered in the past few weeks. On assessment she is confused with an AMTS of 6/10. She has a flat affect. Cardiorespiratory examination is normal although the respiratory rate is 22 at rest. Oxygen saturations are 97% on air. Heart rate is 85bpm regular and blood pressure is 100/55 mmHg.

Examination of her neurological system reveals prominent paraesthesia in both feet with absent vibration sense and proprioception. The ankle and knee reflexes are absent and plantar responses are extensor. There is mild distal weakness in the legs with MRC grading 4/5. Examination of the cranial nerves shows no evidence of nystagmus but visual acuity is reduced to 6/20 bilaterally. She has an ataxic gait.

Her medical history is significant only for coeliac disease and she is taking no regular medications. Routine blood results show:

Haemoglobin	7.6g/L	Sodium	137mmol/L	Magnesium	0.81mmol/L
Cell volume	103.2 fL	Potassium	4.6mmol/L	CRP	4.5mg/L
White cells	5.9x10^9/L	Urea	1.9mmol/L	Thyroid tests	Normal
Neutrophils	4.0x10^9/L	Creatinine	55mol/L	HIV	Negative
Lymphocytes	1.9x10^9/L	Calcium (corr)	2.43mmol/L	Syphilis	Negative
Platelets	300x10^9/L	Phosphate	1.01mmol/L		

The patient's friend tells you she has recently started using recreational party drugs.

Which of the following inhaled recreational drugs most explains the above picture?

A. Crack (cocaine)
B. H (heroin)
C. Hippie crack (nitrous oxide)
D. Poppers (amyl nitrate)
E. Spice (synthetic cannabinoid

C. Hippie crack (nitrous oxide)

Nitrous oxide use can precipitate severe vitamin B12 deficiency with pronounced neurological and haematological signs, particularly in susceptible individuals

The above vignette describes a state of profound vitamin B12 deficiency with clinical signs of subacute combined degeneration of the cord. The constellation of signs of ataxia, loss of deep tendon reflexes with dorsal column signs (loss of proprioception and vibration sense) and paraesthesias with positive Babinski response are known as myelosis funicularis and is highly suggestive of B12 deficiency. In this patient there are also signs of confusion and of reduced visual acuity which increase likelihood of B12 deficiency.

Nitrous oxide, also known as nitrous, hippie crack, laughing gas or whippet, is a relatively recent addition to the arsenal of recreational drugs of abuse. It is sold as cartridges to the catering industry for use in making whipped cream, but can be released and inhaled directly or through balloons or bags. Erroneously regarded as a safe drug it is reported to cause euphoric symptoms in users with a brief onset of actions. It is used clinically as a dental anaesthetic and anxiolytic and hence also promotes feelings of relaxation, detachment and sensory changes. Deaths have been reported due to hypoxia since catering nitrous oxide is not mixed with oxygen as it is when used medically.

Nitrous oxide can cause a rapid depletion in vitamin B12 stores in users, particularly in susceptible individuals such as vegans, anorexics, coeliac patients and those with problems with intestinal absorption. Nitrous oxide reacts with cobalt in the vitamin B12 molecule converting it from an active to an inactive bivalent form, thereby rendering it unusable in vivo. Symptoms of rapid neurological and haematological deterioration can occur including subacute combined degeneration of the cord, depression, dementia, psychosis and blindness, as well as megaloblastic anaemia. Chronic use can lead to pancytopaenia. In the acute phase, symptoms are reversible on cessation of the drug and high dose vitamin B12 supplementation. Chronic users may suffer irreversible neurological damage.

Poppers (amyl nitrate) cause euphoria and sensory dissociation on inhalation but long term effects are rare. A serious complication of their use is the oxidation of ferrous ions within haemoglobin molecules to ferric ions, creating methaemoglobinaemia, reducing the oxygen delivering capacity of the blood. They are not indicted in vitamin deficiencies.

Crack cocaine causes myriad sympathomimetic and serotonergic effects in users but is not directly attributable to anaemia or avitaminosis although this can occur secondarily in the serotonin syndrome. Crack cocaine can also cause choreoathetoid movements in some users. Similarly, heroin causes analgesic and depressant effects at opioid receptors but is not attributable directly to haematological or neurological symptoms although these may be seen in long term users due to self neglect. Spice is a synthetic cannabinoid drug with similar but augmented effects to cannabis. CNS effects of its use include agitation, confusion, psychosis, convulsions, nystagmus and blindness. It is rare for haematological effects to occur although renal failure may be seen.

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A 42-year-old woman is admitted for dysuria, fever and rigors that have developed over the past 24 hours. A urine sample is sent to the lab and cultures demonstrate sensitivity to gentamicin. Her renal function is normal and so she is commenced on a typical loading regime.

She is stable on a twice-daily regime of gentamicin for several days. Blood tests are measured one day, pre-and post-evening dose:

Pre-dose	0.6mg/L	(<1)
One hour post-dose	7mg/L	(3-5)

What is the most appropriate action to take?

A. Decrease the dose
B. Decrease the time interval between doses
C. Increase the time interval between doses
D. Recheck levels around the next dose
E. Withhold the next dose

A. Decrease the dose

For multiple daily dose regimens of gentamicin, if the pre-dose ('trough') concentration is high, the interval between doses must be increased. If the post-dose ('peak') concentration is high, the dose must be decreased

The correct answer is to decrease the dose. The post-dose ('peak') concentration is high. As such, the dose is too high and so the **dose should be reduced**.

Decreasing the time interval between doses is incorrect. This would further increase the gentamicin levels, which is inappropriate.

Increasing the time between doses is also incorrect. This would be appropriate if the pre-dose ('trough') level was too high, not the post-dose level.

Rechecking levels is not sufficient. Action needs to be taken given the result - the dose needs reducing.

Withholding the next dose would be an appropriate step if both pre-dose and post-dose levels were too high. The patient should then be rediscussed with microbiology.

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You are working in the renal ward when you are asked to urgently see a 70-year-old woman. She has just returned from a tunnelled central venous catheter insertion for a planned start on dialysis and has developed tachycardia and hypotension. You note that the procedure report suggests a difficult insertion poorly tolerated by the patient. On return to the ward, she complained to the nurse of numbness around her mouth and tingling of her tongue.

She has type 2 diabetes mellitus with nephropathy and atrial fibrillation. Medication includes alfacalcidol, bisoprolol, bumetanide, and edoxaban.

Observations are as follows.

- Respiratory rate 30
- Oxygen saturations 94% on air
- Heart rate 180 beats per minute
- BP 100/65 mmHg

- T 37.4ºC

ECG shows a regular broad complex tachycardia at 180 beats per minute. An urgent blood gas shows results as follows.

Na +	136 mmol/L	(135 - 145)
K +	5.0 mmol/L	(3.5 - 5.0)
Glucose	6.1 mmol/L	(3.3 - 5.8)

What is the most appropriate immediate course of action?

A. Administer 10% calcium gluconate and organise urgent haemodialysis
B. Administer IM glucagon
C. Administer IV lipid emulsion
D. Commence IV antibiotics for line infection
E. Urgent DC cardioversion

C. Administer IV lipid emulsion

Local anaesthetic toxicity can present with CNS (e.g. reduced GCS, seizures) and cardiovascular (e.g. tachyarrhythmias, hypotension, bradyarrhythmias) features. Treatment of severe toxicity is with IV lipid emulsion.

The correct answer **administer IV lipid emulsion**. This patient has likely developed local anaesthetic toxicity during the line insertion. Early clues include circumoral numbness, tongue paraesthesia, and dizziness. Patients can then develop CNS toxicity including drowsiness and coma. In this case, the patient has developed cardiovascular toxicity which can include arrhythmias such as ventricular tachycardia. The treatment of choice is IV lipid emulsion, which is thought to bind the local anaesthetic in the serum, therefore facilitating its clearance from the tissues in which it has a toxic effect.

Administer 10% calcium gluconate and organise urgent haemodialysis is incorrect. This would be the treatment for severe hyperkalaemia in a dialysis-dependent patient. While this can lead to ventricular tachycardia, the normal potassium on the blood gas is reassuring.

Administer IM glucagon is incorrect. This is a treatment option for beta-blocker overdose, which typically presents with bradycardia and resultant hypotension. Tachyarrhythmias are not a feature. It can also be used in hypoglycaemia but this would be unlikely to result in the clinical picture described. The glucose on the blood gas is also normal.

Commence IV antibiotics for line infection is incorrect. This would be very early for line infection to have developed and the lack of fever goes against this differential. Sepsis obviously could result in hypotension but would be unlikely to cause VT, which is the primary cause of the haemodynamic instability in this case.

Urgent DC cardioversion is incorrect. This would be the treatment of choice for any tachyarrhythmia with shock, defined by the resuscitation council as a systolic BP <90 mmHg. This patient's blood pressure is above this so other measures should be pursued first, particularly in the context of a toxic cause with a recognised antidote available.

Question 70 of 138

A 48-year-old man is reviewed in the Medical Admissions unit complaining of palpitations. He has a past medical history of hereditary obstructive cardiomyopathy (HOCM), for which he has taken amiodarone for the last year. He has no personal or family history of autoimmune disease.

On examination, he is flushed and diaphoretic. He appears thin, and he has a fine tremor when his hands are outstretched. There is no discernible goitre.

His observations are as follows: heart rate 102/min; blood pressure 126/82 mmHg; respiratory rate 14/min; temperature 37.9ºC; oxygen saturations 97% on air.

Selected blood test results are shown below:

Free T4	28.6 pmol/L	(12 - 22)
Free T3	9.1 pmol/L	(3.1 - 6.8)
TSH	< 0.05 mU/L	(0.27 - 4.2)

Given the likely diagnosis, what is the most appropriate management option?

A. Carbimazole
B. Lithium
C. Lugol's iodine
D. Prednisolone
E. Propylthiouracil

D. Prednisolone

In amiodarone induced thyrotoxicosis type 2 the management is with corticosteroids rather than carbimazole

The correct answer is **prednisolone**. This patient has a likely diagnosis of amiodarone-induced thyrotoxicosis (AIT) type 2. The thyroid function tests are in keeping with thyroid overactivity, whilst the examination findings clearly describe him as clinically thyrotoxic. In the absence of an alternative explanation (e.g. known Graves disease), this is most likely due to the patient taking amiodarone. The diagnosis of AIT type 2 is more likely than AIT type 1, as AIT type 2 is a destructive thyroiditis that tends not to present with goitre formation. The initial management of AIT type 2 is with corticosteroids such as prednisolone.

Carbimazole is incorrect. This would be an appropriate initial management option for this patient if he had a goitre on examination, and hence if the clinical picture were more consistent with AIT type 1. Antithyroid drugs are favoured in AIT type 1, where the pathophysiology involves excess thyroid hormone synthesis stimulated by the iodide load from amiodarone therapy (the Jod-Basedow phenomenon).

Lithium is incorrect. This would be an appropriate management option for either AIT type 1 or type 2 that was refractory to initial therapy (with either antithyroid drugs or corticosteroids respectively).

Lugol's iodine is incorrect. Lugol's iodine is used for its antithyroid properties in the management of severe thyrotoxicosis, and as a preparation for thyroidectomy surgery in patients with Graves disease. It is not routinely used in the management of AIT type 2, where the anti-inflammatory properties of corticosteroids are preferred.

Propylthiouracil is incorrect as corticosteroids, rather than antithyroid drugs are the first-line management option for AIT type 2.

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You are called to the emergency department to review a 33-year-old man. He is an Estonian national and works on a farm. His spoken English is poor but he tells you he feels unwell. He is being treated in a side-room because of loose stools.

On examination he is drooling slightly and has watery eyes. His pupils are equal in size but miotic. His blood pressure is 102/58 mmHg and an ECG shows sinus bradycardia.

How should this patient be managed? :

A. Amiodarone
B. Pyridostigmine
C. Glucagon
D. Haemodialysis
E. Atropine

E. Atropine

Organophosphate insecticide – atropine

This man has features of organophosphate poisoning and demonstrates some of the anticholinesterase effects exerted by industrial insecticides. The only suitable treatment listed is atropine. Atropine exerts an anticholinergic effect at the level of the muscarinic receptor, thus opposing the effects of accumulated acetylcholine.

Amiodarone is not typically used to treat bradyarrhythmias. Glucagon can be used in beta-blocker overdose, but beta blockade would not cause the miotic pupils or lacrimation. Pyridostigmine is an anticholinesterase agent used in the treatment of myasthenia gravis; it would do nothing to help in this situation.

Organophosphates are not amenable to haemodialysis (remember the 'BLAST' mnemonic for overdoses that can be dialysed:

Barbiturates Lithium

Alcohols e.g. ethylene glycol

Salicylates Theophyllines)

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A 17-year-old woman with a history of epilepsy and deliberate self-harm is brought into the emergency department with a Glasgow coma score of 13 (E3 V4 M6) and respiratory rate of 8/min.

On examination, her pulse is 56/min and regular, blood pressure 110/60 mmHg and her chest is clear. She has no signs of injury but an empty packet of diazepam was found in her handbag. Whilst the patient is breathing room air a nurse in the emergency department has taken an arterial blood gas shown below.

pH	7.39
pO2	10.1 kPa
pCO2	5.6 kPa
BE	0.8 mEq/l

What is the best initial management?

A. Flumazenil 200mcg infused IV over 15 minutes
B. Flumazenil 200mcg bolus IV over 15 seconds
C. Naloxone 400mcg IM injection
D. Urgent intubation with anaesthetic support
E. Supportive care only

E. Supportive care only

The blood gas shows her ventilation appears inadequate but at this stage the best management is supportive, allowing levels of diazepam to reduce over time, something that can be easily assessed clinically with and with the aid of an arterial blood gas sample.

Administering the benzodiazepine antidote flumazenil to an epileptic puts her at high risk of seizures. Naloxone is an antidote to opiate overdose. If however despite simple manoeuvres her ventilation deteriorated or GCS dropped to less than 8, anaesthetic support should be sought.

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A 55-year-old woman presents to the emergency department with shortness of breath. She has no significant past medical history and does not have any regular medications. She smokes ten cigarettes daily and lives alone.

Her observations are heart rate 110 beats per minute, blood pressure 120/88 mmHg, respiratory rate 18/minute, oxygen saturations 97% on 4L of oxygen and temperature 37ºC.

Clinical examination is unremarkable.

Blood tests:

Hb	138 g/L	Male: (135-180)Female: (115 - 160)
Platelets	180 * 10 /L 9	(150 - 400)
WBC	5.2 * 10 /L 9	(4.0 - 11.0)
Na +	138 mmol/L	(135 - 145)
K +	4.2 mmol/L	(3.5 - 5.0)
Urea	4.2 mmol/L	(2.0 - 7.0)
Creatinine	88 µmol/L	(55 - 120)
CRP	4 mg/L	(< 5)
D-dimer	2000 ng/mL	(<250)
Trop	8 ng/L	(<15)

A chest x-ray is normal.

A CTPA demonstrates a large saddle embolus. There is no CT or echocardiographic evidence of right heart strain.

She is commenced on a heparin infusion. She successfully manages to wean oxygen by day 5 of treatment and has some repeat blood tests prior to a switch to an oral anticoagulant.

Blood tests:

Hb	136 g/L	Male: (135-180)Female: (115 - 160)
Platelets	32 * 10 /L 9	(150 - 400)
WBC	4.6 * 10 /L 9	(4.0 - 11.0)

Given the likely diagnosis, what is the most suitable medication to switch to from the heparin infusion?

A. Abciximab
B. Argatroban
C. Enoxaparin
D. Platelet transfusion
E. Warfarin

B. Argatroban

Heparin-induced thrombocytopenia: anticoagulation can be provided by direct thrombin inhibitor e.g. argatroban

Argatroban is the correct answer. The patient has heparin-induced thrombocytopenia, as evidenced by a > 50% drop in platelets with a nadir platelet count > 20 * 10 /L, occurring between days 5-10 post heparin administration, in the absence of another cause. She does not have any clinical evidence of thrombosis clinically but is at high risk of thrombotic events. Argatroban is a small molecule direct thrombin inhibitor and it is given intravenously. Patients can be transitioned to an oral anticoagulant once the platelet count has recovered.

Abciximab is incorrect. This is a glycoprotein IIb/IIIa receptor antagonist and is used as an antiplatelet medication rather than an anticoagulant.

Enoxaparin is incorrect. This is a rarer cause of induced-thrombocytopenia but is not recommended in the management of this condition.

Platelet transfusion is incorrect. The patient is not bleeding and the platelet count is > 30. Therefore platelets are not indicated. Indeed, they may increase thrombotic risk.

Warfarin is incorrect. The patient can be transitioned from argatroban to warfarin once the platelet count has recovered.

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Sonia is a 24-year-old woman presenting to the emergency department after being brought in by her flatmate. Sonia was found collapsed on the ground surrounded by unmarked bottles. She was noted to have been arguing with her boyfriend earlier today and given her history of depression, her friend thought she should have checked to ensure Sonia was safe. She is unsure is the bottles contained medications or other liquids.

Sonia was initially significantly disorientated on presentation and could not accurately recall where she was or why she was in the hospital. She began to experience nausea and vomiting while in the emergency department. After 1 hour in the emergency department, she became incredibly drowsy and became unrousable.

Physical examination was otherwise unremarkable. Skin colour was relatively normal. There was no tenderness on palpation of her abdomen. She is tachypnoeic with a respiratory rate of 30 breaths/minute.

Blood tests are as follow:

Hb	140 g/L	Male: (135-180)Female: (115 - 160)
Platelets	250 * 10 /L 9	(150 - 400)
WBC	7.0 * 10 /L 9	(4.0 - 11.0)
Na +	138 mmol/L	(135 - 145)
K +	4.2 mmol/L	(3.5 - 5.0)
Urea	5.0 mmol/L	(2.0 - 7.0)
Creatinine	100 µmol/L	(55 - 120)
CRP	<5 mg/L	(< 5)
Bicarbonate	12 mmol/L	(22 - 29)
Chloride	98mmol/L	(95-105)
Measured osmolarity	350mOsm/kg	(275-295)
Bilirubin	14 µmol/L	(3 - 17)
ALP	60 u/L	(30 - 100)
ALT	30 u/L	(3 - 40)
Î³GT	40 u/L	(8 - 60)
Albumin	34 g/L	(35 - 50)

Arterial blood gases showed:

pH	7.27	7.35-7.45
pO2	13.7 kPa	11-13 kPa
pCO2	3.2 kPa	4.7 - 6.0kPa
HCO3	12	22-26
BE	-4	-2 - +2

An ECG was performed and this showed sinus tachycardia. No abnormalities of the QRS complex was noted.

What is the likely agent that had been taken?

A. Aspirin
B. Ethylene glycol
C. Iron tablets
D. Organophosphate pesticide
E. Paracetamol

B. Ethylene glycol

Metabolic acidosis with a high anion gap and high osmolar gap is seen in ethylene glycol poisoning

The patient has taken an overdose of an unknown substance. Her only symptoms included nausea, vomiting before progressing to drowsiness and stupor, all relatively non-specific.

Her blood tests do provide additions information as this shows a metabolic acidosis from her arterial blood gas. Further assessment of her blood tests reveals an underlying raised anion gap along with a raised osmolar gap. Osmolar gap is calculated by measured serum osmolarity - calculated serum osmolarity (2x Na + glucose + urea). A gap of >=10 suggests an elevated osmolar gap.

Causes of a raised osmolar gap are alcohol ingestion (including methanol and ethylene glycol), sugars (mannitol and sorbitol), lipids (hypertriglyceridemia) and proteins (hypergammaglobulinemia). Given the combination of her blood tests and symptoms, ethylene glycol remains the likely substance ingested.

Aspirin can cause metabolic acidosis with a raised anion gap. However, it will be associated with more symptoms including tinnitus and fevers. Also, it is not associated with a raised osmolar gap.

Iron overdose will present with other symptoms including haematemesis and abdominal pains. It can present with a raised anion gap metabolic acidosis.

Paracetamol overdose will not result in the blood tests seen. Also, it can present with hepatic dysfunction before progression to hepatic encephalopathy in severe cases.

Organophosphate poisoning is unlikely as the patient does not display any clear cholinergic symptoms including salivation, lacrimation or diarrhoea.

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A 32-year-old mechanic is admitted to the emergency department. He is drowsy and confused. He is accompanied by his girlfriend who informs you they had been drinking alcohol the previous night. She became concerned when he appeared increasingly confused throughout the day despite no further alcohol intake. She tells you his mood has been low over the last number of months and has recently expressed suicidal ideation.

On your assessment his Glasgow coma scale is 14/15, pupils are equal and reactive to light. His Blood pressure is 124/81mmHg with a heart rate of 97/minute. Oxygen saturations are 94% on room air and he is tachypnoeic with a respiratory rate of 32/minute. There is bilateral flank tenderness, the clinical exam is otherwise unremarkable.

His urine toxicology screen is clear. Urine dipstick is positive for blood. Arterial blood gas:

PH	7.10
PO2	12.3Kpa
PCO2	2.6Kpa
Bicarbonate	8.0mEq/L
Lactate	2.3mmol/L

Biochemistry:

Na +	145 mmol/L	(135 - 145)
K +	3.8 mmol/L	(3.5 - 5.0)
Cl +	105 mmol/L	(98-106)
Urea	11mmol/L	(2.0 - 7.0)
Creatinine	113µmol/L	(55 - 120)
Glucose	5.7mmol/L	(3.9-5.8)
Serum Osmolality	358mOsm/Kg	(275-295)
Ethanol	<10mmol/L	
Paracetamol	0mg/dl	

Which of the following is the most likely cause for this man's presentation?

A. Alcoholic ketoacidosis
B. Ethylene glycol toxicity
C. Alcohol intoxication
D. Methanol poisoning
E. Paracetamol overdose

B. Ethylene glycol toxicity

Metabolic acidosis with a high anion gap and high osmolar gap is seen in ethylene glycol poisoning

This question requires you to identify a metabolic acidosis with a high anion gap and a high osmolar gap. This can be seen due to the presence of several different compounds or their toxic metabolites (glycols, methanol, oxoproline, lactate, ketones, aspirin, etc). In this case, the clinical history allows you to identify the likely cause.

Ethylene glycol and methanol are commonly found in automotive coolant, antifreeze, windshield wiper fluid, solvents, cleaners, and fuels. This patient is a mechanic with easy access to these compounds. Both methanol and ethylene glycol are metabolized by alcohol dehydrogenase (ADH) before having their effects via toxic metabolites. Ethanol competitively inhibits ADH, thereby diminishing the formation of toxic metabolites of the parent compound. Our patient had been drinking alcohol the previous night which may have masked his early symptoms and delayed the formation of toxic metabolites.

The clinical findings can help differentiate between methanol and ethylene glycol toxicity. Advanced methanol toxicity is associated with retinal injury and visual disturbance. Ethylene glycol more commonly presents with oliguria, hematuria, and flank pain. This patient had microscopic haematuria and bilateral flank pain. Definitive diagnosis can be made with direct testing of toxic alcohols or by identifying the consumed substance.

Calculating the anion gap:

Anion gap mmol/L = sodium mmol/L - (chloride mmol/L + bicarbonate mmol/L). Calculated anion gap = 32 (normal range 8->12).

Calculating the osmolar gap:

Osmolal gap = measured serum osm (358) - calculated serum osm (307). Serum osmolality = (2 Ã— Na + (BUN / 2.8) + (glucose / 18) + (ethanol / 3.7). Calculated serum osmolality = 307 mOsm/kg.

Measured serum osmolality = 358mOsm/kg.

Osmolal gap = 51(normal range (-10) - (+10).

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A 47-year-old Latvian man is brought to the Emergency Department by two of his friends. He is confused and aggressive and walks with an unsteady gait. History is difficult to obtain from the patient due to slurring of his speech and a slight language barrier. You note that he fails to meet your gaze when you are talking to him. Examination shows a GCS of 13/15 but he is drowsy and irritable. There is a vague smell of alcohol on his breath. He is flushed in the face and his eyes are red and hyperaemic. Pupils are slow to react to direct light but equal in size. The cardiorespiratory examination is unremarkable aside from a respiratory rate of only 8/min but SpO2 is 98% on air. The abdomen is soft and non-tender with no hepatomegaly and no signs of chronic liver disease. Despite the department being well lit, he asks you to turn the lights on.

Biochemistry reveals:

Haemoglobin	149g/L	Sodium	150mmol/L	pH	7.25
White cells	8.9x10 /L 9	Potassium	5.9mmol/L	pC02	3.9kPa
Neutrophils	5.9x10 /L 9	Urea	13.8mmol/L	pO2	12.6kPa (air)
Platelets	229x10 /L 9	Creatinine	156mol/L	HCO3-	12.5mmol/L
Prothrombin time	11 sec	Glucose	24.7mmol/L	BE	-13.2mEq/L
Ethanol	110mg/dL	Chloride	100mmol/L	Lactate	2.0mmol/L
Anion gap	38	Osmolar gap	25 (-10 to +15)		

What is the most appropriate next step in the management of this patient?

A. Begin high dose oral folic acid and vitamin B complex
B. Establish IV access and begin fast fluids with Pabrinex I+II (Vitamin B12)
C. Establish IV access and send blood for methanol levels; give oral ethanol in the form ofneat 40% whisky at a dose of 175ml
D. Establish IV access and send blood for methanol levels; begin IV fomepizole 1000mg in500ml 5% dextrose over 30 minutes
E. Refer to Intensive Care to begin haemofiltration

D. Establish IV access and send blood for methanol levels; begin IV fomepizole 1000mg in500ml 5% dextrose over 30 minutes

Methanol poisoning is preferentially treated with IV fomepizole; this should be commenced immediately the suspicion is raised

The clinical picture in this scenario is typical of an acute alcohol intoxication syndrome with descriptors of ataxia, confusion, aggression and drowsiness. Often there will be a smell of alcohol present when examining the patient and unfortunately, a coherent history is often absent due to poor compliance and often incapacity. However, in the vignette, there are features which might not be expected with a case of ethanol intoxication such as difficulties with vision, often detected by the failure of making any eye contact or of requesting extra lighting in a well-lit room. This suggests damage to the optic nerve and is a feature of methanol poisoning. Methanol toxicity is often very difficult to distinguish from ethanol intoxication but visual symptoms should prompt consideration coupled with a careful history.

Similar to drinkable ethanol, methanol is rapidly metabolised by hepatic enzymes; ethanol dehydrogenase converts methanol to formaldehyde and aldehyde dehydrogenase further metabolises this to formic acid (formate) which is extremely toxic to the central nervous system. Accumulation of formate in the central nervous system leads not only to cellular hypoxia due to interruption of mitochondrial cytochrome activity but also a profound acidosis. Coupled with this methanol itself, similar to ethanol, causes central nervous and respiratory depression further augmenting its toxicity.

Typically bloods will show a metabolic acidosis with a high anion gap - often co-ingestion of ethanol will have occurred and this may account for some of the unmeasured ions but often the plasma ethanol level will not be sufficiently raised to explain it in totality. Calculation of the osmolar gap may also be elevated initially which is not seen so much with ethanol toxicity.

Treatment of methanol toxicity is by blockade of the metabolism of methanol to formate leading to renal clearance of the unmetabolised methanol. Both ethanol and fomepizole are competitive inhibitors of methanol metabolism as they preferentially have an affinity for both ethanol dehydrogenase and aldehyde dehydrogenase and so toxic formate is not produced. Fomepizole, although very expensive, is preferentially used over ethanol as there is a risk of precipitating ethanol toxicity with its associated central nervous and respiratory depression. If ethanol is used as an antidote, regular monitoring of plasma ethanol concentrations are needed to ensure maintenance within the range of 100-200mg/dL. In this case, plasma ethanol levels are within this range anyway and methanol toxicity is still occurring. Both fomepizole and ethanol need to be continued until plasma methanol concentrations fall below <50mg/L.

In addition, intravenous folinic acid should be given as this aids in the speedy metabolism of formic acid and helps in reducing the central nervous toxicity. Unfortunately, oral folic acid is insufficiently rapidly absorbed to be helpful in this situation. Supplementation of B vitamins is important in cases of any alcohol toxicity as this avoids development of Wernicke's encephalopathy and this should take the form of Pabrinex I+II, however, it is not the most pressing concern in a potential methanol toxicity case. Oral vitamin B complex is important in chronic alcoholism but not in an acute situation. Haemofiltration is a potential mechanism for clearing methanol from the circulation, however, this is usually reserved for occasions when fomepizole fails or is not tolerated.

In cases of methanol toxicity, an intensive care opinion may be useful however as central venous access may be required to administer bicarbonate in severe acidosis or in case of respiratory depression requiring intubation and assisted ventilation.

Note that in cases of suspected methanol poisoning, commencing an antidote immediately is important and should be done before methanol levels have been confirmed.

Question 77 of 138

A 30-year-old man was referred to endocrinology clinic for the assessment of the adverse consequences of anabolic steroid use. The patient reported that he had recently decided to stop using anabolic steroids, having been a regular user for the previous 5 years to support his bodybuilding training. The patient cited the potential health risks of long-term anabolic steroid use as the reason behind his decision to cease his use.

The patient had followed a regime of an intramuscular injection of long-acting synthetic testosterone derivative every two weeks. The patient took a 4-week break from anabolic steroids every 12 weeks, in an attempt to limit the adverse side-effects. The patient denied ever having used oral or topical synthetic testosterone preparations and had always used sterile injecting equipment.

The patient stated that he had developed significant gynaecomastia and also suffered significant male pattern baldness since starting to use anabolic steroids. In addition, 2 years previously he had suffered a ruptured right biceps tendon while exercising, requiring a surgical repair and had a prolonged period of rehabilitation. There was no history of symptoms suggestive of cardiac or liver disease. The patient took no regular prescribed medications and reported an alcohol consumption of between 15 to 20 units per week.

General examination of the patient revealed a highly muscular and lean adult male. Moderate gynaecomastia was present, but there were no other signs of chronic liver disease. Examination of the cardiovascular and respiratory systems was unremarkable.

Please see below for the available results of blood tests taken prior to the patient's attendance at the clinic.

HbA1C	47 mmol / mol (reference < 42)
Total cholesterol	6.1 mmol / L (reference < 5.0)
Fasting LDL cholesterol	5.0 mmol / L (reference < 3.0)
Fasting HDL cholesterol	1.1 mmol / L (reference > 1.2)
Prolactin	490 mU / L (reference 80 - 400)
Luteinising hormone	*result pending*
Follicle-stimulating hormone	*result pending*
Testosterone	*result pending*
Epitestosterone	*result pending*

What pattern of results for the pending blood tests is consistent with the patient's anabolic steroid use?

A. Elevated testosterone:epitestosterone ratio; normal luteinising hormone; normal folliclestimulating hormone

B. Suppressed testosterone:epitestosterone ratio; elevated luteinising hormone; suppressedfollicle-stimulating hormone

C. Suppressed testosterone:epitestosterone ratio; suppressed luteinising hormone;suppressed follicle-stimulating hormone

D. Elevated testosterone:epitestosterone ratio; suppressed luteinising hormone; suppressedfollicle-stimulating hormone

E. Normal testosterone:epitestosterone ratio; elevated luteinising hormone; elevated folliclestimulating hormone

D. Elevated testosterone:epitestosterone ratio; suppressed luteinising hormone; suppressedfollicle-stimulating hormone

The patient has developed some of the typical unwanted effects associated with anabolic steroid use; namely gynaecomastia and accelerated male pattern baldness. In addition, tendon rupture is frequently associated with anabolic steroid use due to the disproportionate enhancement of muscle bulk compared to tendon strength. Other common unwanted effects include acne, testicular atrophy (or clitoral hypertrophy in females), impaired glucose tolerance and a deranged blood lipid profile. More worryingly, anabolic steroid use is also associated with serious liver disease and an increased mortality from heart disease.

Epitestosterone is a naturally occurring stereoisomer of testosterone produced by the testes. In healthy males, the testosterone:epitestosterone ratio is typically 1:1. However, exogenous administration of testosterone does not increase levels of epitestosterone, so anabolic steroid use is associated with an increase in the testosterone:epitestosterone ratio (typically to greater than 4:1). In anti-doping testing for competitive sports, an elevated testosterone:epitestosterone ratio is considered possible evidence of anabolic steroid use.

Physiological production of testosterone is promoted by the release of the gonadotropins luteinising hormone and follicle stimulating hormone by the pituitary gland. Both these hormones increase the number of testosterone-producing Leydig cells in the testes, with luteinising hormone also increasing testosterone production by Leydig cells. Elevated testosterone levels reduce gonadotropin production via negative feedback to the hypothalamus (causing reduced gonadotropin releasing hormone production) and the pituitary gland. Therefore, high levels of exogenous testosterone will act to suppress luteinising hormone and follicle stimulating hormone.

Question 78 of 138

A 64-year-old man presents with several months of weight loss, anxiety, and palpitations. His wife mentions that he keeps the house temperature cool as he is generally feeling too hot. He has a background of ventricular tachycardia, for which he has been taking amiodarone for some years.

On examination, he has a slight tremor and appears thin. His heart rate is 105/min. There is no goitre present.

Blood tests are taken:

Thyroid stimulating hormone (TSH)	<0.05 mU/L	(0.5-5.5)

Free thyroxine (T4)	25 pmol/L	(9.0 - 18)

He is subsequently referred for an ultrasound scan of the neck, which shows absent vascularity and probable gland destruction.

What is the most appropriate treatment?

A. Carbimazole
B. Iodine
C. Prednisolone
D. Propranolol
E. Propylthiouracil

C. Prednisolone

In amiodarone induced thyrotoxicosis type 2 the management is with corticosteroids rather than carbimazole

This patient is likely experiencing amiodarone-induced thyrotoxicosis (AIT). This can be divided into two types. Type 1 results from excess iodine-induced thyroid hormone synthesis; a goitre is typically present. Type 2 results from amiodarone-related destructive thyroiditis and, as in this scenario, a goitre is absent. Differentiation of the two types is important as they have different management. The ultrasound scan here also shows probable gland destruction, pointing towards type 2. Management involves the use of corticosteroids and so **prednisolone** is correct.

Carbimazole is typically used for the treatment of hyperthyroidism and would be suitable for the management of type 1 AIT. However, this scenario is more likely to be occurring as a result of type 2 AIT and so prednisolone remains more appropriate.

Iodine may be used as part of the management of hyperthyroidism generally, as well as a thyroid storm. Definitive management for type 2 AIT, however, is corticosteroid use.

Propranolol is a beta-blocker and can be used for short-term symptom relief of hyperthyroidism. It does not play a role in the definitive management of type 2 AIT.

Propylthiouracil is another drug used in the management of hyperthyroidism. Again, with the management of type 2 AIT specifically, corticosteroids are more appropriate.

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A 45-year-old man was found unconscious at home by his partner, with empty packs of flecainide, codeine and valerian root tablets besides him. He was only found after his partner got worried when he had locked himself in his room and after 4 hours not heard any sounds from the room. He had been depressed for the past couple of months and relations between him and his partner had been strained. He has a past medical history of back pain, paroxysmal atrial fibrillation, angina and hypertension.

On examination, he was drowsy, unable to give a history, with a heart rate of 50bpm, respiratory rate of 16 breaths per minute, oxygen saturation of 96% on air, and a blood pressure of 110/58mmHg. Pupils were 3mm and equal and reactive to light.

Investigations:

Na+	136 mmol/l
K+	4.6 mmol/l
Urea	10.9 mmol/l
Creatinine	110 µmol/l
Serum bilirubin	30 µmol/l
Serum alkaline phosphatase	135 IU/l
Serum aspartate aminotransferase	50 IU/l
C Reactive protein	2mg/l
Haemoglobin	14.6 g/dl
White cell count	5.6 x 109/L
INR	1.4

ABG (on air):

pH	7.258
pO2	11.7 kPa
pCO2	3.4 kPa
Lactate	1.6 mmol/l
Base Excess	-8.4 mmol/l
Bicarbonate	11.9 mmol/l

ECG showed bradycardia with widened QRS complexes and giant inverted T waves. After fluid resuscitation,

what is the next single most important management step?

A. Activated Charcoal
B. Magnesium Sulphate
C. Sodium Bicarbonate
D. Haemodialysis
E. N-Acetyl Cysteine infusion (NAC)

C. Sodium Bicarbonate

This patient has overdosed on a number of medications, the most toxic being codeine and flecainide. He has evidence of cardiac arrhythmias, bradycardia and metabolic acidosis consistent with flecainide overdose, and also mildly deranged liver functions. There is no evidence of respiratory depression.

Flecainide is a class 1c antiarrhythmic, which blocks sodium channels leaving the action potential unchanged. Cardiac features predominate, causing bradycardia, hypotension, nodal and ventricular tachycardia. ECG abnormalities include marked prolongation of the QRS and Q-T interval and giant inverted T waves. Other features include hypoxia, respiratory depression, metabolic acidosis, and reduced renal function. Nausea, vomiting, coma and convulsions have been reported.

As the time of ingestion is unknown, possibly up to 4 hours ago, he has probably missed the window for gastric decontamination with activated charcoal. The presence of QRS prolongation and acidosis would require rapid correction with sodium bicarbonate infusions, ideally via a central line, to reverse the effects of flecainide.

Haemodialysis are of no value in eliminating flecainide from the system. Magnesium is indicated if there is evidence of a prolonged QT interval. NAC is indicated for paracetamol overdose.

Valerian root tablets are herbal sleeping remedies and do not have lasting side effects if taken in large quantities.

Question 80 of 138

A 59- year- old male presented with palpitations, excessive sweating and weight loss for two months. Two years ago he was diagnosed with atrial fibrillation and commenced on amiodarone.

On examination, his pulse rate is 80 beats per minute, irregularly irregular, and his blood pressure is 135/80. There was no goitre, no eye signs or hand signs.

Investigations reveal:

Serum free T4	60 pmol/l
Serum free T3	15 pmol/l (5-10)
Serum TSH	<0.05 mU/l
Serum antithyroid peroxidase	negative
TSH receptor antibodies	negative

Radioactive iodine uptake scan showed reduced uptake by the thyroid gland.

What is the most appropriate management for this patient?

A. Discontinue amiodarone and give carbimazole
B. Discontinue amiodarone and give potassium perchlorate
C. Discontinue amiodarone and give prednisolone
D. Radioactive iodine
E. Total thyroidectomy

C. Discontinue amiodarone and give prednisolone

The diagnosis is amiodarone induced thyrotoxicosis (AIT) type 2 which is a destructive thyroiditis treated with prednisolone.

AIT type 2 is differentiated from AIT type 1 by the reduced iodine uptake, absence of thyroid autoantibodies and absence of goitre.

AIT type 1 is treated with carbimazole or potassium perchlorate.

If amiodarone could not be stopped as in ventricular tachycardia, then total thyroidectomy should be considered.

Question 81 of 138

An 85-year-old female is admitted under the general medical unit with acute thoracic back pain from a T6 crush fracture following a fall. She has a past history of systolic heart failure, depression and osteoporosis.

Her regular medications included aspirin, frusemide, spironolactone, bisoprolol, sertraline and calcium, vitamin D and weekly alendronate. These are continued throughout her admission.

Two days into her admission, the nurses note that she is agitated and a bit confused.

On examination, she looks flushed and is tachycardic with a heart rate of 120 beats/min and is hypertensive with a blood pressure of 185/70 mmHg, but is afebrile. Both her pupils are mildly dilated, she is mildly tremulous and is noted to have deep tendon hyperreflexia with easily inducible clonus.

Use of which of the following analgaesic medication could explain her current symptoms?

A. Paracetamol
B. Ibuprofen
C. Oxycodone
D. Tramadol
E. Hydromorphone

D. Tramadol

Serotonin syndrome is a disorder characterised by serotonin excess, usually due to the use of 2 or more serotonergic drugs. Manifestations of the syndrome include changes in mental status, neuromuscular changes and autonomic overactivity. Clinically, this can be observed as hypertension, tachycardia, flushing and sweating, hyperflexia, clonus and muscle rigidity. Other potential signs include fever and changes in mental status, including agitation.

Serotonergic drugs that are associated with serotonin syndrome include tramadol, selective serotonin reuptake inhibitors (SSRI), monoamine oxidase inhibitors (MAOI), triptans and St Johns wort.

The management of serotonin syndrome involves discontinuation of all serotonergic drugs and

supportive care. If required, benzodiazepine can be administered to control agitation. In moderate to severe cases, 5-HT antagonists (e.g. cyproheptadine and chlorpromazine) are sometimes administered.

Question 82 of 138

A 63 year old farmer is brought to the Emergency Department after falling into a trough of organophosphate sheep dip. On arrival in the department he is in extremis with profuse vomiting and productive of copious respiratory secretions. His airway is threatened but he is also agitated and difficult to assess. Respiratory rate is 20 breaths per minute and oxygen saturations are 87% on 4L/min oxygen. Wheeze is audible from the end of the bed. The heart rate is 55bpm and the blood pressure is 92/44mmHg. Heart sounds are normal. He is incontinent of urine and faeces and has severe abdominal pain. There is widespread muscle fasciculation and global weakness. Pupils are pinpoint.

Which of the following best describes the toxic action of organophosphate compounds?

A. Adrenergic blockade due to competitive inhibition of α1, α2, β1 and β3 receptors
B. Adrenergic upregulation due to inhibition of monoamine oxidase
C. Cholinergic upregulation due to inhibition of acetylcholinesterase
D. Cholinergic upregulation due to direct activation at muscarinic M1, M2 and M3 receptors
E. Cholinergic upregulation due to direct activation at nicotinic CNS and muscle receptors

C. Cholinergic upregulation due to inhibition of acetylcholinesterase

Organophosphate poisoning occurs due to inhibition of acetylcholinesterase leading to upregulation of nicotinic and muscarinic cholinergic neurotransmission

Organophosphate insecticides are highly effective pesticidal agents. They work by binding to and irreversibly inactivating the enzyme acetylcholinesterase (AChE) which is essential for respiration in insects. Unfortunately, they also inhibit AChE in humans too, which exerts toxic effects when inadvertent exposure occurs occupationally, and this class of agents can also be used as a chemical weapon in the form of nerve gases.

Due to the non-specificity of acetylcholinesterase in cholinergic synapses, inhibition of the enzyme leads to upregulation of neurotransmission at all cholinergic synapses, giving rise to both nicotinic and muscarinic symptoms. Classically, the muscarinic symptoms seen can be remembered by the mnemonic SLUDGE: salivation, lacrimation, urination, defecation, gastrointestinal pain, emesis, and also pupillary constriction, bronchoconstriction and bradycardia. Nicotinic symptoms include headache, anxiety, fasciculations, weakness, ataxia, confusion and respiratory depression.

Sometimes there is variation in the extent of symptoms observed due to the relative upregulation of each neurotransmitter symptom with either predominant nicotinic symptoms or muscarinic ones, for example either bradycardia due to muscarinic overdrive or tachycardia due to nicotinic drive.

Knowledge of the pathophysiological effects of organophosphates is important because it guides treatment. Severe exposure to organophosphates may cause death within minutes due to respiratory failure. Provision of supplemental oxygen is important. The first life-saving intervention which may be provided is intravenous atropine which is a competitive antagonist at muscarinic receptors, predominantly subtypes M1, M2 and M3. This increases heart rate and improves respiratory function by relaxing bronchial smooth muscle and drying secretions. Atropine administration may need to be repeated multiple times at doses up to 2mg every five minutes until heart rate is above 80. Definitive treatment may be provided by administration of pralidoxime or obidoxime which are oxime drugs which bind to an allosteric moiety of AChE and causes a conformational change in the enzyme allowing it to be released from the organophosphate. Organophosphates irreversibly bind to AChE when in their physiological state but conformational change by oximes allows this to be undone. This is time dependent however and a process known as aging occurs whereby the longer the enzyme is inhibited the less likely the oxime is to be able to reanimate it. In warfare, soldiers are often supplied with auto-injectors containing atropine, pralidoxime and midazolam as an immediate response to exposure to nerve agents.

While exposure to organophosphates often produces hypotension and bradycardia with bronchoconstriction, they have no effect on adrenergic neurotransmission.

It is also important to note that organophosphate exposure is from environmental sources and as such decontamination is vital to prevent exposure to healthcare providers and appropriate personal protective equipment must be worn by rescuers.

Question 83 of 138

A 49-year-old woman with a diagnosis of Hashimoto's thyroiditis is seen in clinic with recurrence of lethargy, constipation, cold intolerance and pedal oedema. These symptoms had previously resolved on starting treatment with levothyroxine.

Her co-morbidities include type 2 diabetes mellitus and hypertension. She was also diagnosed with mycobacterium tuberculosis of the lung five months ago. Her current medications are levothyroxine, amlodipine, ramipril, metformin, gliclazide, rifampicin, isoniazid and pyridoxine.

Her temperature is 36.5°C, pulse 55 beats per minute, blood pressure 165/102 mmHg and respiratory rate 15 breaths per minute.

Thyroid function tests showed:

Free thyroxine (T4)	5 pmol/L (10-25)
Free triiodothyronine (T3)	3 pmol/L (5-10)
Thyroid-stimulating hormone	7.2 mU/L (0.4-5.0)

What is the likely cause of her symptoms?

A. Vitamin B6 deficiency
B. Rifampicin
C. Isoniazid
D. Non-compliance with medications
E. Amlodipine

B. Rifampicin

Rifampicin is a P450 enzyme inductor

In patients with treated hypothyroidism, thyroxine requirements are increased by cytochrome P450 inducing drugs such as rifampicin. Isoniazid does not have this effect.

Non-compliance with levothyroxine may also give a similar picture, but there is no firm evidence

Question 84 of 138

A 52-year-old farmer was brought into the Emergency Department by his wife. Over the last few hours, he developed severe diarrhoea, passing 10 loose stools in the last four hours. He was also passing urine more frequently and on a couple of occasions was incontinent of urine. He also complained of feeling unwell with a headache and nausea and as well as a cough with white phlegm, and at times felt very short of breath particularly upon coughing. He denied the presence of chest pain. He had a past medical history of COPD, atrial fibrillation, depression and hypertension and his drug history comprised of Seretide 2puffs BD, salbutamol PRN, digoxin 250mcg OD, warfarin 2mg OD, sertraline 150mg OD, diazepam 2mg BD PRN and amlodipine 5mg OD.

Examination revealed the presence of an unwell and unkempt gentleman. His heart rate was 46 bpm and regular, his blood pressure was 88/48mmHG, his respiratory rate was 18, oxygen saturations 96% on air and temperature 35.7ºC. His BM was 3.8 mmol/l. Examination of his cardiovascular system revealed the presence of warm well perfused peripheries with normal heart sounds and a JVP of 3cm. Examination of his respiratory system revealed the presence of copious upper airways secretions but the absence of respiratory distress. Examination of his gastrointestinal system revealed the presence of excess salivation but was otherwise unremarkable. Examination of his neurological system revealed the presence of bilateral pupil constriction but otherwise no abnormalities and a GCS of 15.

He was promptly transferred to the resuscitation area and cannulated with 2 large bore cannulae. Stat intravenous saline 3 litres were promptly infused and an urgent medical consult was requested. Initial investigations revealed the following:

Hb	140 g/l
Platelets	198 * 10 /l9
WBC	6.6 * 10 /l9
CRP	4 mg/l

Coagulation screen: INR 2.2, APTT and fibrinogen within normal limits Portable chest x-ray: poor quality rotated film. Normal appearance of heart and lung fields.

ECG: sinus bradycardia heart rate 45bpm, normal sinus rhythm, normal QRS and QTc intervals, no acute ST/T changes

Arterial blood gases on air:

pH	7.33
Pa02	12.6 KPa
PaCO2	5.8 kPa
HCO3	18 mmol/l
BE	-4

Of the following options which is the most appropriate curative management step?

A. Commence digoxin fragment binding antibody (Digibind)
B. Commence broad spectrum intravenous antibiotics and instigate sepsis protocol
C. Commence intravenous noradrenaline
D. Commence intravenous flumazenil
E. Commence intravenous atropine

E.Commence intravenous atropine

Organophosphate insecticide – atropine

This gentleman has presented with organophosphate toxicity, as manifest by excess salivation, defecation, urinary frequency and copious respiratory secretions. His blood pressure was low and he was bradycardic. He had a history of depression and was prescribed a large dose of sertraline, and statistically was therefore at greater risk of deliberate self-harm (though it may also have been accidentally ingested). Of the following options, atropine is the only antidote to organophosphate and is, therefore, the most appropriate answer (note the question asked for the option most likely to lead to a cure, rather than the next best step, excluding option 3).

Of the remaining options, his symptoms do not correlate well with digoxin toxicity and his ECG has no changes other than bradycardia making this a less likely option. Even if he had ingested diazepam in excess there is no indication to commence flumazenil, and there is little evidence to suggest that he Is suffering from sepsis.

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A 70-year-old man is admitted to hospital with shortness of breath and a productive cough. Following initial investigation a community-acquired pneumonia is diagnosed and treatment with IV fluids and IV antibiotics initiated promptly. Subcutaneous enoxaparin for venous thromboprophylaxis was initiated on the day of admission. Due to persistent hypotension and oliguria secondary due to presumed septic shock, the patient was admitted to the General Intensive Care Unit for inotropic support around 12 hours after initial presentation.

The patients past medical history featured a previous ST elevation myocardial infarction, treated with primary percutaneous coronary interventions one year previously. Regular medications included Aspirin, Clopidogrel, Ramipril, Bisoprolol and Atorvastatin. The patient had no known drug allergies and lived independently in a house with his wife.

With the above management, the patients condition stabilised and he was discharged to a respiratory ward on day 3 of the admission. Routine blood tests taken on day 7 of admission demonstrated a marked fall in platelet count compared to admission bloods. Full examination of the patient at this time did not demonstrate any evidence of venous thrombosis with no skin changes. Following advice from haematology further investigations were requested as detailed below.

Blood results	Day 0 (admission)	Day 3	Day 7
Platelets (x 10>3 / microlitre)	189	156	87
International normalised ratio	1.1	1.3	1.2

Heparin induced thrombocytopenia antibodies: positive (moderate titre)

What further investigation is required (if any) to confirm a diagnosis of heparin induced thrombocytopenia?

A. Serotonin release assay
B. Repeat heparin induced thrombocytopenia antibodies
C. Venous doppler ultrasound of lower limbs
D. Fibrinogen break-down products
E. No further investigation required

A. Serotonin release assay

The diagnosis of heparin induced thrombocytopenia is based on the correlation of the clinical presentation and laboratory results. It is helpful to calculate a pre-test probability score using a tool such as the Warkentin probability scale to assist with interpretation of HIT antibody results. In this case the patient has an intermediate clinical probability of HIT based on the fall in platelet count > 50 % of baseline between day 5-10 of initiating heparin therapy. However, the presence of an alternative cause of thrombocytopenia (severe sepsis) reduces the probability of HIT.

The combination of moderate clinical probability and moderate HIT antibody titre indicate the need for additional testing. Functional assays such as the serotonin release assay have a high sensitivity and specificity for HIT (> 95 %) and so will confirm or exclude a diagnosis of HIT.

Given the clinical picture, it would be prudent in this case to hold further heparin anticoagulants and introduce a non-heparin anticoagulant such as Bivalirudin to reduce the risk of HIT related thrombosis while further testing is being conducted.

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A 62-year-old man is seen in the Emergency Department with a rash which has developed over the last 24 hours. He has had increasing malaise throughout the day. His past medical history includes hypertension and gout. He was started on allopurinol 3 weeks ago due to increasing frequency of his gout attacks. He also takes ramipril and amlodipine.

On examination he has an erythematous maculopapular rash over his whole body, worst on his trunk. He is tachycardic at 105 beats per minute with a blood pressure of 110/55 mmHg and a temperature of 38.1 ºC. He has some residual pain in his right great toe but no other focal signs or symptoms.

His blood tests are as follows:

Hb	130 g/l	Na +	141 mmol/l
Platelets	550 * 10 /l 9	K +	4.7 mmol/l
WBC	12.5 * 10 /l 9	Urea	6 mmol/l
Neuts	6 * 10 /l 9	Creatinine	95 µmol/l
Lymphs	5 * 10 /l 9	CRP	65 mg/l
Eosin	1.5 * 10 /l9		

What is the most likely diagnosis?

A. Drug reaction with eosinophilia and systemic symptoms (DRESS) syndrome
B. Stephens-Johnson syndrome
C. Anaphylactoid reaction
D. Toxic epidermal necrolysis
E. Reactive arthritis

A. Drug reaction with eosinophilia and systemic symptoms (DRESS) syndrome

The presence of maculopapular rash, fever and raised lymphocytes and eosinophils points to a likely diagnosis of DRESS (drug reaction with eosinophilia and systemic symptoms) syndrome. This is a rare complication following treatment with chemotherapy agents, allopurinol or antiepileptics.

Stevens-Johnson syndrome and toxic epidermal necrolysis are part of a spectrum of skin reaction and can also occur with allopurinol. However typically patients present with a target-like rash and progressive blistering and eventual skin detachment. Eyes and other mucosal surfaces are often involved. Anaphylactoid reactions are immediately occurring and present with angioedema, bronchospasm and hypotension. Reactive arthritis presents with a triad of arthritis, urethritis and eye involvement.

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A 38-year-old man presents to the emergency department following the ingestion of a staggered paracetamol overdose, after an argument with his partner. He appears well and N-acetylcysteine is administered.

Twenty-four hours later, he deteriorates clinically and appears confused. Further blood tests are taken, the results are pending, and the medical team is discussing the option of a liver transplant.

What single feature would justify this?

A. Arterial pH <7.3
B. Creatinine >300 µmol/l
C. Grade III or IV encephalopathy
D. Prothrombin time >100 seconds
E. Staggered overdose requiring N-acetylcysteine

A….Arterial pH <7.3

Liver transplantation criteria in paracetamol overdose: pH < 7.3 more than 24 hours after ingestion

The correct answer is an **arterial pH <7.3**. The King's College Hospital criteria define two categories of patients with poor prognosis following paracetamol overdose. One cohort is those in whom arterial pH is less than 7.3, over 24 hours after ingestion. The other cohort is patients who have all three of the following: creatinine >300 µmol/l, prothrombin time >100 seconds and grade III/IV encephalopathy. Therefore, of the options above, the only option which, alone, would warrant consideration of transplantation is arterial pH <7.3.

Creatinine >300 µmol/l alone is therefore incorrect. This forms part of three elements, as described above, which must all be present.

For the same reason, **grade III or IV encephalopathy** alone is also therefore incorrect. It must be present alongside elevated creatinine and prothrombin time.

Prolonged prothrombin time is also incorrect for the reasons described above.

Whether the overdose was **staggered and/or required N-acetylcysteine** infusion does not form part of the criteria.

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A 19 year old man is brought into the Accident & Emergency department by his parents who are very concerned about his well being, and a medical opinion is sought. He arrived home from an evening with friends and was behaving bizzarely. When you talk to him, he tells you that he is feeling on top of the world and that he is an angel sent from god. He also describes seeing music as colours coming out of the radio earlier in the evening. He describes colours being more vivid and vibrant but denies any hallucinations. He has been unable to sleep for the past 24 hours

On examination, he is confused and disorientated but alert, skin colour is normal, central capillary refill time less than 2 seconds with moist mucous membranes. Normal eye movements, no peripheral focal neurology HR 115 sinus tachycardia (ECG normal) Blood tests (essentially normal) RR 16. Pupils dilated but responsive to light equally BP 174/98. When you are testing his eye movements he describes seeing trailing colours and when he closes his eyes he can see afterimages that persist for a few seconds. He repeatedly asks you for glasses of water during the consultation and drinks them quite rapidly.

His mother says that he has no past history of medical problems and that his mental state has been normal until today and that he was well before he left the house 6 hours ago this evening. There is a family history of depression and he has recently left university due to not achieving the required grades

Towards the end of the consultation, he suddenly becomes very agitated and fearful, pointing at a wall believing that he is being chased by a monster, his family are unable to comfort him or lessen his agitation.

What is the the most appropriate treatment to give at this time?

A. Haloperidol 5mg IM
B. Lorazepam 1mg IM
C. Lorazepam 1mg PO
D. Olanzapine 10mg PO
E. Lorazepam IV 1mg and cyproheptadine.

C. Lorazepam 1mg PO

This man is suffering from LSD intoxication. He is describing the symptom of synaesthesia where sensations are perceived in a different modality, in this case seeing music He is also describing palinopsia which is where an after image persists for a few seconds after looking away from the original stimulus. This can happen in a clear sensorium if on focuses on an image for long enough but then the colours will be inverted. In palinopsia there is no inversion of colours. Palinopsia and synaesthesia can occur in temporal lobe epilepsy and other organic conditions, but is quite typical of hallucinogenic usage, particularly LSD. LSD intoxication can also cause expansive mood and grandiosity as seen here and this may rise to delusional intensity. Illusions (distortions of normal perception) and hallucinations in any modality are relatively common as is agitation and anxiety.

The answer is C. In the fist instance reassurance should be used and the patient should be placed in a calm environment with familiar people around. If this fails then benzodiazepenes should be administered. They should be tried orally first as the rate of action PO is similar to IM and injection may cause unecceasry distress in an agitated patient. The question tells you that he has drank water during the consultation, so it is reasonable to try PO first, if this fails then IM would be the next line.

Options A and D could be used as second line therapy if benzodizaepenes fail and also can be used in the treatment of mania. Mania is not the diagnosis here as there is a short history of onset, palinopsia and synaesethesia are much less common and you should not see dilated pupils.

Option E would be a treatment for severe seritonergic syndrome. LSD intoxication can cause serotinergic syndrome, especially if taken in conjunction with other drugs such as MDMA, SSRIs or TCAS. However, there is no evidence of this in our individual.

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A 66-year-old woman is reviewed in the rheumatology clinic for ongoing rheumatoid arthritis, which has been difficult to control. Several joints are currently flaring up and her current agent is not effective in controlling her symptoms.

After some consideration, she is switched to another agent. Blood tests are measured before she starts:

eGFR	>90 ml/min/1.73 m^2	(>90)
Creatinine	56 µmol/L	(55 - 120)

Several weeks after commencing the new drug, her blood tests are measured again:

eGFR	27 ml/min/1.73 m^2	(>90)
Creatinine	198 µmol/L	(55 - 120)

What is she most likely to have started?

A. Ciclosporin
B. Etanercept
C. Infliximab
D. Leflunomide
E. Sulfasalazine

A. Ciclosporin

Ciclosporin may cause nephrotoxicity

Of the options above, **ciclosporin** is best known to cause nephrotoxicity. It is sometimes used as a disease-modifying agent for rheumatoid arthritis. As well as being nephrotoxic, it is hepatotoxic, causes fluid retention, hyperkalaemia, hypertrichosis, gingival hyperplasia and other side effects.

Etanercept is a newer, biologic drug that interferes with tumour necrosis factor-alpha. It is not

widely known to be nephrotoxic. There are some case reports but overall ciclosporin is a much more likely cause.

Infliximab is another biologic used in the management of rheumatoid arthritis and also interferes with tumour necrosis factor-alpha. Like etanercept, it is not particularly known for nephrotoxicity, unlike cyclosporin.

Leflunomide has a similar side effect profile to methotrexate, affecting a wide number of systems, but is not strongly associated with nephrotoxicity.

Sulfasalazine is an aminosalicylate, sometimes used in the management of rheumatoid arthritis. It is not strongly linked to nephrotoxicity.

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A 19-year-old male is found collapsed in a dark alley. He is of no known abode. He is very drowsy but oriented to person, place and time with a Glasgow coma scale of 14/15 (E3, V5, M6). He smells of alcohol but denies having consumed any. His clinical examination is significant for impairment in visual acuity bilaterally. He has a tremor of his outstretched hands but the rest of his examination is normal. His blood pressure is 145/76mmHg with a pulse of 101/min regular, respiratory rate of 22/min and an oxygen saturation of 95% on room air.

Investigations:

Hb	11.3 g/dl
MCV	102 fl
Platelets	110 * 10 /l9
WBC	12 * 10 /l9
Creatinine	117 umol/L
Urea	9 umol/L
Na+	146 mmol/L
K+	5.4 mmol/L
CL-	109 mmol/L

Arterial blood gas:

pH	7.21
PaO2	10.9 kPa
PaCO2	4.2 kPa
HCO3	16 mEq/L

Considering the ingested toxin, what is the next best step in your management?

A. Fomepizole
B. N-acetyl cysteine
C. Activated charcoal
D. Gastric lavage
E. Sodium bicarbonate

A…Fomepizole

Answer: Fomepizole or 4-methylpyrazole.

This patient has ingested methanol. Clue to this is the fact that he smells of alcohol but denies having consumed any, though definitive diagnosis is based on a raised serum methanol level. Initial symptoms are similar to those felt in alcohol intoxication including ataxia, disinhibition, headaches, nausea and vomiting. These are followed by drowsiness which may progress to coma. Metabolic acidosis with raised anion gap is common.

He has visual impairment indicating severe methanol toxicity. Visual signs present in methanol toxicity include, photophobia, blurred vision, poor visual acuity and blindness. Fundoscopy could reveal optic disc swelling or pseudoglaucomatous changes.

This patient needs urgent treatment with i.v fomepizole. If this is not available then i.v ethanol is an adequate alternative though it will produce symptoms similar to alcohol intoxication.

Fomepizole acts by inhibiting the primary step in the conversion of methanol to formic acid, a highly toxic metabolite. Specifically it competitively inhibits alcohol dehydrogenase, the enzyme that converts methanol to formaldehyde which is then further oxidised to formic acid by formaldehyde dehydrogenase.

The signs of methanol toxicity are caused my formic acid so inhibition of its synthesis is a very important step in its management.

Dialysis may be needed to help rapid elimination of the toxin from the system. The following are the indications for haemodialysis in methanol intoxication:

- worsening acidosis despite sodium bicarbonate treatment
- visual involvement
- quantity of consumed methanol exceeding 30ml
- serum methanol level greater than 20mg/dl

Other conditions that may be present in methanol toxicity include:

- Seizures
- cardiac arrhythmias and heart failure
- haemorrhagic pancreatitis
- parkinsonism secondary to haemorrhagic and non haemorrhagic damage to the basal ganglia

He is acidotic and whilst sodium bicarbonate could help with acidosis it is unlikely to lead to overall improvement.

N-acetyl cysteine is used in paracetamol overdose.

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A 49-year-old woman comes to the Emergency department suffering from nausea and lethargy which has increased over the past few days. She has undergone a renal transplant for end stage renal failure due to chronic reflux nephropathy some 3 months ago. You understand her GP prescribed an antibiotic for a respiratory tract infection without checking for potential interactions with her ciclosporin based immunosuppressive regime. Creatinine has increased significantly, and ciclosporin is above the upper limit of the recommended range.

Investigations:

Na +	142 mmol/l
K +	5.1 mmol/l
Urea	8.2 mmol/l
Creatinine (3 months ago)	161 µmol/l
Creatinine (today)	225 µmol/l

Which of the following antibiotics is she most likely to have been prescribed?

A. Amoxicillin
B. Cephalexin
C. Clarithromycin
D. Doxycycline
E. Levofloxacin

C. Clarithromycin

The key here is to recognise that ciclosporin is metabolised by CYP3A4, and clarithromycin and erythromycin are both potent CYP3A4 inhibitors, leading to a potential elevation in ciclosporin levels, and subsequent nephrotoxicity. Because of the danger of either CYP3A4 inhibition or activation in patients taking ciclosporin, patients are advised to consult their transplant team before starting any new medication.

Amoxicillin is predominantly excreted unchanged in the urine, as is cephalexin. Levofloxacin undergoes very limited biotransformation, with 85% excreted in the urine. Doxycycline is

concentrated in bile, not metabolised via the P450 system. After 3 days around 40% is excreted in the urine and approximately 30% in faeces.

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A 48-year-old woman is brought in by ambulance to the emergency department, having been found at home by her neighbour with a decreased level of consciousness. She has a past medical history of open-angle glaucoma and is on regular acetazolamide.

On examination, she is obtunded. Her observations are heart rate of 111 beats per minute, blood pressure 93/65mmHg, respiratory rate of 22/min, temperature 36.7ºC, oxygen saturations of 94% on room air. Cardiovascular, respiratory and abdominal examinations are otherwise unremarkable. Her Glasgow coma scale is 10/15 (E2, V3, M5).

IV fluids are administered and arterial blood gas and blood tests are obtained. Arterial blood gas results:

pH	7.24	(7.35-7.45)
paO2	10.2 kPa	(10-14)
paCO2	5.5 kPa	(4.5 - 6.0
Base excess	-4.2 mmol/L	(-2 - +2)
Chloride	95 mmol/L	(95-105)
Bicarbonate	14 mmol/L	(22-26)
Glucose	5.0 mmol/L	(4-11)
Lactate	2.2 mmol/L	(0.5-2.0)
SaO2	95 %	(94-98)

Blood test results:

Hb	137 g/L	Male: (135-180)Female: (115 - 160)
Platelets	189 * 10 /L 9	(150 - 400)
WBC	4.4 * 10 /L 9	(4.0 - 11.0)
Na +	140 mmol/L	(135 - 145)
K +	5.0 mmol/L	(3.5 - 5.0)
Urea	8.0 mmol/L	(2.0 - 7.0)

Creatinine	111 µmol/L	(55 - 120)
CRP	8 mg/L	(< 5)
Measured osmolality	320 mOsm/Kg	(280-290)

What is the most likely diagnosis?

A. Acetazolamide administration
B. Addisonian crisis
C. Ethylene glycol administration
D. IV sodium chloride 0.9% administration
E. Type 1 renal tubular acidosis

C. Ethylene glycol administration

Metabolic acidosis with a high anion gap and high osmolar gap is seen in ethylene glycol poisoning

Ethylene glycol administration is the correct answer. This is a case of a raised anion gap metabolic acidosis with a raised osmolar gap. The anion gap is calculated as (sodium + potassium) - (chloride + bicarbonate). In this case, it is 36 mmol/L, which is raised (normal range 8-16 mmol/L). The osmolar gap is calculated as osmolality (measured) - osmolarity (calculated). Osmolarity (calculated) is [2*sodium + glucose + urea]. In this case, it is (320) - (293) = 27mOsm (normal range < 10), indicating a raised osmolar gap. A high osmolar gap means the presence of an unmeasured solute in high amounts e.g. ethylene glycol in this case.

Acetazolamide administration is incorrect. This causes a normal anion gap metabolic acidosis.

Addisonian crisis is incorrect. This is a cause of a normal anion gap metabolic acidosis. The anion gap is raised in this case.

IV sodium chloride 0.9% administration is incorrect. This will cause a hyperchloremic acidosis with a normal anion gap.

Type 1 renal tubular acidosis is incorrect. This is a cause of a normal anion gap metabolic acidosis.

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A 62-year-old gentleman presents to the emergency department with central chest pain. He describes his pain as a heavy pressure in the middle of his chest which radiates to his left arm. It has improved with the use of his GTN spray, but he did not want to use more than two puffs as he is concerned about getting a headache with its us. He has a past medical history of angina, previous myocardial infarction three years ago and he is a current smoker with a 25 pack-year history.

His ECG demonstrates ST depression in V3 to V5 of 2mm, pathological Q waves and T wave inversion. His troponin is raised at 350ng/L. He is given aspirin, clopidogrel and morphine. His pain settles and he starts to feel comfortable. He has a high GRACE (Global Registry of Acute Coronary Events) score at 122, meaning that he has a 7.9% chance of death within 6 months. Following discussion with the cardiology registrar will be scheduled for an angiogram within 12 hours.

How should his acute coronary syndrome be further managed?

A. Fondaparinux
B. No further anticoagulants
C. Unfractionated heparin
D. Heparin sodium
E. Warfarin

C. Unfractionated heparin

The correct answer is unfractionated heparin. This is a patient who has presented with chest pain and diagnosed with an NSTEMI and deemed to be at sufficiently at high risk to warrant an urgent angiography. NICE recommends that patients should be treated with aspirin and clopidogrel immediately and this should be combined with fondaparinux unless at high risk of bleeding or angiography is planned within 24 hours. If angiography is planned, then unfractionated heparin should be used. This is because angiography has a risk of haematoma formation and this risk is increased when fondaparinux is used compared to unfractionated heparin as the latter has a much shorter half-life.

Question 94 of 138

A 43-year-old man attends the emergency department complaining of visual blurring, headache and nausea. He has a past medical history of alcohol dependency. He is not on any regular medications. He smokes 20 cigarettes daily and drinks at least one bottle of vodka daily. He is homeless and occasionally sources alcohol from other members of the homeless community.

His observations are as follows: heart rate 111 beats per minute, respiratory rate 28/minute, oxygen saturations 97% on room air, blood pressure 123/77 mmHg and temperature 37.1ºC.

He appears intoxicated. The cardio-respiratory examination is normal. His abdomen is mildly tender but soft and bowel sounds are present. Neurological examination is difficult but there are appears to be some blurring of his vision. His pupils are dilated. There is no epithelial defect upon staining of the eye.

Venous blood gas:

pH	7.31	(7.35-7.45)
HCO3	15 mmol/L	(22-26)
lactate	2.8 mmol/L	(0-2)
Potassium	4 mmol/L	(3.5-5.5)
Sodium	138 mmol/L	(135-145)
Cl-	96 mEq/L	(98-106

Based on the likely diagnosis, what is the most appropriate medication regimen?

A. Folinic acid
B. IV acetazolamide
C. Prednisolone
D. Thrombolysis
E. Topical antibiotics

A. Folinic acid

Folinic acid can be used in methanol poisoning to reduce ophthalmological complications

Folinic acid is the correct answer. This patient is likely to have methanol poisoning, based on the history of nausea, tachypnoea, visual disturbance, elicit alcohol use and raised anion gap (31) metabolic acidosis. Folinic acid can be used as adjunctive treatment in methanol poisoning to reduce the risk of ophthalmological complications. Anchor treatments for methanol poisoning include ethanol/fomepizole, sodium bicarbonate +/- haemodialysis if necessary.

Prednisolone is incorrect. This can reduce the risk of visual complications in giant cell arteritis but this man is too young to have a diagnosis of giant cell arteritis, despite his headache.

IV acetazolamide is incorrect. This can be used to reduce the risk of visual complications in acute angle-closure glaucoma. However, it typically causes a unilateral red eye, which is not mentioned here.

Topical antibiotics is the wrong answer. The absence of an epithelial defect in the eye makes a corneal ulcer and infection unlikely.

Thrombolysis is incorrect. The clinical history suggests methanol poisoning rather than a stroke as a cause of visual loss.

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A 48-year-old female presented with general fatigue, and nausea. She denies having pyrexia, vomiting diarrhoea or any pains. She appeared dehydrated but otherwise, observations and physical examination were within normal limits.

Blood results are as follows:

Na +	130 mmol/l
K +	4.8 mmol/l
Urea	18 mmol/l
Creatinine	162 µmol/l
Lithium	1.6 mmol/l (0.4-1.0 mmol/l)

Looking back at past results from 3 weeks ago, her renal function was in the normal range.

She has a history of bipolar disorder, diet-controlled diabetes and hypertension. She has been compliant with her lithium tablets and undergoing regular checks for the levels.

The patient revealed that her General Practitioner had recently started her on a new tablet 2 weeks ago.

Which of the following would most likely be the precipitant for her symptoms?

A. Indapamide
B. Amlodipine
C. Dihydrocodeine
D. Spironolactone
E. Propranolol

A. Indapamide

Lithium toxicity can be precipitated by thiazides

Acute renal impairment and lithium toxicity can be precipitated by any medicines that can impair renal function or induce hyponatraemia:

- Angiotensin-converting enzyme (ACE) inhibitors
- Diuretics (particularly thiazides including bendroflumethiazide and indapamide)
- Non-steroidal anti-inflammatory drugs (NSAIDs)

Target concentrations of lithium:

- acute episode (mania, hypomania, depression) 0.6-1.0 mmol/L (elderly 0.4-0.8 mmol/L).
- prophylaxis of bipolar affective disorder 0.4-0.8 mmol/L.
- toxic range usually >1.5 mmol/L; however, may begin at >1.0 mmol/L (levels >2 mmol/L need urgent treatment).

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An elderly lady brings her 32-year-old lodger into the emergency department after she witnesses him having two generalised seizures, lasting for around 4 minutes before spontaneously terminating, followed by a loss of consciousness lasting for around 10 minutes before slowly becoming rousable again. He is not known to have a history of epilepsy and does not take any regular medications. He denies any history of illicit drugs, smoking or excessive alcohol intake, corroborated by his landlady, who reports not to have seen him ever drinking in the 4 years he has been living in her loft space. Apart from a mild headache, he claims to have been well recently, continuing to work on his start-up computer software in the loft. She states that he has always 'kept himself to himself' in a cluttered dark loft space without a window, with a simple mattress and a small wardrobe. He didn't even ask for an additional heater during the cold winter as he thought 'the loft gas boiler provides enough warmth'. On examination, he is slightly sleepy but rousable. His Glasgow Coma Score is E4 V5 M6. Apart from erythematous lips, you note no other remarkable features. Neurological examination of his cranial nerves, upper and lower limbs are unremarkable. There is no ophthalmoscope available. His observations are heart rate 90/min and regular, BP 132/88 mmHg, sats 98% on air.

His blood tests demonstrate:

Hb	159 g/l
Platelets	282 * 10 /l9
WBC	7.9 * 10 /l9

Na +	143 mmol/l
K +	4.2 mmol/l
Urea	6.1 mmol/l
Creatinine	88 µmol/l
CRP	3 mg/l

His arterial blood gas demonstrate:

pH 7.25	
PaO2	15.8 kPa
PaCO2	2.9 kPa
Lactate	4.5 mmol/l

HbCO	24%

A CT head demonstrates a small area of acute ischaemic change in the right globus pallidus.

Which is the most appropriate next management?

A. High flow oxygen
B. Aspirin 300mg
C. Clopidogrel 300mg
D. PO chlordiazepoxide 40mg QDS
E. Start levetiracetam 250mg BD

A. High flow oxygen

The young patient presents with new seizures of on a background of a mild headache, red lips and a significantly raised COHb. This is a history suggestive of carbon monoxide poisoning. The ischaemic change on CT head, particularly in the globus pallidus with haemorrhagic transformation, a classic neurological consequence of carbon monoxide poisoning. It should be noted that pulse oximetry and PaO2 are commonly normal in CO poisoning. Carbon monoxide reduces haemoglobin binding capacity and not the amount of O2 dissolved in blood. A metabolic acidosis is present in the blood gas secondary to lactic acidosis from systemic ischaemia. If fundoscopy could have been performed, a cherry-red spot in the macula is the classic finding.

The initial management of CO poisoning is high flow oxygen, which reduces the half-life of CO binding to haemoglobin from 300 to 90 minutes. It is recommended that patients with severe acidosis (pH<7.1), COHb >25%, continued loss of consciousness and severe end organ failure be transferred to a unit for hyperbaric oxygen therapy.

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A 65-year-old man presents to the emergency department with dysuria and abdominal pain. He has a past medical history of chronic kidney disease stage IV. His medications include atorvastatin, amlodipine and ramipril.

On examination, there is mild suprapubic tenderness. Urinalysis:

Nitrites	positive
Leucocytes	+++
Blood	+
Protein	+
Glucose	negative

Blood tests:

Hb	137 g/L	Male: (135-180)Female: (115 - 160)
Platelets	189 * 10 /L 9	(150 - 400)
WBC	8.2 * 10 /L 9	(4.0 - 11.0)
Na +	137 mmol/L	(135 - 145)
K +	4.2 mmol/L	(3.5 - 5.0)
Urea	22.1 mmol/L	(2.0 - 7.0)
Creatinine	278 µmol/L	(55 - 120)
EGFR	23 mL/min/1.73m2	(90-120)
CRP	48 mg/L	(< 5)

Given the likely diagnosis, which antibiotic would be unsuitable?

A. Amoxicillin
B. Cephalexin
C. Ciprofloxacin
D. Co-amoxiclav
E. Nitrofurantoin

E. Nitrofurantoin

Nitrofurantoin is best avoided in patients with CKD stage 3 or higher due to the significant risk of treatment failure and occurrence of side effects due to drug accumulation

Nitrofurantoin is correct. The patient has a urinary tract infection. Nitrofurantoin is a reasonable choice in some circumstances. However, this antibiotic should be avoided in patients with CKD stage 3 or higher due to the significant risk of treatment failure and occurrence of side effects due to drug accumulation.

Amoxicillin is incorrect. This may be a suitable choice for a urinary tract infection in some circumstances. Again, a dose reduction could be considered in severe renal impairment but it is not contraindicated.

Cephalexin is not the right answer. This is a potential antibiotic choice for a urinary tract infection. The BNF states the dose adjustment for renal impairment is a max dose of 1.5 g daily if eGFR 10- 40 mL/minute/1.73 m2.

Ciprofloxacin is not the right answer. This would be a suitable choice for a urinary tract infection in some circumstances. The BNF states the dose adjustment in renal impairment is to give 250-500 mg every 12 hours if eGFR 30-60 mL/minute/1.73 m2 or every 24 hours if eGFR is less than 30 mL/minute/1.73 m2.

Co-amoxiclav is not the right answer. This is a possible antibiotic choice for a urinary tract infection. The BNF states the dose adjustment in renal failure if eGFR 10-30 mL/minute/1.73 m2 is one 250/125 strength tablet every 12 hours or one 500/125 strength tablet every 12 hours.

Question 98 of 138

A 29-year-old male from Eastern Europe presents to the Emergency Department with 48 hours of severe 'burning and sharp' electrical pains in his arms and legs. He is well known to the department, having been treated for alcohol withdrawal a number of times in the past and is a known intravenous drug user. He reports no weakness, dysarthria or hallucinations. He reports drinking about 7 pints of beer in the past 24 hours, which he says is normal for him, and has used intravenous heroin daily for the past 2 weeks.

On examination, both upper and lower limbs are rigid and mildly bradykinetic. A bilateral resting tremor is noted in both hands. Reflexes and sensation are normal. Examination of cranial nerves and eye movements are unremarkable.

His initial blood tests return and are as follows:

Hb	163 g/l
Platelets	276 * 10 /l9
WBC	5.5 * 10 /l9
Vitamin B12	202 ng/l
Folate	471 (>317 nmol/l)

Na +	142 mmol/l
K +	4.3 mmol/l
Urea	6.4 mmol/l
Creatinine	85 µmol/l
CRP	7mg/l
HIV	negative

Which investigation is most likely to yield the underlying diagnosis?

A. DAT scan
B. Anti-GM1b antibodies
C. Borrelia burgdorferi serology
D. Urine porphybilinogen
E. Serum heavy metals

E. Serum heavy metals

The patient has presented with extrapyramidal signs in the context of a HIV-negative intravenous drug user with alcohol excess. The differential causes for Parkinsonism should be considered: idiopathic, genetic, trauma, vascular etc.

Urinary porphobilinogen is a diagnostic test for acute intermittent porphyria (AIP), typically presenting with significant abdominal pain, urinary symptoms and a peripheral neuropathy. AntiGM1b are antibodies typically present in multifocal motor neuropathy with conduction block, an immune disorder affecting motor peripheral nerves alone. Heavy metal poisoning must be considered in a young patient with Parkinsonism, in addition to the MPTP toxins found in classic cases of intravenous drug users in 1980s California. This patient is vulnerable to metals such as manganese, which is often found in 'dirty' preparation of intravenous heroin from Eastern Europe and produces extrapyramidal symptoms similar to idiopathic Parkinson's disease. Similar symptoms should be suspected in heavy industry workers.

Question 99 of 138

A 24-year-old woman presents to the emergency department following a paracetamol overdose. She advises you that she took 20 x 500mg tablets 26 hours ago. Her body weight is 62kg. On examination there is no evidence of decompensated liver failure, she is not confused, and her abdomen is soft and non-tender.

Blood results are as follows:

Hb	122 g/L	Male: (135-180)Female: (115 - 160)
Platelets	164 * 10 /L 9	(150 - 400)
WBC	8.2 * 10 /L 9	(4.0 - 11.0)
Na +	138 mmol/L	(135 - 145)
K +	4.6 mmol/L	(3.5 - 5.0)
Urea	4.2 mmol/L	(2.0 - 7.0)
Creatinine	72 µmol/L	(55 - 120)

Bilirubin	14 µmol/L	(3 - 17)
ALP	66 u/L	(30 - 100)
ALT	28 u/L	(3 - 40)
Î³GT	52 u/L	(8 - 60)
Albumin	46 g/L	(35 - 50)

INR	6.2	(0.9-1.1)
Prothrombin time	82 secs	(12-16 secs)
Activated partial thromboplastin time (APTT)	28 secs	(25-35 secs)
Fibrinogen	4.5 g/L	(2 - 4)

Paracetamol level at 26 hours	Awaited

How will you manage this patient?

A. Activated charcoal
B. Await paracetamol level results
C. Discuss with the liver transplant unit
D. Gastric lavage
E. Start N-acetylcysteine

E. Start N-acetylcysteine

Paracetamol overdose: if presentation > 24 hours after an overdose start acetylcysteine if the patient is jaundiced, has hepatic tenderness or an elevated ALT

Start N-acetylcysteine is correct. Acetylcysteine should be started immediately in all patients if it is thought that 150 mg/kg or more paracetamol (or an unknown amount) has been ingested as an acute overdose (i.e. all doses taken within one hour) or if they are symptomatic with jaundice or hepatic tenderness. The patient in this case has ingested 10,000 mg / 62 kg = 161 mg/kg. She should therefore start N-acetylcysteine immediately. If the patient is asymptomatic and has ingested less than 150 mg/kg, then TOXBASE recommends waiting for blood results before considering treatment with N-acetylcysteine.

Start or continue treatment with acetylcysteine if:

- ALT is above the upper limit of normal, or
- INR is greater than 1.3 (in the absence of another cause, e.g. warfarin), or
- Paracetamol concentration is detectable

Activated charcoal is incorrect. Activated charcoal can be considered if the paracetamol has been ingested within one hour of presentation.

Await paracetamol level results is incorrect. Since the patient has ingested > 150 mg/kg of paracetamol she should be started on acetylcysteine immediately. Blood tests including paracetamol level are then monitored serially to determine the ongoing need for N-acetylcysteine treatment.

Discuss with the liver transplant unit is incorrect. There are no indications for liver transplant as per the King's college hospital criteria which includes:

- Arterial pH < 7.3 (24 hours after ingestion)
- All of the following: prothrombin time > 100 seconds, creatinine > 300 µmol/l, and grade III or IV encephalopathy

Gastric lavage is incorrect. Gastric lavage is not recommended due to concerns over perforation.

This was a previously widely favoured method that has now been all but abandoned due to a lack of evidence of efficacy and the risk of complications.

Question 100 of 138

A 44-year-old man presents to the emergency department complaining of breathlessness. He reports that he was leaving a restaurant because he was starting to feel lightheaded, developed a headache and was breathless.

An ABG done on admission demonstrated carbon monoxide at 26%.

Three hours later he feels tired and weak, but his headache and lightheadedness have improved since admission. He has a past medical history of asthma which he reports to be well controlled, rarely needing his blue inhaler. He smokes 15-ciagarettes per day and has a 23 pack-year history. He works as a teacher in central London. On examination, he is receiving 15L of oxygen via a nonrebreather mask. His chest is clear on auscultation. Neurological examination is normal. An ECG demonstrates no ischaemic changes.

Observations:

Saturations	100%
Respiratory rate	16/min
Blood pressure	126/87mmHg
Heart rate	84/min
Temperature	36.8°C

What is the most appropriate management plan?

A. Wean off oxygen guided by standard pulse oximetry saturation levels

B. Prescribe IV mannitol

C. Maintain high-flow oxygen until asymptomatic or carbon monoxide levels are <10%

D. Refer urgently for hyperbaric oxygen treatment

E. Arrange urgently for non-invasive ventilation

C. Maintain high-flow oxygen until asymptomatic or carbon monoxide levels are <10%

The answer is to maintain high-flow oxygen until asymptomatic or carbon monoxide levels are <10%. Weaning off oxygen is not appropriate to be guided by pulse oximetry as standard pulse oximetry will detect carbon monoxide bound haemoglobin as well and are therefore falsely reassuring. IV mannitol is indicated if cerebral oedema is anticipated, such as in severe cases, or established. Hyperbaric oxygen again also reserved for severe cases or pregnancy, and noninvasive ventilation has no role in carbon monoxide poisoning.

Question 101 of 138

A 23-year-old woman is admitted to the emergency department with agitation and she refuses to answer any questions. Her partner is with her and reports she has become increasingly agitated throughout the day. He states she has no past medical history but is on some tablets for her mood which her GP has recently changed. You find that she is taking sertraline and mirtazapine, but the patient and her partner are unable to tell you dosages.

The patient allows you to examine her briefly. No abnormality is found on cardio-respiratory and abdominal examination. You note her pupils are dilated, her reflexes are brisk and myoclonus is present. Her observations are shown below:

Temperature	38.8ºC
Heart rate	110/min
Blood pressure	178/102mmHg
Respiratory rate	25/min
Oxygen saturation	98% on air

Blood tests are in progress. You prescribe some intravenous fluids and lorazepam to calm the patient.

Given the diagnosis, what other treatment should the patient receive?

A. Dantrolene

B. Cyproheptadine

C. Bromocriptine

D. Co-amoxiclav

E. Procyclidine

B. Cyproheptadine

Treatment of severe serotonin syndrome- cyproheptadine (5-HT2 receptor antagonist)

The patient is presenting with serotonin syndrome. The clues in the history that lead to the diagnosis are the fact that she is on 2 serotonergic agents, she has autonomic excitability (tachycardia, hypertension etc), and neurological features such as hyperreflexia. The differential diagnosis could be neuroleptic malignant syndrome which also presents with autonomic excitability, however, the patient is not taking a neuroleptic and the neurological features would be different (hyporeflexia is expected).

Treatment of serotonin syndrome is usually supportive but in severe cases, cyproheptadine can be used and so option 2 is correct.

Dantrolene is a muscle relaxant that acts via inhibiting calcium release in muscle tissue and is used for the treatment of neuroleptic malignant syndrome.

Bromocriptine is a dopamine agonist, also used in the treatment of neuroleptic malignant syndrome.

Co-amoxiclav is an antibiotic that can be used for numerous infections. Although the patient is febrile, there is no other information from the history or examination to suggest an infection, therefore is not the correct answer.

Procyclidine is an anticholinergic used for acute dystonia.

Question 102 of 138

A 66-year-old gentleman was admitted for an elective aortic valve replacement. His past medical history included a non-ST elevation myocardial infarction three years prior, as well as hypertension and hypercholesterolaemia. He also suffered a deep vein thrombosis 16 years ago after open reduction and internal fixation of a tibial fracture requiring treatment with warfarin postoperatively. There was no family history of note. He was currently taking aspirin 75mg OD, clopidogrel 75mg OD, ramipril 5mg OD, bisoprolol 2.5mg OD, atorvastatin 20mg ON and lansoprazole 15mg OD.

Two days prior to the operation the patient was commenced on an intravenous heparin infusion which was continued perioperatively. The operation was deemed successful and the infusion was discontinued. He was commenced on enoxaparin 40mg OD sc as prophylaxis for venous thromboembolism postoperatively. He was otherwise making a good post operative recovery and was already mobilising readily. He complained of no chest pain or shortness of breath with no syncopal episodes. He resumed a full and normal diet and his bowel habits were normal. There was no evidence of bleed or any other adverse symptoms.

Routine post operative bloods at day 5 were as follows:

Hb	110 g/l
MCV	81 fl
Haematocrit	0.36
Platelets	28* 10 /l9
WBC	9.0 * 10 /l9
PT	36 seconds
APTT	86 seconds
D-dimer	136 ng/mL
Fibrinogen	236 g/L

Na +	142 mmol/l
K +	4.5 mmol/l
Urea	6.3mmol/l

Creatinine	77 µmol/l
Bilirubin	17 µmol/l
ALP	101 u/l
ALT	16 u/l
Albumin	39 g/l

Blood tests at day 2 were as follows:

Hb	116 g/l
Platelets	161 * 10 /l9
WBC	10.0 * 10 /l9

What is the single most appropriate management step?

A. Stop heparin and commence treatment with vitamin K
B. Continue with enoxaparin
C. Stop all anticoagulation until platelet count is >100 x 10^9/l
D. Stop enoxaparin and commence danaparoid
E. Stop enoxaparin and commence warfarin

D. Stop enoxaparin and commence danaparoid

The diagnosis is strongly suggestive of heparin induced thrombocytopaenia type 2, given the treatment with intravenous heparin, the platelet count in the absence of other haematological abnormalities, the absence of clinical signs of haemorrhage and the onset of low platelet count at day 5 post operatively. This patient is at very high risk of an arterial or venous thrombotic event and needs urgent anticoagulation. Warfarin should not be used alone and there is a 10% crossreactivity with LMWH, and so of the options available anticoagulation with danaparoid is the most suitable option.

Question 103 of 138

A 45-year-old man presents to the emergency department complaining of palpitations. He states that these started suddenly 2 hours ago. He has no past medical history and takes no regular medication. An ECG (electrocardiogram) is done and shows atrial flutter with 2:1 block

His observations are the following Sats 97% on air

Heart rate 150 bpm Respiratory rate 22/min Blood pressure 105/78 mmHg Temperature 36.8ºC

Which of the following treatments should be avoided?

A. Electrical cardioversion
B. Atenolol
C. Diltiazem
D. Digoxin
E. Flecainide

E. Flecainide

You risk causing 1:1 conduction if you give flecainide in atrial flutter

Flecainide slows the rate of atrial flutter which can 1:1 conduction and cardiovascular compromise. It can also cause 'organisation' of atrial fibrillation turning it into atrial flutter with 1:1 conduction.

Class IA and class IC antiarrhythmic drugs should not be given to treat atrial flutter unless patients are first pre-treated with a drug that blocks AV (atrioventricular) node conduction such as a beta blocker or calcium channel blocker.

All of the other options can be used to treat atrial flutter

- Cardioversion should be used first line
- In patients who are not suitable for cardioversion beta blockers and calcium channel blockers can be used to slow AV node conduction
- Digoxin used to be first line treatment for ventricular rate control in flutter. Now it is used mostly in patients with heart failure alongside a beta blocker. It is also used in patients whose rate has not been adequately controlled by a calcium channel blocker, a beta blocker or both.

Management of Atrial flutter

- Is similar to that of atrial fibrillation although medication may be less effective
- Atrial flutter is more sensitive to cardioversion however so lower energy levels may be used
- Radiofrequency ablation of the tricuspid valve isthmus is curative for most patients

Question 104 of 138

A 31-year-old man is brought to the Emergency Department from a nightclub. He is accompanied by a friend who tells you he has taken the street drug NRG-1. The patient is agitated and difficult to assess as he will not stay on the trolley. He is sweating profusely. Formal assessment is impossible but he is obviously confused and appears to be hallucinating. Examination shows a heart rate of 139bpm with a blood pressure of 158/105mmHg. Heart sounds are rapid but with no murmurs. The chest is clear to auscultation and oxygen saturations are 99% on air but his respiratory rate is 36/min. He is globally hypertonic with marked clonus in the lower limbs. Tympanic temperature is 40.6°C.

Blood results show:

Haemoglobin	129g/L	Sodium	119mmol/L	Creatine Kinase 1895ng/mL
Haematocrit	0.29	Potassium	5.5mmol/L	CRP 8.9mg/L
White cells	15.6x10^9/L	Chloride	90mmol/L	
Neutrophils	11.7x10^9/L	Urea	7.2mmol/L	
Platelets	303x10^9/L	Creatinine	100µmol/L	

Urine is dilute but contains blood, protein and ketones on dipstick analysis

Which one of the following interventions is the most appropriate in the first instance?

A. Rapid intravenous infusion of 0.9% sodium chloride with 1g IV paracetamol
B. Rapid intravenous infusion of 3% sodium chloride with 1g IV paracetamol
C. Slow intravenous infusion of 3% sodium chloride with 10mg IV midazolam
D. Slow intravenous infusion of 3% sodium chloride with 1g IV paracetamol and oralcyproheptadine 12mg
E. Intravenous methylene blue (methylthioninium chloride) 1mg/kg over 5 minutes

C. Slow intravenous infusion of 3% sodium chloride with 10mg IV midazolam

NRG-1, a synthetic cathinone, can cause agitation, hyponatraemia and serotonin syndrome. Treatment is with benzodiazepines, cooling and hypertonic saline if hyponatraemic. Patients may require intubation and paralysis to control hyperpyrexia

Hyperpyrexia is a consequence of muscle activity, not hypothalamic dysregulation so paracetamol is not useful. Indeed, if multiorgan failure develops, paracetamol is not conjugated and excreted safely, thus paracetamol toxicity may worsen the picture. Intravenous benzodiazepines are the mainstay in reducing agitation and helping to control muscle activity and this should be commenced in the short term. Given the concurrent hyponatraemia and elevated CK slow fluids with hypertonic saline is advised.

External cooling manoeuvres such as ice baths may be tried but when hyperpyrexia rises above 40°C it is safer to anaesthetise and paralyse patients and this patient is likely to warrant this.

Cyproheptadine is a specific 5HT-2A receptor antagonist and may be used in moderate serotonin syndrome. In this case, it is unlikely the patient will be cooperative enough to take it and its effects will be too slow. This option also includes paracetamol making this an incorrect answer.

Methylene blue is an antidote for methaemoglobinaemia; it would be unusual for NRG-1 to cause this although users of the drug often display a blue tinge to their skin for unknown reasons. The methaemoglobin level is not given here so use of methylene blue is not advisable.

Question 105 of 138

A 26 year old male is admitted to hospital with difficulty in breathing, wheeze and a cough productive of green sputum. He is assessed and determined to have a community acquired lower respiratory tract infection with biochemical and radiological evidence lending weight to this diagnosis. His medical history is significant for capricious asthma and he has been admitted to intensive care previously and ventilated. Currently he is taking salbutamol and formoterol/budesonide inhalers at their maximum doses, montelukast, aminophylline and omalizumab (Xolair) monthly subcutaneous injections.

Which of the following antibiotics is best avoided in this patients treatment?

A. Amoxicillin
B. Azithromycin
C. Ciprofloxacin
D. Doxycycline
E. Gentamicin

C. Ciprofloxacin

Co-administration of aminophylline and ciprofloxacin can cause significant toxicity and should be avoided

This question is about drugs which interact via their effect on hepatic cytochrome metabolising enzymes. Pharmacokinetically speaking, any drug has the potential to affect how another is processed, metabolised or excreted but only some have a relevant clinical effect.

In the above vignette we are concerned about a newly commenced antibiotic affecting one of the patients regular medications and causing a deleterious effect. Salbutamol and formoterol are beta agonist drugs which have their own side effects of adrenergic updrive but they rarely have any clinically relevant interactions. Similarly, the inhaled steroid budesonide is seldom affected by other co-administered drugs to a vast degree. Omalizumab is a monoclonal antibody to unbound human IgE and is used as an adjunct treatment in atopic asthma in steroid resistant patients who display extreme histamine sensitivity and elevated IgE levels. It has few clinical interactions. Aminophylline derivatives are moderately active bronchodilators which work two-fold; by inhibiting phosphodiesterases and hence ultimately dampening inflammatory cytokines, and also by acting as an antagonist at adenosine receptors in the heart, brain and lung. It is metabolised extensively by the liver, predominantly by the enzyme 1A2 and has a narrow therapeutic window with elimination pathways becoming saturated even at therapeutic doses. Any drug which inhibits cytochrome enzyme 1A2 can rapidly lead to aminophylline toxicity.

Ciprofloxacin in particular, is a strong inhibitor of enzyme 1A2 causing a decrease in function of at least 80% and an average rise in serum concentrations of metabolising substrate of five-fold. Coadministration of ciprofloxacin with aminophylline is extremely likely to cause toxicity.

The use of macrolide antibiotics with aminophylline is contentious. Most macrolides actually induce activity of 1A2 leading to a reduction in aminophylline levels (similar to the effect of long term smoking). Both aminophylline and macrolide antibiotics however tend to predispose to prolongation of myocardial repolarisation time and there is a risk of ventricular arrhythmia. Azithromycin only poses a theoretical risk rather than an observed one and dose reduction and drug monitoring of aminophylline levels is appropriate if the two drugs must be used together. Erythromycin is best avoided.

Doxycycline use with aminophylline can cause theoretical fluctuations in serum aminophylline levels but regular monitoring of drug levels is sufficient to avoid toxicity.

Neither amoxicillin nor gentamicin have clinically relevant drug interactions with aminophylline.

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A 27-year-old patient has been asked to attend the emergency department 2 weeks post colectomy and stoma formation due to the following blood results:

Hb	110 g/L	Male: (135-180)Female: (115 - 160)
Platelets	412 * 10 /L 9	(150 - 400)
WBC	10 * 10 /L 9	(4.0 - 11.0)
Na^+	147 mmol/L	(135 - 145)
K^+	4.9 mmol/L	(3.5 - 5.0)
Urea	16.9 mmol/L	(2.0 - 7.0)
Creatinine	244 µmol/L	(55 - 120)
CRP	65 mg/L	(< 5)

She has been struggling to manage her stoma due to high output of between 1-2litres of liquid stool per day. She has been using rehydration salts and loperamide at home.

Abdominal x-ray: unremarkable appearances Stool culture: negative

What further treatment would be most appropriate to reduce her high-output diarrhoea?

A. Bismuth subsalicylate
B. IV fluids
C. Morphine sulphate
D. Octreotide
E. Vancomycin

D. Octreotide

Octreotide may reduce high output diarrhoea following ileostomy or colectomy. It can be given by continuous subcutaneous infusion

Octreotide is correct. This can be given via a continuous subcutaneous infusion in the management of high-output stoma diarrhoea.

Bismuth subsalicylate is incorrect. It is usually used for symptoms of dyspepsia and is occasionally used in the management of travellers' diarrhoea. There is no evidence to support its use in the reduction of diarrhoea as a result of a high-output stoma.

Morphine sulphate is incorrect. Although morphine and other opiate-class medications can cause constipation as a side effect, they are not used routinely in the management of high-output stomas.

IV fluids is incorrect. Using IV fluids to manage acute kidney injury would be entirely appropriate, however, it would not have any effect on the ongoing diarrhoea itself.

Vancomycin is incorrect, this would be the treatment for *C. difficile* diarrhoea, however, in this case, stool samples were unremarkable.

Question 107 of 138

A 28-year-old woman attends the emergency department with reduced consciousness. She has a past medical history of depression which is treated with sertraline. She also has chronic pain (for which she takes tramadol, gabapentin, ibuprofen, and a lidocaine patch) and epilepsy (which is managed with phenytoin).

On examination, her Glasgow Coma Score is 11 (M4, V4, E3). You also note mydriasis, hyperreflexia, and clonus.

What drug is most likely implicated?

A. Gabapentin
B. Ibuprofen
C. Lidocaine
D. Phenytoin
E. Tramadol

E. Tramadol

Tramadol co-prescribed with SSRIs is a common cause of serotonin syndrome

The patient has clinical features of serotonin syndrome.

Tramadol is correct. Tramadol co-prescribed with SSRIs is a common cause of serotonin syndrome.

Gabapentin is incorrect. The use of SSRIs with gabapentin can increase the risk of hyponatraemia. **Lidocaine** is incorrect. There are no interactions with SSRIs and lidocaine.

Phenytoin is incorrect. The use of sertraline with phenytoin increases the risk of phenytoin toxicity. Phenytoin toxicity manifests predominantly as nausea, and central nervous system dysfunction (particularly confusion, nystagmus, and ataxia), with depressed conscious state, coma, and seizures occurring in more severe cases. Cardiac complications such as arrhythmias and hypotension can also occur. The clinical features, in this case, are more in keeping with serotonin syndrome.

Ibuprofen is incorrect. The use of these two agents increases bleeding risk. It is important to remember that SSRIs have an anti-platelet effect since serotonin is an important transmitter involved in platelet function.

Question 108 of 138

A 27-year-old man is brought to the Emergency Department following a suspected deliberate overdose. On arrival, he is drowsy with a GCS of 13/15 (E3V4M6) and confused. His airway is currently patent. The respiratory rate is 30 breaths per minute although oxygen saturations are 98% on 2L/min oxygen. The chest is clear to auscultation. Heart rate is 108bpm and blood pressure is 96/48mmHg. The ECG shows sinus tachycardia only. He complains of severe abdominal pain and is groaning on examination. There is significant tenderness in the right upper quadrant. Bowel sounds are hyperactive. Neurological examination is difficult but discloses downgoing plantars and reactive, mid-size pupils.

Blood tests show:

Haemoglobin	112g/L	Sodium	131mmol/L	Glucose	17.8mmol
MCV	96fL	Potassium	Haemolysed	Lactate	5.3mmol/L
White cells	15.7x109/L	Urea	15.6mmol/L	pH	7.14
Neutrophils	13.7x109/L	Creatinine	207mol/L	pCO2	2.9kPa
Platelets	278x109/L	ALP	234U/L	pO2	19.2kPa
INR	1.2	ALT	2453U/L	Bicarbonate	9.4mmol/L
PT	14secs	AST	3109U/L	Base excess	-15.4mEq/L
APTT	49.2secs	Bilirubin	36mol/L	Anion gap	30.8

The laboratory comments there is significant haemolysis of the samples. He has a massive vomit whilst being assessed, of black liquid and fresh blood.

Which of the following drugs is this patient most likely to have overdosed upon?

A. Aspirin
B. Ferrous sulphate
C. Ibuprofen
D. Paracetamol
E. Venlafaxine

B. Ferrous sulphate

Severe iron toxicity presents with liver failure, gastrointestinal caustic damage and coagulopathy with raised APTT. Early hyperglycaemia and extensively haemolysed samples may also indicate significant iron burden

Iron is a surprisingly toxic drug. When taken in overdose symptoms may be expected to be seen at ingestions of 20mg/kg. 200mg/kg can be expected to be fatal. Symptoms of direct corrosion on the gastrointestinal tract may be seen within six hours of ingestion with abdominal pain, vomiting, diarrhoea and GI bleeding. Stool and vomitus may be black or grey unless frank bleeding predominates. During this interval, a hallmark of iron toxicity is a raised blood glucose; only a few drugs may cause this in overdose. Hypoglycaemia occurs later in presentation due to liver failure. Other early features may include a neutrophil leucocytosis and significant haemolysis of samples due to the high plasma iron burden. Arterial blood gases will show a significant metabolic acidosis with a high anion gap. Lactate may be elevated but not sufficiently to explain the anion gap. Coagulopathy develops rapidly but the APTT tends to be prolonged in iron toxicity compared to the prolongation of prothrombin time in a paracetamol poisoning. In the ensuing hours, iron deposition within the myocardium, brain and liver can be expected to cause rapid cardiac failure, encephalopathy and hepatocellular necrosis with liver failure respectively. Renal failure develops due to hypovolaemia and acute tubular necrosis.

Unless treated, death may result from haemorrhagic volume loss or from hepatic failure, although in significant overdose, early death results from respiratory failure due to coma. Treatment for iron overdose is with decontamination of the bowel, including whole bowel irrigation if needed and chelation therapy with desferrioxamine.

Although paracetamol toxicity can be expected to cause fulminant hepatic failure with renal failure and coagulopathy, the INR would be expected to be significantly deranged if this was the culpable drug given the extent of other organ damage. Also hypoglycaemia would be expected due to liver failure.

Aspirin, when taken in significant overdose, causes a characteristic constellation of symptoms including metabolic acidosis (although alkalosis may be seen at lower dose intoxications), convulsions, tinnitus, hyperpyrexia and pulmonary oedema. Often ventricular arrhythmia occurs due to metabolic acidosis and intracellular anaerobic respiration. Rarely coagulopathy develops but usually in association with disseminated intravascular coagulation which is not the case here. Aspirin overdoses may present with hyperglycaemia however and along with iron and aminophylline, these agents should be considered when unexplained hyperglycaemia is seen.

Ibuprofen and other non-steroidals are of low toxicity unless taken in massive overdose. Gastrointestinal upset and occasional GI haemorrhaging may be seen but liver failure and coagulopathy is rare. In massive overdose, central nervous depression with cerebellar signs and global electrolyte imbalances may be seen with blood dyscrasias. Multi-organ failure may develop but individual organ dysfunction is less likely.

Venlafaxine, a noradrenaline and serotonin modulator, may cause serotonin syndrome and sympathomimetic symptoms, including arrhythmia, hyperpyrexia and respiratory failure. Hepatitis is rare.

Question 109 of 138

A 21-year-old woman presents to the emergency department after her flatmate found her drowsy in her bedroom. She had been incontinent of urine.

On examination the patient is obtunded with a Glasgow Coma Score of 7 (E1V2M4). The pupils are 7mm bilaterally and sluggishly reactive to light. The heart rate is 133bpm and the blood pressure is 99/65mmHg.

A 12-lead ECG reveals sinus tachycardia with a QT interval of 510ms. The QRS duration is 115ms.

What is the most likely cause for this patient's presentation?

A. Amyl nitrate toxicity
B. Gamma hydroxybutyric acid toxicity
C. Toluene solvent toxicity
D. Tricyclic antidepressant toxicity
E. Heroin toxicity

D. Tricyclic antidepressant toxicity

Dilated pupils in combination with ECG changes including prolonged QT interval and QRS duration (borderline prolonged in this case) should suggest tricyclic antidepressant toxicity.

Features of toluene toxicity include irritation to the eyes, nose and respiratory tract from inhalation, along with confusion, ataxia, headache, slurred speech and euphoria. Prolongation of the QT interval is not usually associated.

Features of amyl nitrate toxicity include blurred vision, xanthopsia and haemoptysis. It does not typically affect the corrected QT interval.

Features of gamma hydroxybutyric acid (GHB) toxicity include CNS and respiratory depression, hypersalivation, bradycardia and hypotension. It does not typically affect the corrected QT interval.

Heroin toxicity typically results in depression of the respiratory and central nervous systems and pin-point pupils (rather than mydriasis in this case). It does not typically affect the corrected QT interval.

Question 110 of 138

A 48-year-old farmer is found in a state of distress having collapsed whilst mixing insecticides in his barn. On arrival in the Emergency department, he is confused, salivating excessively, and has been incontinent of urine and faeces. You note noisy breathing, his blood pressure is 90/60 mmHg, with a pulse of 48 beats per minute.

Which of the following is the most appropriate intervention?

A. Atropine
B. Hyoscine
C. Glucagon
D. NG activated charcoal
E. Noradrenaline

A,. Atropine

Organophosphate insecticide – atropine

The clue here is insecticide exposure, coupled with evidence of muscarinic effects of organophosphate poisoning including excessive secretions, incontinence, hypotension and bradycardia. Time is of the essence, and administration of atropine in this situation may be lifesaving.

Pralidoxime is also used once atropine has been administered, although it's additive benefit is debated.

Hyoscine is less potent versus atropine and is mainly used in palliative care to manage pain from the gastrointestinal tract and excessive respiratory secretions. Glucagon is used as a treatment for beta-blocker overdose. Activated charcoal may be of value but is subsidiary to use of anticholinergics. Anticholinergics are also preferred to sympathetic agonists such as noradrenaline.

Question 111 of 138

A 33 year female of Arabic descent with bullous pemphigus is admitted electively for intravenous immunoglobulins. She has no other past medical history, no recent travel history and has lived in rural Norfolk her whole life. On day 4 of a 5 day planned course of treatment, she develops pyrexia, nausea and vomiting. On examination, you note mild neck stiffness and photophobia. She also complains of a posterior headache. Her serum markers are as below:

WBC	14.0 * 10 /l9
Neuts	11.0 * 10 /l9

Na +	133 mmol/l
K +	3.8 mmol/l
Urea	4.7 mmol/l
Creatinine	80 µmol/l
CRP	4 mg/l

A CT head is unremarkable. The immunoglobulin is stopped. You perform a lumbar puncture: WCC 10/mm3, protein 0.4g/L, no organisms, opening pressure 17.8 cm H2O. Blood cultures taken during a temperature spike grow no organisms after 48 hours.

What is the diagnosis?

A. Aseptic meningitis
B. Bullous pemphigus flare
C. TB reactivation, resulting in TB meningitis
D. Viral meningitis
E. Bacterial meningitis

A. Aseptic meningitis

Aseptic meningitis is a classic complication of IVIg treatment. This patients inflammatory markers are unremarkable, as is the rest of her septic screen. There is little to suggest worsening of underlying bullous pemphigus. Aseptic meninigits occurs in 1% of patients with intravenous immunoglobulin. The key is to rule out any infective causes and to stop the immunoglobulin treatment until this is been done. Modern preparations can be stored in a fridge for months: there

Question 112 of 138

A 17-year-old girl presents to the emergency department with her mother. She is drowsy, ataxic, vomiting, her heart rate is 130 beats per minute and her blood pressure 130/84 mmHg. Her only past medical history is epilepsy which has recently been well controlled. After arguing with her boyfriend 40 minutes ago she took an unknown quantity of sodium valproate.

Following initial fluid resuscitation which of the following would you implement?

A. Activated charcoal
B. Observe for 3 hours and discharge
C. Fomepizole
D. Loperamide
E. Transfer to psychiatric bed

A. Activated charcoal

As the question states that she has taken the overdose less than an hour previously she would be suitable for activated charcoal.

Of the other options, discharging her while severely symptomatic, or transfer to a psychiatric ward while there are acute medical issues would be inappropriate.

Fomepizole is used in methanol or ethylene glycol poisoning.

The question does not give any indication for loperamide at present.

Question 113 of 138

You are called to review a 48-year-old woman in the Surgical Ward. She was admitted directly to the ward five hours ago post-operation for an elective open right hemicolectomy for adenocarcinoma of the caecum. The operation was completed without any concern. For postoperative pain relief, the patient has a local anaesthetic infusion to rectus sheath catheters and an oxycodone PCA. No significant previous medical history. No known drug allergies.

The patient had been reviewed by the nursing team half an hour before for postoperative antibiotics and replacement intravenous fluids. Her observations were all within normal parameters, however, the patient complained of new tinnitus.

She has just had a self-terminating seizure and remains post-ictal with a GCS of 10. Her observations are:

- Heart rate 45 bpm
- Blood pressure 78/35 mmHg
- Respiratory rate 25/min
- Temperature 36.5ºC
- Blood glucose 4.4mmol/l

What treatment should be started immediately?

A. Intravenous levetiracetam loading dose
B. Intravenous fluid and vasopressor
C. Intravenous lipid emulsion
D. Major haemorrhage transfusion
E. Intravenous glucose

C. Intravenous lipid emulsion

Local anaesthetic toxicity can present with CNS (e.g. reduced GCS, seizures) and cardiovascular (e.g. tachyarrhythmias, hypotension, bradyarrhythmias) features. Treatment of severe toxicity is with IV lipid emulsion.

Local anaesthetic toxicity is correct. The patient has a local anaesthetic infusion in place. This may be delivering local anaesthetic directly into the bloodstream or the local anaesthetic infusion may have inadvertently been connected to the intravenous access port. This is severe toxicity with seizures, bradycardia, hypotension and low GCS. Tinnitus, perioral numbness, metallic taste and auditory changes can be earlier symptoms. In this case of severe toxicity, lipid emulsion should be administered. This acts as a binding agent for the circulating local anaesthetic molecules.

Intravenous levetiracetam loading dose is incorrect. The patient's seizure is secondary to local anaesthetic toxicity and not epilepsy. Although further seizure control may be required the underlying cause should be treated immediately to prevent further seizures, cardiovascular collapse and death.

Intravenous fluid and vasopressor are incorrect because the patient's deteriorating clinical condition does not fit with sepsis. They have had an operation without complication, are already on prophylactic antibiotics and their clinical signs do not make sepsis the most likely diagnosis. IV fluids and vasopressors may be required in the course of their treatment for local anaesthetic toxicity but they do not treat the underlying cause of the deterioration.

Major haemorrhage transfusion is incorrect. Although postoperative haemorrhage should always be considered there is a more likely diagnosis in this case. Although the operation progressed well there is a risk of postoperative bleeding. If the patient is displaying symptoms of haemorrhage and cardiovascular shock then the surgical team and anaesthetic team must be notified immediately to facilitate an urgent return to theatre. Activation of the major haemorrhage protocol may be appropriate depending on the clinical picture.

Intravenous glucose is incorrect because the clinical scenario points towards local anaesthetic toxicity. Although it is important to ensure that glucose is correct to be within normal range, it is more important to start the lipid emulsion immediately. This will prevent further seizures, cardiovascular collapse and death.

Question 114 of 138

A 57-year-old man is bought to ED by ambulance after being found on the street unwell.

On examination, he is maintaining his own airway but is tachypnoeic with a respiratory rate of 28 breaths/minute. He complains of a headache and blurred vision and has poor coordination on examination.

After a short period in the department, he deteriorates further and is drowsy with a GCS of 11. An arterial blood gas (ABG) shows:

pH	7.2	(7.35-7.45)
pO2	16.4 kPa	(10.0-13.0 kPa)
pCO2	2.3 kPa	(4.50-6.00 kPa)
HCO3-	14 mmol/L	(22.0-28.0 mmol/L)
BM	6.4 mmol/L	(3.9-8.0 mmol/L)
Na+	143 mmol/L	(135-145 mmol/L)
Cl-	98 mmol/L	(98-107 mmol/L)

What is the most appropriate next step in management for this patient?

A. Activated charcoal
B. Ethanol
C. Fomepizole
D. Haemodialysis
E. Naloxone

C. Fomepizole

Methanol poisoning is preferentially treated with IV fomepizole; this should be commenced immediately the suspicion is raised

This patient presents with symptoms of methanol poisoning (tachypnoea, headache, blurred vision, impaired coordination and reduced consciousness). The significant metabolic acidosis with raised anion gap should increase your suspicion. The immediate management of methanol poisoning is with the antidote **fomepizole**.

Activated charcoal has no effect to bind methanol and therefore plays no role in the use of methanol poisoning.

Ethanol can be used in methanol poisoning as it competitively binds to alcohol dehydrogenase and blocks methanol binding. However, it is no longer used first-line in the UK since the introduction of fomepizole.

Haemodialysis may be considered for this patient but fomepizole would be the next step in management while this was arranged.

Naloxone is used in opioid overdose but has no role in methanol poisoning.

Question 115 of 138

A 65-year-old female presents to respiratory outpatients with an 8-week history of gradually worsening shortness of breath. Her exercise tolerance has reduced to 50 yards from several miles; she reports chronic lower back pain but no other symptoms. Her past medical history includes type 2 diabetes mellitus, psoriasis, recurrent urinary tract infections and ischaemic heart disease. She currently takes metformin 500 mg BD, bisoprolol 2.5 mg OD, aspirin 75 mg OD, atorvastatin 40 mg ON, ramipril 2.5 mg and nitrofurantoin 50 mg ON with no change in the last 6 months. She has never smoked, has no pets and works as a secretary. On examination, the patient is breathless on minimal exertion. Observations are normal, there is no clubbing and she has fine endinspiratory crepitations at both bases. A CT chest shows ground glass changes with minimal honey combing at both bases.

What is the most likely cause of her underlying respiratory condition?

A. Asbestosis
B. Psoriasis
C. Metformin
D. Ankylosing spondylitis
E. Nitrofurantoin

E. Nitrofurantoin

This patient has a history and examination consistent with pulmonary fibrosis. Her CT chest supports this demonstrating ground glass and honey combing at both bases. Pulmonary function tests will demonstrate a restrictive pattern and arterial blood gas analysis will show a type 1 respiratory failure. Long-term use of nitrofurantoin is known to cause a basal pattern of pulmonary fibrosis and is the most likely culprit in this case. There is no suggestion of asbestos exposure in the history and both psoriasis and ankylosing spondylitis classically cause an apical pattern of fibrosis. Lower back pain is a common medical complaint in this age group and ankylosing spondylitis tends to present in younger males.

Question 116 of 138

A 64-year-old woman comes to the Emergency department complaining of palpitations at rest, and worsening angina over the past month. She has been treated with amiodarone for the past 3 years for recurrent ventricular tachycardia. She has a blood pressure of 110/70 mmHg, pulse of 95 beats per minute, and a fine tremor. There is no goitre. TSH is suppressed at less than 0.05 U/ml.

You suspect she has amiodarone induced thyrotoxicosis.

How best can you determine the underlying pathophysiology?

A. IL6 level
B. Thyroglobulin level
C. TSH level
D. Duration of amiodarone therapy
E. Colour flow doppler ultrasonography

E. Colour flow doppler ultrasonography

Type 1 amiodarone induced thyrotoxicosis is caused by increased production of thyroid hormone, most likely as a result of the excess iodine load administered to the patient as a result of amiodarone treatment. Type 2 amiodarone induced thyrotoxicosis is caused by a destructive thyroiditis. Colour flow doppler ultrasonography needs to be performed by an experienced operator, but is thought to distinguish between the two causes of amiodarone induced thyrotoxicosis around 80% of the time. In patients where the diagnosis is uncertain, radioiodine uptake, (which is low in type 2 disease) my further help differentiating between the two.

Type 2 disease is generally seen later in the time course of amiodarone therapy, although this isn't an invariable differentiating factor, and data on the usefulness of IL6 and thyroglobulin is conflicting.

Question 117 of 138

An 82-year-old man presents with pain following a fall. He was brought in by ambulance after he slipped and hurt himself on the pavement. He ended up tripping whilst returning from the local shops. He remembers his fall and noted immediate pain in his right leg, and was unable to get up. A passerby called an ambulance and he was brought into the hospital. He has a past medical history of ischaemic heart disease, mild dementia, hypertension and high cholesterol.

Pelvic X-rays demonstrated an intertrochanteric right hip fracture. He is due to be operated on within the five hours and is put nil by mouth and started on IV fluids.

How should his risk of venous thromboembolism (VTE) be managed?

A. Low molecular weight heparin before surgery
B. Unfractionated heparin before surgery
C. Delay VTE prophylaxis until following surgery
D. Mechanical VTE prophylaxis before surgery
E. IV heparin infusion until surgery

D. Mechanical VTE prophylaxis before surgery

The correct answer is mechanical VTE prophylaxis before surgery. This is a patient with a hip fracture due to be operated on within 12 hours. He should have careful mechanical VTE prophylaxis with stockings or pneumatic compression despite having a fracture. If surgery was to occur in over 12 hours then low molecular weight heparin should be started, with the last dose given 12 hours before surgery. If he also had renal failure, then unfractionated heparin should be used and also stopped 12 hours before surgery.

Question 118 of 138

A 56-year-old man is brought into hospital with palpitations, restlessness and agitation. His wife tells you he has lost 2 kilograms of weight over the past month. She attributes this to persistent diarrhoea over the same period.

He has a past medical history of persistent atrial fibrillation and takes amiodarone.

On examination, he has a heart rate of 144 beats per minute and his ECG confirms atrial fibrillation with no ischaemic changes. You are unable to palpate any abnormality of the thyroid cartilage.

Investigations are as follows:

TSH	0.1 mU/L
Tc scan 99	reduced uptake

Which of the following is the most important step in this patient's immediate management?

A. Carbimazole
B. Corticosteroids
C. Propylthiouracil
D. Thyroidectomy
E. Increase dose of amiodarone

B. Corticosteroids

In this case, amiodarone is likely the underlying cause of this man's fast AF. Indeed he has a number of symptoms suggestive of hyperthyroidism.

The mainstay of treatment, in this case, would be to give steroids. The absence of a goitre points towards type 2 AIT. The reduced uptake on radioisotope imaging reflects the destructive changes to the thyroid seen in this type of AIT.

Increasing the dose of amiodarone may help with cardioversion but will not treat the underlying cause. Treating with anti-thyroid drugs, such as carbimazole or PTU, may help with thyrotoxicosis but again will not reverse the destructive changes to the thyroid gland. At this stage surgical removal of the thyroid is not indicated.

Question 119 of 138

You are asked to review a 23-year-old patient with a history of ulcerative colitis who was admitted one week ago with a flare unresponsive to steroids. However, over the last three days, he has noticed a visible tremor, and the nurses have noted higher blood sugars on the ward.

On examination, his BP is 154/104mmHg, and his fasting blood glucose is 6.0. His abdomen is soft, with generalised lower abdominal discomfort but no guarding. His chest is clear.

He reports feeling more stressed recently at work and tells you he often uses cannabis in his downtime. The nurses have noticed him leaving the ward often during their shift.

Which medication was he most likely started on?

A. Azathioprine
B. Ciclosporin
C. Infliximab
D. Mesalazine
E. Prednisolone

B. Ciclosporin

Cannabidiol may increase the concentration of ciclosporin

Ciclosporin is the correct answer. In this case, a patient with a severe flare of UC refractory to steroids might be started on ciclosporin. This question assesses knowledge of ciclosporin's side effects and its interaction with cannabis. Ciclosporin may interact with cannabidiol, and adverse effects include tremor, impaired glucose tolerance and susceptibility to infection. Cannabis use can increase ciclosporin levels due to inhibition of cytochrome P450 enzymes by cannabinoids such as CBD.

The other medications listed are used in the treatment of ulcerative colitis but do not exhibit this combination of side effects or interactions with cannabis (as seen on the BNF interaction checker).

Azathioprine commonly interacts with cannabidiol. The levels of azathioprine are increased as cannabidiol decreases its metabolism. Side effects of azathioprine include bone marrow suppression, thrombocytopenia and increased susceptibility to infections. However, the mentioned side effects in the case don't align with azathioprine side effects.

Infliximab is not known to interact with cannabidiol. Side effects include diarrhoea, infusionrelated reactions and increased susceptibility to infections.

Mesalazine is not known to interact with cannabidiol. Side effects include arthralgia, pancreatitis (rarely), skin reactions and diarrhoea.

Prednisolone is not known to interact with cannabidiol. It is unlikely that this patient would be on this medication if the current episode is unresponsive to steroids. Side effects include fluid retention, increased risk of infection, peptic ulcer and hypertension.

Question 120 of 138

A 45 year-old Russian woman complains of a 2 week history of joint pain affecting her wrists, shoulders and knees bilaterally. She also reports calf pain bilaterally. She has been started on first line treatment for pulmonary tuberculosis 2 months previously. Symptoms resolved after the addition of aspirin.

Which drug is most likely causing her symptoms?

A. Rifampicin
B. Isoniazid

C. Pyrazinamide
D. Ethambutol
E. Streptomycin

C. Pyrazinamide

By far the most common cause of arthralgia from the above is pyrazinamide.

Question 121 of 138

A 32-year-old male illicit drug user is caught attempting to sell cocaine on the street by the police. On seeing the officers approach him, he swallows a plastic bag containing around 1.5g of cocaine in an attempt to destroy the evidence. The police officers bring the gentleman into the emergency department. He is asymptomatic and complains of no chest pain. His ECG is unremarkable and his admission blood tests demonstrate no abnormalities, including a negative troponin.

What should you do next?

A. Treat presumptively as acute coronary syndrome
B. Discharge the patient to police custody
C. Await 12 hour troponin and if negative, discharge to police custody
D. Repeat ECG and if no dynamic changes, discharge to police custody
E. Monitor the patient until the ingested cocaine is excreted

A. Monitor the patient until the ingested cocaine is excreted

Cocaine produces significant cardiovascular sequelae, classically coronary vasospasm resulting in cardiac ischaemia, presenting as chest pain and similar ECG changes to acute coronary syndrome. Similar vasospasm can lead to transient cerebral ischaemia, resulting in ischaemic infarcts. In addition, cardiac dysrhythmias are also common, including sinus tachycardia, sinus bradycardia, supraventricular tachyarrhythmias, bundle branch block, ventricular fibrillation and torsades de pointes. In this scenario, it is clear that the ingested cocaine has not penetrated to coverings and released systemically. However, the danger remains until the ingested cocaine has been excreted. Patients like this should be monitored for cardiovascular and cerebral sequelae until the ingested amounts are collected.

Question 122 of 138

You are asked to review a 44-year-old lady in the emergency department. She is well known to the mental health liaison team because of her multiple suicide attempts. She is agitated and slightly confused and unable to give a precise history. She is able to tell you that she can't see clearly and has a dry mouth.

Her sister explains that the patient has been prescribed medicines for chronic neuropathic back pain and has taken them all after splitting up with her boyfriend.

On examination she has symmetrically dilated pupils but no focal motor deficit. Her heart sounds are normal and her lung fields are clear on auscultation. An ECG shows sinus tachycardia with a QRS of 170.

Bloods results are as follows:

Hb	125 g/l
Platelets	440 * 10 /l9
WBC	9.9 * 10 /l9
Na +	135 mmol/l
K +	4.2 mmol/l
Urea	5.1 mmol/l
Creatinine	88 µmol/l
Plasma Paracetamol	<5mmol/l

You request an arterial blood gas which shows the following readings:

pH	7.29
pCO$_2$	3.4 kPa
pO$_2$	14.5 kPa

What is the most appropriate next step in this patient's management?

A. IV sodium bicarbonate
B. Haemodialysis
C. IV lignocaine
D. IV amiodarone
E. Naloxone

A. IV sodium bicarbonate

The culprit here is a tricyclic antidepressant, such as dosulepin or amitriptyline. The first step in treating this patient is to reverse the metabolic acidosis with IV bicarbonate. Dialysis is of no benefit in this scenario.

The dilated pupils and absence of respiratory depression point away from opioid overdose, making naloxone the wrong answer.

Question 123 of 138

A 56 year old gardener is brought to the Emergency Department by his wife. He was bitten on the right hand by a snake while clearing bushes in his back garden. It is identified as a common European adder. There is an obvious, red puncture site on the dorsum of the right hand with significant swelling of the entire hand to the level of the wrist. He is in significant discomfort. Examination reveals a normal cardiovascular examination with a heart rate of 112bpm, blood pressure of 137/90mmHg and normal respiratory rate and oxygen saturations. Capillary glucose is 5.6mmol. ECG shows normal sinus rhythm with normal PR, QRS and QT intervals. Routine blood tests including liver function, electrolytes, creatine kinase, full blood count and arterial blood gases are normal.

Which of the following interventions is the most appropriate at this stage?

A. Application of a tourniquet above the site of envenomation to prevent systemic toxicity
B. Bathe the site of envenomation in hot, sterile water to inactivate the venom
C. Intravenous access and rapidly infuse 0.9% sodium chloride with 1g intravenousparacetamol
D. Intravenous access and administer 200mg IV hydrocortisone, 10mg IV chlorphenamineand 0.5mg IM adrenaline 1:1000
E. Intravenous access and administer 10ml adder antivenin in 500ml 0.9% sodium chlorideover 30 minutes

B. Bathe the site of envenomation in hot, sterile water to inactivate the venom

Adder bites are rare, but when they occur may be extremely painful; the mainstay of treatment is analgesia and supportive therapy. Discuss the use of antivenin with NPIS and do not apply a tourniquet

Application of a tourniquet is absolutely contraindicated since it increases the likelihood of localised necrosis of the affected limb as well as the risk of venous thrombosis which in turn increases the likelihood of central venous thrombosis on release of the tourniquet, as well as cardiac arrhythmia due to the systemic flooding of released potassium and other necrosis factors from tissue damage and haemolysis in a ligatured extremity when the tourniquet is removed.

Unlike venom of the United Kingdom's only other native poisonous animal, the weeverfish (Trachinidae family), viper venom is not heat labile. Application of cold water to a bite may help to assuage pain, but hot water will not deactivate the venom. Weeverfish stings may be treated by bathing the affected limb (usually a foot) in hot water to denature the active proteins.

Adder antivenin is readily available for use in severe bites and can be obtained by contacting local zoos or the National Poisons Information Service. However, severe anaphylactic reactions to the antivenin are common and may be seen in up to 20% of recipients, particularly in atopic or asthmatic individuals. For this reason, antivenin is reserved only for severe systemic envenomations in patients with resistant hypotension, new ECG changes, significant rise in white cell count, raised CK, metabolic acidosis or swelling involving more than half the affected limb or crossing a joint boundary, e.g. beyond the wrist if bitten on the hand. The patient in the above scenario has none of these symptoms. Similarly, in the absence of any symptoms suggestive of an acute anaphylactoid reaction, treatment with adrenaline and hydrocortisone is unnecessary, although antihistamines may help symptomatically. Often, symptomatic treatment is all that is required with analgesia taking priority. Intravenous analgesia is often faster acting and fluids may be required if swelling is significant or vomiting is profuse.

Question 124 of 138

A 25-year-old man attends the emergency department with his partner. He reports having taken an overdose of paracetamol approximately 30 hours ago. Initially, he vomited several times but has not done so for the last 10 hours. He complains of mild vague abdominal discomfort.

On examination, he has some right upper quadrant tenderness on palpation. There is no guarding and there is no obvious jaundice present.

What is the most appropriate management?

A. Send paracetamol levels and await the result to decide on N-acetylcysteine

B. Start N-acetylcysteine after taking paracetamol levels

C. Start N-acetylcysteine and then take paracetamol levels

D. Start N-acetylcysteine, paracetamol levels not required

E. Supportive management

B. Start N-acetylcysteine after taking paracetamol levels

Paracetamol overdose: if presentation > 24 hours after an overdose start acetylcysteine if the patient is jaundiced, has hepatic tenderness or an elevated ALT

For patients who have taken an overdose of paracetamol with presentation later than 24 hours, start treatment before blood tests are available if the patient has visible jaundice or hepatic tenderness or has taken 150mg/kg or more of paracetamol as per Toxbase guidance, and start treatment after blood results if there is a raised ALT or INR, or detectable paracetamol level.

Given the clinical picture, this patient's right upper quadrant tenderness is highly likely to represent hepatic tenderness so he should be commenced on treatment. The best practice is to take the paracetamol levels before starting the infusion. Hence the option to **start Nacetylcysteine after taking paracetamol levels** is correct. This is because if paracetamol levels are taken during N-acetylcysteine infusion, some analysers may significantly underestimate paracetamol levels.

As this patient has hepatic tenderness, it would not be correct to wait for the levels before starting treatment. Hence, the option to **send paracetamol levels and await the result to decide on N-**acetylcysteine is incorrect.

Due to this potential interference with the analyser, the option to **start N-acetylcysteine and then take paracetamol levels** is incorrect.

Taking paracetamol levels is helpful in management and is recommended by Toxbase. Therefore, the option to **start N-acetylcysteine, paracetamol levels not required** is incorrect.

This patient warrants N-acetylcysteine treatment, therefore **supportive management** is incorrect.

Question 125 of 138

A 45-year-old man presents to the emergency department with haematemesis. He states that he drank 'some liquid' in order to end his life. His observations are as follows: heart rate 105 beats per minute, blood pressure 125/75 mmHg, respiratory rate 24 breaths per minute, temperature 37.2ºC, and SpO2 96% on air. On examination, you note nystagmus, ataxia, and ophthalmoplegia.

Blood results are as follows:

Hb	145 g/L	Male: (135-180)Female: (115 - 160)
Platelets	85 * 10 /L 9	(150 - 400)
WBC	20.8 * 10 /L 9	(4.0 - 11.0)
Na +	138 mmol/L	(135 - 145)
K +	3.5 mmol/L	(3.5 - 5.0)
Urea	8.4 mmol/L	(2.0 - 7.0)
Creatinine	110 µmol/L	(55 - 120)
CRP	22 mg/L	(< 5)
Glucose	5.1 mmol/L	(3.9 - 5.6)
Serum osmolarity	310 mOsm/kg	(275 - 295)

What is the treatment of choice?

A. Activated charcoal

B. Ethanol

C. Folinic acid

D. Fomepizole

E. Haemodialysis

D. Fomepizole

Ethylene glycol toxicity management - fomepizole. Also ethanol / haemodialysis

The patient has clinical and biochemical features of ethylene glycol toxicity.

The osmolar gap = osmolality (measured) - osmolarity (calculated). A normal osmolar gap should be < 10.

The calculated osmolarity in this case = (2 x [Na+]) + [glucose] + [urea]) = 289.5 mOsm/kg. Therefore the osmolar gap = 310 - 289.5 = 20.5 mOsm/kg.

The patient thus has a high osmolar gap which indicates the presence of an abnormal solute present in significant amounts.

Causes of a high osmolar gap include:

- Mannitol
- Methanol
- Ethylene glycol

Fomepizole is correct. For patients with ethylene glycol toxicity, early administration of fomepizole or ethanol will minimise the further metabolism of ethylene glycol and the development of clinical and metabolic complications. Fomepizole is the agent of choice if available.

Activated charcoal is incorrect. Activated charcoal is of no use since it does not adsorb significant quantities of ethylene glycol.

Ethanol is incorrect. Fomepizole is the preferred antidote since, unlike ethanol, it does not require regular monitoring of blood concentrations or cause inebriation. Ethanol is an effective antidote but is associated with more complications. If there is any delay in obtaining fomepizole, administer ethanol urgently initially, followed by fomepizole when available.

Folinic acid is incorrect. Folinic acid is used to reduce ophthalmological complications in methanol poisoning.

Haemodialysis is incorrect. The patient is symptomatic and requires urgent treatment. Haemodialysis has a role in severe or refractory cases.

Question 126 of 138

A 20-year-old university student attends the emergency department with his friends who were concerned after they found him urinating on the living room sofa. On examination, he appeared agitated, sweaty, and had occasional muscle twitching.

Observations: heart rate 110 beats per minute, blood pressure 165/105 mmHg, temperature 37.9ºC.

On review of his medical record, you can see he takes citalopram for depression and has also recently been started on another medication.

What other medication is most likely to account for his presentation?

A. Cyclizine
B. Cyproheptadine
C. Methylene blue
D. Phenoxymethylpenicillin
E. Tramadol

E. Tramadol

Tramadol co-prescribed with SSRIs is a common cause of serotonin syndrome

The patient in the stem is likely presenting with serotonin syndrome, as indicated by disorientation, agitation, muscle twitching, hypertension, tachycardia, and pyrexia.

Tramadol is the correct answer. When this is co-prescribed with SSRIs such as citalopram it can precipitate serotonin syndrome. This is because tramadol acts as a central serotonin reuptake inhibitor and so further increases serotonin levels.

Cyclizine is incorrect. There is no evidence to suggest that cyclizine is implicated in the development of serotonin syndrome.

Cyproheptadine is incorrect. Cyproheptadine can be used as a treatment for serotonin syndrome. Other treatments can include intravenous fluids and benzodiazepines.

Methylene blue is incorrect. It is a thiazine dye used to treat methemoglobinemia and is not implicated in the development of serotonin syndrome.

Phenoxymethylpenicillin is incorrect. Other antibiotics such as ciprofloxacin, isoniazid, and linezolid can potentiate serotonin syndrome, however, there is no evidence to suggest that phenoxymethylpenicillin is a cause.

Question 127 of 138

A 30-year-old man attends the emergency department with chest pain. He describes the pain as a squeezing sensation in the centre of his chest. There is no radiation of his pain but he feels mildly breathless. He works as a city lawyer which he admits can be particularly stressful. He is an occasional smoker and binge-drinks alcohol on the weekends. He has no other past medical history.

On examination, he is tremulous and sweating profusely. He has a temperature of 37.9ºC. His heart rate is 140bpm and regular with a blood pressure of 143/87mmHg. His chest is clear with normal heart sounds. Saturations are 96% on air. He has increased rigidity in his upper and lower limbs. His pupils are dilated and reactive to light.

Investigations:

Hb	151 g/L	Male: (135-180)Female: (115 - 160)
Platelets	288 * 10 /L $_9$	(150 - 400)
WBC	9.1 * 10 /L $_9$	(4.0 - 11.0)
Na $_+$	149 mmol/L	(135 - 145)
K $_+$	5.4 mmol/L	(3.5 - 5.0)
Urea	10.3 mmol/L	(2.0 - 7.0)
Creatinine	134 µmol/L	(55 - 120)
D-Dimer	398 ng/L	(<400)
Troponin	67 ng/L	(0-14)

ECG - ST depression in I, aVL, V5 and V6.

He is diagnosed with an NSTEMI and dual antiplatelet therapy, fondaparinux and sublingual glyceryl trinitrate (GTN) is initiated for acute coronary syndrome (ACS). His pain begins to settle after GTN.

What is the most appropriate medication to give in addition to the ACS protocol?

A. Atenolol
B. Sodium nitroprusside
C. Diazepam
D. Intravenous fluids
E. Glyceryl trinitrate infusion

C. Diazepam

Cocaine toxicity - avoid beta-blockers

This man is likely to have taken a cocaine overdose. Symptoms include an increased sympathetic drive causing hyperthermia, sweating, tachycardia and dilated pupils. Chest pain is secondary to vasospasm of the coronary arteries causing ECG changes and raised cardiac enzymes. Benzodiazepines are indicated in cocaine toxicity and in this scenario, ACS treatment should be initiated with the addition of benzodiazepines such as diazepam.

Sodium nitroprusside is an indicated additive in the instance of cocaine toxicity and hypertension.

Glyceryl trinitrate infusions are indicated in ACS with ongoing chest pain. This patient has shown good response to sublingual GTN, making a GTN infusion less appropriate at this stage.

Intravenous fluids are likely to also be prescribed to treat his co-existing acute kidney injury. However, in the instance of cocaine toxicity and chest pain, diazepam is the more appropriate initial choice.

Atenolol and other beta blockers should be avoided or used with caution in cocaine toxicity due to the risk of unopposed alpha-agonism. Benzodiazepines should be given instead, making diazepam the correct answer.

Question 128 of 138

A 35-year-old farmer who is in financial difficulties presents to the Emergency department some 40 minutes after taking an unknown amount of paraquat-based weed killer. On arrival in the Emergency department, he has a Glasgow coma scale score of 15 and tells you he regrets what he has done. He feels sick and says there is a burning sensation in his mouth. On examination his blood pressure is 135/80 mmHg, his pulse is 85 beats per minute and regular. There are no abnormal physical signs.

Which of the following is the most important intervention?

A. Administration of emetic
B. Gastric lavage
C. IV normal saline
D. IV sodium bicarbonate
E. Oral fuller's earth

E. Oral fuller's earth

Both activated charcoal and fuller's earth are recognised to adsorb paraquat in vitro. They are thought to be most effective when given in the first two hours after overdose, although benefit may still be present up to 12hrs after ingestion. Neither has significant toxicity issues.

Gastric lavage is not recommended. Administration of emetic risks spreading caustic material further, potentially driving greater absorption of paraquat. IV normal saline and IV sodium bicarbonate do not affect prognosis in paraquat overdose. No anti-inflammatory, immunosuppressive, or anti-oxidant interventions have demonstrated a benefit either.

Question 129 of 138

A 63-year-old woman is brought in by ambulance to the emergency department, having been found at home by her neighbour with a decreased level of consciousness. She has a past medical history of osteoarthritis and hypertension. She takes ramipril and uses ibuprofen regularly for pain relief.

Her observations are heart rate of 118 beats per minute, blood pressure 95/66mmHg, respiratory rate of 20/min, temperature 36.7ºC and oxygen saturations of 94% on room air.

Cardiovascular, respiratory and abdominal examinations are otherwise unremarkable. Her Glasgow coma scale is 11/15 (E3, V3, M5).

IV fluids are administered and an arterial blood gas and blood tests are obtained.

Arterial blood gas results:

pH	7.24	(7.35-7.45)
paO2	10.2 kPa	(10-14)
paCO2	5.7 kPa	(4.5 - 6.0
Base excess	-4.2 mmol/L	(-2 - +2)
Chloride	95 mmol/L	(95-105)
Bicarbonate	14 mmol/L	(22-26)
Glucose	5.0 mmol/L	(4-11)
Lactate	2.5 mmol/L	(0.5-2.0)
SaO2	94 %	(94-98)

Blood test results:

Hb	137 g/L	Male: (135-180)Female: (115 - 160)
Platelets	189 * 10 /L $_9$	(150 - 400)
WBC	4.4 * 10 /L $_9$	(4.0 - 11.0)
Na $_+$	141 mmol/L	(135 - 145)
K $_+$	5.0 mmol/L	(3.5 - 5.0)
Urea	8.0 mmol/L	(2.0 - 7.0)
Creatinine	111 µmol/L	(55 - 120)
CRP	8 mg/L	(< 5)
Measured osmolality	324 mOsm/Kg	(280-290)

What is the most likely diagnosis?

A. Aspirin overdose
B. Ethylene glycol administration
C. IV sodium chloride 0.9% administration
D. NSAID treatment
E. Ramipril overdose

B. Ethylene glycol administration

Metabolic acidosis with a high anion gap and high osmolar gap is seen in ethylene glycol poisoning

Ethylene glycol administration is the correct answer. This is a case of a raised anion gap metabolic acidosis with a raised osmolar gap. The anion gap is calculated as (sodium + potassium) - (chloride + bicarbonate). In this case, it is 37 mmol/L, which is raised (normal range 8-16 mmol/L). The osmolar gap is calculated as osmolality (measured) - osmolarity (calculated). Osmolarity (calculated) is [2*sodium + glucose + urea]. In this case, it is (324) - (295) = 29mOsm (normal range < 10), indicating a raised osmolar gap. A high osmolar gap means the presence of an unmeasured solute in high amounts e.g. ethylene glycol in this case.

Aspirin overdose is incorrect. This can cause a high anion gap metabolic acidosis with a high osmolar gap. However, If the aspirin overdose was sufficiently large to cause the patient to be obtunded with a low GCS, we would expect a respiratory alkalosis and possibly convulsions. Aspirin has direct effects on the respiratory centre and causes tachypnoea and respiratory alkalosis. In this case, the oxygen saturations are 94%, the pO2 is low normal, the pCO2 is on the upper end of normal and the respiratory rate is merely 20, making this a less likely cause clinically than ethylene glycol.

IV sodium chloride 0.9% administration is incorrect. This causes a normal anion gap metabolic acidosis with high chloride. Additionally, the fluids were administered after she had become unwell, excluding this as a cause of her presentation.

NSAID treatment is incorrect. This can cause a type 1 renal tubular acidosis, which will have a normal anion gap.

Ramipril overdose is incorrect. This is associated with a type 4 renal tubular acidosis, which will have a normal anion gap. Furthermore, if the patient took a significant ramipril overdose, we might expect the blood pressure to be lower.

Question 130 of 138

You are looking after a 76-year-old man on an orthogeriatric ward. He is day 7 post dynamic hip screw for neck of femur fracture. He appears to be doing relatively well, but the nurse has noticed that he has a dark black necrotic looking skin over his left iliac fossa, and has asked you to come and look at it.

His past medical history includes hypertension, mild chronic kidney disease and atrial fibrillation.

He is currently on Ramipril 2.5mg once daily, Bisoprolol 5mg once daily, and treatment dose Clexane (patient was on Warfarin pre-operatively).

Pre-admission operative bloods:

Hb	142 g/l
Platelets	212 * 10 /l9
WBC	14.1 * 10 /l9
Na +	138 mmol/l
K +	4.6 mmol/l
Urea	8.2 mmol/l
Creatinine	167 µmol/l

Bloods yesterday:

Hb	112 g/l
Platelets	27 * 10 /l9
WBC	10.1 * 10 /l9
Na +	136 mmol/l
K +	4.0 mmol/l
Urea	9.1 mmol/l
Creatinine	182 µmol/l

What should you do with regards to his Clexane?

A. Stop Clexane and give Warfarin (target INR 2-3)
B. Stop Clexane and give treatment dose Argatroban
C. Switch treatment dose Clexane to Unfractionated Heparin infusion
D. Give 1 pool of platelets
E. Switch treatment dose Clexane to prophylactic Clexane

B. Stop Clexane and give treatment dose Argatroban

This is a case of heparin induced thrombocytopaenia

Criteria	2	1	0
Thrombocytopaenia	>50% fall, and nadir >20*10 /l9	30-50% fall, or nadir 10-19*10 /l9	fall < 30%, or nadir < 10*10 /l9
Timing of platelet fall	Between 5-10 days, or <1 day if exposure to heparin within last 20 days	consistent with immunisation, unclear due to missing samples	fall <4 days after exposure (with no recent exposure history
Sequelae	Thrombosis, skin necrosis or systemic reaction post-bolus	Progressive or recurrent thrombosis, suspected but unproven thrombosis, erythematous skin reaction	
Alternative cause	None	Possible	Definite

- 0-3 - Low
- 4-5 - Intermediate
- 6-8 - High

This patient fits all the criteria for heparin induced thrombocytopenia

The recommendations for treatment are as follows:

- Therapeutic danaparoid should be used
- The anticoagulant effect of danaparoids can be measured with anti-Xa assay
- Therapeutic dose fondaparinux is an acceptable alternative anticoagulant (although it is unlicensed)
- Therapeutic anticoagulation should be continued for 3 months (in those with thrombosis) and 4 weeks (in those without thrombosis)
- When transitioning from argatroban to warfarin, the INR should be >4 for 2 days prior to discontinuing argatroban
- Warfarin should not be used till platelet count is back in normal range

Question 131 of 138

A 61-year-old man with a history of bipolar affective disorder presents to the Emergency Department with a 2-week history of feeling unwell. This started off as a coarse tremor in the limbs and has now progressed to slurred speech and disorientation. On examination, you find an ataxic gait and myoclonus. His lithium level 2.6. You diagnose lithium toxicity. In addition to bipolar affective disorder, he has a history of rheumatoid arthritis, atrial fibrillation and GORD.

Which of the following recent events is most likely to explain the cause of his toxicity?

A. Recent addition of carbamazepine to augment lithium
B. Recent prescription of naproxen for knee pain
C. Recent increase in caffeine intake
D. Recent addition of amiodarone for atrial fibrillation
E. Recent use of sodium bicarbonate tablets for symptoms of dyspepsia

B. Recent prescription of naproxen for knee pain

The answer is B. NSAIDs reduce excretion of lithium in the kidneys and can, therefore, increase serum lithium levels and give rise to lithium toxicity as seen above. ACE inhibitors, ARBs and thiazide diuretics also reduce the kidney's ability to excrete lithium.

Caffeine and sodium bicarbonate both increase the excretion of lithium from the body and therefore will reduce serum lithium levels

When carbamazepine is added to lithium therapy, it is known to increase the risk of neurotoxicity, but it does this without increasing serum lithium levels. Similarly, use of amiodarone with lithium is known to increase the risk of VT, but it does this without increasing lithium levels

Question 132 of 138

A 25-year-old female was admitted to the emergency department following an overdose of unknown tablets 4 hours ago. Her past medical history includes depression, previous overdoses and gastric ulcer disease. On examination, she was drowsy but easily rousable with a Glasgow Coma Score of 14. She has a heart rate of 112 beats per minute, blood pressure of 106/60mmHg, respiratory rate of 22 and saturations of 96% on air.

Electrocardiogram shows a sinus tachycardia with QRS complexes measuring 160 ms. An arterial blood gas on air revealed the following results:

pH	7.29
pCO2	6.2 kPa
pO2	10.5 kPa
HCO3-	18 mmol/l
BE	-6.6 mmol/l

Which of the following management is the most appropriate?

A. Intravenous magnesium
B. Activated charcoal
C. Gastric lavage
D. Sodium bicarbonate
E. 5% dextrose infusion

D. Sodium bicarbonate

Tricyclic Antidepressant (TCA) overdose is most likely in this case, based on the presentation, mixed acidosis seen on ABG and ECG changes. TCA overdose can present in various ways. Cardiovascular effects include tachycardia, prolong QRS complexes and induce cardiac arrhythmias. Central nervous system effects include altered mental status, coma and seizures.

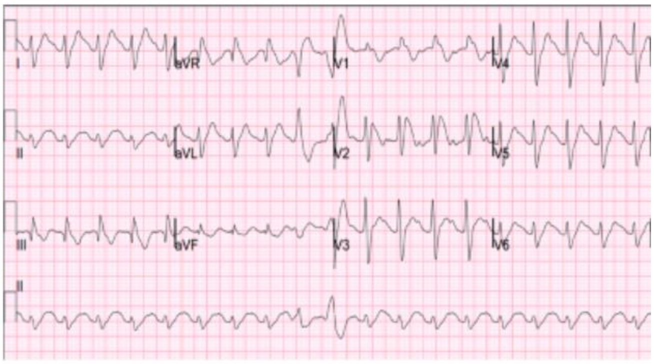
- Intravenous magnesium: no indications in this case of TCA overdose
- Activated charcoal: only appropriate if GCS is not reduced and if patients present within 2 hours of ingestion
- Gastric lavage: may be considered for potentially life-threatening TCA overdoses only when it can be delivered within 1h of ingestion and the airway is protected
- Sodium bicarbonate: is the main treatment for dysrhythmias or hypotension associated with TCA overdoses
- 5% dextrose infusion: has no role in treatment of TCA poisoning or fluid resuscitation

Question 133 of 138

A 40-year-old woman is brought into the emergency department after being found unresponsive by her partner. Her partner reports that she has been well up until today. She has a past medical history of fibromyalgia, migraines and depression for which she has tried several pharmacological agents.

On examination, she has a heart rate of 120 beats/min with a blood pressure of 131/76mmHg. Her chest sounds are clear with a respiratory rate of 28 breaths/min and oxygen saturations of 94% on air. She is drowsy but responsive to voice and her pupils are dilated. She smells strongly of alcohol.

An ECG is taken:



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Given the likely diagnosis, what is the recommended management for this patient?

A. DC cardioversion
B. Fomepizole
C. Haemofiltration
D. Magnesium sulphate
E. Sodium bicarbonate

E. Sodium bicarbonate

Sodium bicarbonate is correct. This patient has a likely diagnosis of a tricyclic antidepressant (TCA) overdose given the tachycardia and dilated pupils in combination with this patient's ECG. This patient's ECG shows tachycardia with widened QRS complexes which is a characteristic feature of a TCA overdose. The past medical history of depression is another suggestion that this patient may be prescribed tricyclic antidepressants. The medical management includes intravenous sodium bicarbonate, particularly if there are any arrhythmias or QRS widening on the ECG. Of note, a QRS interval > 160ms is associated with an increased risk of arrhythmias (e.g. ventricular fibrillation).

DC cardioversion is incorrect. This is used in the cardioversion of tachyarrhythmias with adverse features (e.g. ischaemia, shock, syncope and heart failure). In patients with a TCA overdose, DC cardioversion is unlikely to be successful even in the presence of a ventricular arrhythmia. In this circumstance, intravenous sodium bicarbonate is still recommended along with supporting any additional features (e.g. intubation for low GCS or IVF/phenylephedrine for hypotension).

Fomepizole is incorrect. This is used in the management of ethylene glycol and methanol poisoning. Although this patient smells strongly of alcohol which is suggestive of ethylene glycol or methanol use, the past medical history of depression and the patient's ECG findings are more likely to be secondary to a TCA overdose.

Haemofiltration is incorrect. Although haemofiltration can remove some substances from the circulation (e.g. barbituates, lithium, alcohol, salicylates and theophylline), it has no role in the management of a TCA overdose.

Magnesium sulphate is incorrect. This is used in the management of torsades de pointes, a polymorphic ventricular tachycardia associated with a widened QTc interval. This arrhythmia is more likely to be present if the patient had taken an overdose of a serotonin-selective reuptake inhibitor (SSRI) which is also used to treat depression. However, this arrhythmia is not what is seen here making magnesium sulphate incorrect.

Question 134 of 138

A 58-year-old gentleman is admitted to the acute medical unit with abnormal movements and confusion. He has a past medical history of bipolar disorder and epilepsy. His drug history includes lithium and sodium valproate. On examination you note choreathetoid movements of all four limbs. His Glasgow coma score is 11 (M5, E3, V3).

Blood results are as follows:

Hb	145 g/l	Na^+	138 mmol/l
Platelets	524 * 10^9/l	K^+	3.9 mmol/l
WBC	11.6 * 10^9/l	Urea	10.5 mmol/l
Neuts	9.8 * 10^9/l	Creatinine	125 µmol/l

Lithium level	2.6 mmol/L (therapeutic range 0.4 to 1.0 mmol/L)

What definitive treatment does this patient require?

A. Sodium bicarbonate
B. Gastric lavage
C. Haemodialysis
D. Whole bowel irrigation
E. Reassurance

C. Haemodialysis

Acute on chronic overdose of lithium can lead to serious toxicity even after a modest overdose, as the the extracellular tissues are already saturated with lithium

Acute on chronic overdose of lithium can lead to serious toxicity even after a modest overdose, as the extracellular tissues are already saturated with lithium.

Dialysis should be considered in symptomatic lithium toxicity and:

- A lithium concentration greater than 5.0 mmol/L in patients with acute lithium overdose and who are not prescribed lithium
- A lithium concentration greater than 2.5 mmol/L in patients with chronic poisoning

Haemodialysis should be continued until:

- All new neurological features have resolved, and
- Lithium concentration remains stable at less than 1mmol/L

Gastric lavage, even if possible within one hour of overdose, is likely to be of limited value as slowrelease tablets do not disintegrate in the stomach and most are too large to pass up to a lavage tube.

Whole bowel irrigation can be used, however, there are no clinical trials to confirm its efficacy.

Some studies have shown that urinary alkalisation with sodium bicarbonate may be of use, however the evidence is weak.

Ultimately, this patient has severe neurological features, and a lithium level > 2.5 mmol/L in the context of chronic poisoning, and therefore the definitive management required is haemodialysis. The patient should be fluid resuscitated with crystalloid while discussing the case with a renal physician to arrange dialysis.

Question 135 of 138

A 42-year-old man presents to his GP, wanting to discuss his alcohol intake. For several years, he has been drinking 15-20 units per day. He is now keen to cut down to recommended limits but is enquiring about potential pharmacological options that can be used to aid him.

What can be used to promote abstinence by causing unpleasant symptoms?

A. Acamprosate
B. Diazepam
C. Disulfiram
D. Naltrexone
E. Thiamine

C. Disulfiram

Disulfram is an acetaldehyde dehydrogenase inhibitor. When combined with alcohol, it results in high levels of acetaldehyde which can cause flushing of the skin, nausea, vomiting, arrhythmias, and even death

The correct answer is **disulfiram**. This is an acetaldehyde dehydrogenase inhibitor. By inhibiting this enzyme, consumption of alcohol leads to a build-up of acetaldehyde, which can cause unpleasant symptoms such as flushing of the skin, nausea, vomiting, and arrhythmias. However, a problem with the use of disulfiram is poor compliance, as it does not reduce cravings for alcohol.

Acamprosate is another drug used to promote abstinence. Rather than causing unpleasant symptoms, it reduces the craving for alcohol. It is a weak antagonist of NMDA receptors.

Diazepam is incorrect. Whilst this benzodiazepine is often used in the management of acute withdrawal, it does not cause symptoms like disulfiram.

Naltrexone is incorrect. This is used to treat both opioid and alcohol dependence. Rather than causing unpleasant symptoms, however, it instead reduces cravings and feelings of euphoria associated with substance use disorder.

Thiamine is also incorrect. This is recommended to be given orally to alcoholic patients if their diet may be deficient. It does not cause unpleasant symptoms.

Question 136 of 138

A 27-year-old lady presents to her GP with symptoms of polyuria and dysuria. She is currently 7 weeks into her second pregnancy. Her urine dipstick shows 2+ protein, 2+ blood, 3+ leucocytes and is positive for nitrates. Her records indicate a past reaction to penicillin.

Which of the following would be the most appropriate in her treatment?

A. Nitrofurantoin
B. Trimethoprim
C. Co-amoxiclav
D. Cefalexin
E. Ciclosporin

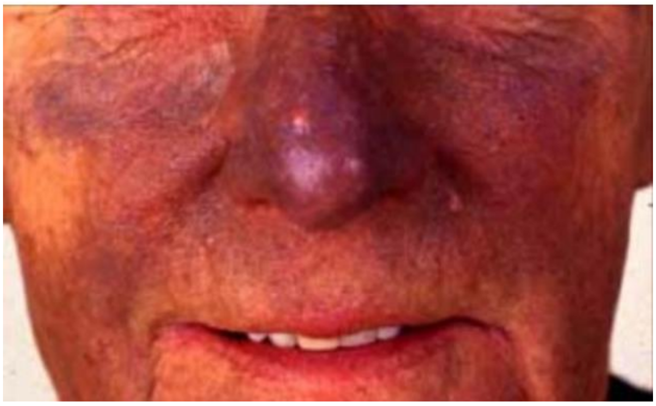
A. Nitrofurantoin

Trimethoprim should be avoided in the first 3 months of pregnancy

Nitrofurantoin has been used extensively in the treatment of UTIs in pregnant ladies in first trimester since the 1950s and has a good safety profile. The anti-folate action of trimethoprim could yield trimethoprim as a second-line agent provided that enough folate cover is offered. NICE guidelines state that 5 mg of folate must be co-administered in case that trimethoprim is prescribed. The antibiotic regime unlike simple UTI lasts for 7 days.

Question 137 of 138

The patient below has noticed a gradual change to his facial skin since starting a new medication a few months ago:



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Which drug has he been taking?

- A. Amiodarone
- B. Phenytoin
- C. Ciclosporin
- D. Digoxin
- E. Sildenafil

A. Amiodarone

The correct answer is **Amiodarone**. This drug, which is used to manage arrhythmias, can cause a bluish-grey discolouration of the skin due to the deposition of lipofuscin pigments. This effect is particularly noticeable on sun-exposed areas such as the face and hands. The condition is usually reversible upon discontinuation of the drug, although it may take several months for the skin colour to return to normal.

The other options are incorrect for the following reasons:

Phenytoin can cause a variety of skin changes including hirsutism, acneiform eruptions, and gingival hyperplasia but it does not typically cause skin discolouration.

Ciclosporin, an immunosuppressant agent used in transplant patients and certain autoimmune conditions, can cause hypertrichosis (excessive hair growth) and gingival hyperplasia. It may also increase susceptibility to infections and malignancies due to its immunosuppressive effects. However, it does not lead to skin discolouration.

Digoxin, a cardiac glycoside used in heart failure and atrial fibrillation, has many potential side effects but they do not include skin discolouration. Instead, digoxin toxicity can manifest with visual disturbances (xanthopsia or yellow vision), gastrointestinal symptoms (nausea, vomiting), neurological symptoms (confusion, delirium), and cardiac arrhythmias.

Finally, **Sildenafil**, commonly known as Viagra and used for erectile dysfunction and pulmonary arterial hypertension, does not typically cause any significant changes in skin colour either. Its most common side effects include headache, flushing, dyspepsia, visual disturbances (blurred vision or cyanopsia - seeing blue-tinted vision), nasal congestion or rhinitis.

Question 138 of 138

A 47-year-old man is admitted to the Medical Assessment Unit with a pneumonia. He has had a cough for the last week which has not settled with amoxicillin from his GP.

His past medical history includes alcoholic liver disease with a hepatic transplant 6 months previously, hypertension and diet-controlled type II diabetes. He has remained abstinent from alcohol for the last 18 months and is a lifelong non-smoker. His medications are ramipril, simvastatin and tacrolimus.

On admission he has a temperature of 38.1 ºC and oxygen saturations of 94% on air. His heart rate is 110 beats per minute and blood pressure is 105/65 mmHg. On auscultation he has left basal crepitations and chest x-ray confirms a left lower lobe pneumonia.

He is started on intravenous fluids, 2 litres oxygen via nasal cannulae, co-amoxiclav and clarithromycin. His statin is withheld.

2 days later, the ward team are asked to review the patient as he has developed limb twitching. During the assessment he has a generalised tonic-clonic seizure which requires lorazepam to terminate it.

Which investigation is most likely to reveal the cause for his seizures?

- A. Blood cultures
- B. CT head
- C. Electrolytes
- D. Lumbar puncture
- E. Tacrolimus level

E. Tacrolimus level

The first issue to consider in this question is whether this gentleman's seizure is most likely to be related to infection or metabolic disturbance.

Given that it is two days following his admission with infection, it is unlikely that he has an undetected meningitis, as he would likely have exhibited other symptoms and become much more unwell in this time.

A CT head would be done prior to a lumbar puncture but would not confirm a diagnosis of meningitis, and this gentleman is unlikely to have a new intracranial pathology given the history. Although febrile seizures are a possibility, they are unusual in adults, and again would be uncommon after 2 days of appropriate antibiotics.

Although blood cultures would of course have been taken, they would not prove that a seizure was the result of infection.

This leaves metabolic causes, including hypoglycaemia, hypomagnesaemia, hyper- and hyponatraemia and an altered tacrolimus level.

Tacrolimus is metabolised by the cytochrome P450 system of enzymes. This gentleman has been started on clarithromycin as an antibiotic, which inhibits this system and is therefore likely to cause an increased blood concentration of tacrolimus. Side effects of high tacrolimus level include hyperkalaemia, hypomagnesaemia, hyperglycaemia, hypertension, twitching and seizures. Although checking electrolytes may give clues as to the cause of this gentleman's seizure, and should definitely be done, a tacrolimus level is necessary to prove the diagnosis.

It is also worth keeping in mind that in this type of patient, the suspicion of withdrawal seizures may be high. However, one would expect to see other signs and symptoms earlier on in the admission.

GERIATRICS
PASSMEDICINE
PART 2
2024

Question 1 of 16

An 85-year-old man with hypertension has been admitted to the hospital following a fall with a subsequent fractured neck femur. Before his admission, he lived with his wife and was independent with all activities of daily living. Four days after his admission, the nursing staff mentioned that he is confused at night and calls for his deceased parents. His vital signs are normal. Upon your assessment, he has bilateral crackles on the auscultation of his chest.

His blood tests show:

Hb	120 g/L	Male: (135-180)Female: (115 - 160)
Platelets	200 * 10 /L 9	(150 - 400)
WBC	7.1 * 10 /L 9	(4.0 - 11.0)
Na +	136 mmol/L	(135 - 145)
K +	4.0 mmol/L	(3.5 - 5.0)
Urea	5.0 mmol/L	(2.0 - 7.0)
Creatinine	100 µmol/L	(55 - 120)

What is the single most likely cause of his symptoms?

A. Alcohol withdrawal
B. Anaemia
C. Constipation
D. Hospital-acquired pneumonia
E. Undiagnosed Alzheimer's

C. Constipation

Constipation can cause delirium in the elderly

The patient presents with acute and fluctuating symptoms of confusion, which are indicative of delirium. This condition can arise from many causes, including infections. **Constipation** should be considered as a potential cause of delirium, particularly after hospitalisation for a fracture due to a fall. Such patients may not be as mobile, have a different diet from their normal, and may be on medications which cause constipation, such as opioids.

It is crucial to exclude **alcohol withdrawal** as a differential diagnosis. Symptoms include confusion, hallucinations and agitation. Symptoms that may help to differentiate alcohol withdrawal from delirium include tremors, sweating, nausea and, if severe, autonomic instability. The most severe symptoms often occur 48-96 hours after a person's last drink. The lack of such features makes alcohol withdrawal unlikely.

This patient is slightly **anaemic**, and this requires a review of his pre-surgery blood tests to determine if it could be due to his recent surgery or other causes. However, this degree of anaemia is less likely to cause delirium.

While **hospital-acquired pneumonia** is frequently implicated in delirium, the current case lacks specific symptoms (including fever and productive cough) or diagnostic blood test results (including deranged white cell count) to substantiate this cause. His chest findings are more likely to be due to atelectasis post-surgery rather than bilateral pneumonia.

Delirium is characterised by acute onset and fluctuating cognitive symptoms. There is no description of cognitive impairment before admission. He is, therefore, more likely to have delirium than **undiagnosed Alzheimer's**. If the patient's confusion is not resolved with treatment, obtaining a comprehensive history and considering further investigations would be necessary.

Question 2 of 16

A 73-year-old man is being treated in hospital for a urinary tract infection. He was initially confused, however, this has since improved. His daughter, however, reports that over the past six months his short-term memory has declined and he is having visual hallucinations.

On physical assessment, his respiratory rate is 16/min, oxygen saturation 95% on air, and the chest is clear. His heart rate is 69/min, blood pressure is 121/80mmHg, and is warm and well perfused. The abdomen is soft and non-tender. His Glasgow coma score is 14 due to confusion for voice and he is afebrile at 36.6ºC. Some mild rigidity is noted on neurological examination but otherwise is normal.

Which medication is most likely to treat his ongoing symptoms?

A. Aspirin
B. Levodopa
C. Memantine
D. Prophylactic antibiotics
E. Rivastigmine

E. Rivastigmine

Acetylcholinesterase inhibitors (e.g. donepezil, rivastigmine) can help alleviate the features of Lewy body dementia

This man has presented with Lewy body dementia. This is evidenced by memory impairment, visual hallucinations, and signs of parkinsonism. Donepezil and rivastigmine are very effective in the treatment of Lewy body dementia therefore rivastigmine is the correct answer.

Aspirin is a treatment that may be used in vascular dementia, however, this is not the diagnosis here. Vascular dementia presents with step-wise determination in memory and is not associated with visual hallucinations or parkinsonism.

Levodopa is a treatment for Parkinson's disease. Although this man is exhibiting some mild parkinsonism on neurological examination, this is likely due to Lewy body dementia given his memory impairment and visual hallucinations. Acetylcholinesterase inhibitors are the most effective treatment in Lewy body dementia and can treat both cognitive and motor symptoms. Therefore, levodopa would not be indicated first-line in this case.

Memantine is used in advanced Alzheimer's dementia with behavioural and psychological symptoms. Alzheimer's is not the diagnosis here as it is not associated with visual hallucinations in a mild or moderate stage and does not cause motor symptoms. Memantine is therefore incorrect as it is not indicated in Lewy body dementia.

As this man has had a continuous six-month history of memory impairment, visual hallucinations, and parkinsonism, recurrent UTIs are unlikely to be the culprit. Therefore, prophylactic antibiotics are not indicated.

Question 3 of 16

A 74-year-old female has been diagnosed with moderate to severe Alzheimer's disease, on a background of a two-year progressive gradual cognitive decline. Her family had tried to cope on their own without seeking medical help, putting it down to old age but now, most likely requires nursing home care. MMSE 7/30. She has a past medical history of previous myocardial infarctions. She has not complained of chest pain recently and her ECG demonstrates no ischaemic changes, a PR interval of 290ms.

What is the most appropriate treatment strategy?

A. Donepezil
B. Memantine
C. Galantamine
D. Rivastigmine
E. Aspirin

B. Memantine

There are two key facts to this patient: firstly, the patients MMSE is suggestive of severe dementia. Secondly, the diagnosis of 1st degree heart block and hence atrioventricular nodal block is a contraindication for cholinesterase inhibitors, which could precipitate complete heart block. In accordance with the latest set of NICE guidelines, donepezil, galantamine and rivastigmine are all appropriate for mild to moderate dementia, defined as MMSE between 10 and 26/30. However, only memantine, an NMDA antagonist, has demonstrated efficacy and is licensed for severe Alzheimer's disease.

Question 4 of 16

A 78-year-old woman with a background of hypertension, depression, and dementia is admitted due to social concerns as her husband is struggling to provide care for her activities of daily living at home. She has her observations taken which include oxygen saturations of 96% on room air, a respiratory rate of 16/min, a heart rate of 47/min, a blood pressure of 101/64mmHg, and a temperature of 36.3ºC.

An ECG is performed which shows normal sinus rhythm with no other abnormalities. A medication review is conducted.

What medication should be stopped?

A. Amlodipine
B. Donepezil
C. Memantine
D. Mirtazapine
E. Sertraline

B. Donepezil

Donepezil is generally avoided (relative contraindication) in patients with bradycardia and is used with caution in other cardiac abnormalities

Donepezil is a cholinesterase inhibitor used to treat Alzheimer's dementia. Due to the procholinergic effects it has, it is strongly associated with bradycardia which is an indication to stop the medication. With a heart rate of 47/min, donepezil should be stopped in this case.

Amlodipine is a dihydropyridine calcium channel blocker that is non-cardio-selective. It is therefore not the medication most likely to be resulting in this woman's bradycardia. As her blood pressure is normal, there is no indication to stop this medication.

Memantine is a N-Methyl-D-Aspartate antagonist used for advanced dementia. Although bradycardia is a reported side effect of memantine, this is relatively rare. As memantine is much less likely to be the cause than donepezil, it would be prudent to hold the donepezil initially.

Mirtazapine can cause prolonged QT interval however the ECG here shows sinus bradycardia and is otherwise normal so it is unlikely to be causing this woman's bradycardia.

Sertraline is another medication that can cause QT prolongation but again as the only positive finding from the ECG is sinus bradycardia, it is not likely to be the cause.

Question 5 of 16

A 82-year-old man is reviewed in memory clinic. He denies any problems himself, but his wife has noticed changes in his memory and therefore asked his GP to see if he has dementia. She has noticed that he at times forgets where he is, forgets that he has asked things in the middle of a conversation, seems to respond to things that are not there and has reported that he 'sees things'. However, at other times he is able to have completely normal conversations. He has a past medical history of prostate cancer which has been managed with hormone therapy, of which she knows he had 'blood tests' to monitor which have been normal. Other than this he has COPD which is well controlled, hypertension and osteoarthritis. He has had no recent changes in medication. He used to smoke, but quit 20 years ago. He used to drink heavily but has completely stopped five years ago.

On examination the patient is alert and orientated. He has a slight tremor in his left hand, and increased tone in muscles.

What is the most likely diagnosis?

A. Lewy-body dementia
B. Alzheimer's disease
C. Brain metastases
D. Vascular dementia
E. Subdural haematoma

A. Lewy-body dementia

A history of progressive decline in cognitive function with fluctuations, visual hallucinations and Parkinsonisms is most likely Lewy-body dementia. The main differential diagnosis is Alzheimer's disease, which has a greater element of progressive cognitive decline, and is not usually associated with hallucinations and fluctuations. Brain metastases are important to exclude but the normal blood tests for prostate cancer - likely PSA - are a good indicator that this is unlikely. Vascular dementia can occur with progressive changes of dementia, especially with a history of high blood pressure and smoking, but this is not what has been described. A subdural haematoma is a strong differential when there is fluctuating consciousness in the context of head trauma and anticoagulant medications.

use at what stages. Please see the link for more details

neuroleptics should be avoided in Lewy body dementia as patients are extremely sensitive and may develop irreversible parkinsonism. Questions may give a history of a patient who has deteriorated following the introduction of an antipsychotic agent

Question 6 of 16

A 72-year-old man presents to the emergency department with confusion. He has a past medical history of hypertension, Parkinson's disease and hypercholesterolemia. His medications include cocareldopa, amlodipine and atorvastatin. He normally lives alone and is independent.

Observations:

- Heart rate 101 beats per minute
- Blood pressure 120/77 mmHg
- Respiratory rate 20/minute
- Oxygen saturations 97% on room air
- Temperature 37.8ºC

On examination, there is suprapubic tenderness. His Glasgow coma scale is 14/15.

He is treated with appropriate antibiotics for a presumed urinary tract infection. Despite clinically and biochemically improving, he remains confused 3-4 days into the admission. Alternative causes of delirium are excluded. He subsequently becomes increasingly agitated and presents a danger to himself and other patients, despite conservative measures designed to re-orient him.

Given the clinical history, what is the most appropriate choice of medication?

A. Clozapine
B. Haloperidol
C. Olanzapine
D. Quetiapine
E. Zopiclone

D. Quetiapine

Quetiapine may be useful in the management of patients with Parkinson's disease who are in an acute confusional state

Quetiapine is correct. This patient presents with delirium secondary to a urinary tract infection on a background of PD. He has a progressively worsening confusional state and conservative measures have failed. He is a danger to himself and others and therefore pharmacological sedation is warranted. The most appropriate choice given his diagnosis of PD is quetiapine, which is less likely to cause worsening of his Parkinsonism than other antipsychotics, which have greater antidopaminergic effects.

Haloperidol is incorrect. This typical antipsychotic is used in the management of delirium. However, it has significant D2 antagonism effects and may therefore worsen the patient's PD.

Olanzapine is incorrect. This antipsychotic can be used in the management of delirium. However, it may worsen PD and therefore should be avoided.

Zopiclone is incorrect. The Z-drug class of medications is not a typical first-line choice for the management of delirium, in Parkinson's disease or otherwise.

Clozapine is incorrect. This atypical antipsychotic can be used in the management of delirium associated with PD. However, quetiapine would be the preferred initial choice due to the potential toxicity of clozapine treatment.

Question 7 of 16

An 85-year-old woman is admitted to the acute medical unit with abdominal pain, swelling and confusion. She has a past medical history of Parkinson's disease, recurrent urinary tract infections and hypertension. She is on amlodipine, co-careldopa and doxazosin. She lives in a care home. Her behaviour is out of character. She is usually pleasant and talkative and there is no history of memory problems.

Her observations are heart rate 88 beats per minute, respiratory rate 18/minute, oxygen saturations 97% on room air, blood pressure 145/88mmHg and temperature 37.1ºC.

Examination reveals impacted faeces in the rectum and some mild suprapubic tenderness. She is inattentive and confused and her cognition fluctuates. She does not tolerate a full neurological exam but you note a unilateral resting tremor and mild bradykinesia.

Urinalysis:

Leucocytes	+++
Nitrites	+
Blood	Negative
Protein	Negative

An ECG is unremarkable. Blood tests:

Hb	136 g/L	Male: (135-180)Female: (115 - 160)
Platelets	189 * 10 /L 9	(150 - 400)
WBC	8.2 * 10 /L 9	(4.0 - 11.0)
Na +	138 mmol/L	(135 - 145)
K +	4.2 mmol/L	(3.5 - 5.0)
Urea	8.9 mmol/L	(2.0 - 7.0)
Creatinine	111 µmol/L	(55 - 120)
CRP	34 mg/L	(< 5)
Calcium	2.24 mmol/L	(2.20 - 2.6)
TSH	1.2 mIU/L	(0.2 - 5.5)

She is treated with antibiotics for a presumed urinary tract infection and laxatives and suppositories for constipation. Within a few days, her inflammatory markers have normalized, she has no clinical features of a urinary tract infection and her bowels are opening twice a day normally.

However, despite environmental modifications, 1:1 nursing and the support of her close family, she remains confused, agitated and inattentive. She is a danger both to herself and to other patients on the ward.

Given the likely diagnosis, what is the most appropriate pharmacological management?

A. Haloperidol
B. Olanzapine
C. Pramipexole
D. Quetiapine
E. Ropinirole

D. Quetiapine

Quetiapine may be useful in the management of patients with Parkinson's disease who are in an acute confusional state

Quetiapine is the correct answer. This patient has delirium that has not responded to conservative measures. Given that she is a danger to herself and others, pharmacological management of her condition is indicated. It is good practice to attempt administration of oral medications in the first instance, where possible. Quetiapine is a suitable choice to aid in the management of delirium in patients with Parkinson's disease as it does not cause worsening of the motor features of Parkinson's disease and can have beneficial effects cognition and psychotic features, if present.

Haloperidol is incorrect. While this is a possible choice for the pharmacological management of delirium, it is a dopamine-antagonist and therefore will worsen the motor features of Parkinson's disease.

Olanzapine is incorrect. This has been shown to cause excessive sedation and worse the motor features of Parkinson's disease.

Ropinirole is incorrect. This is a dopamine agonist used in the management of Parkinson's disease itself. Delirium is a side effect.

Pramipexole is incorrect. This is a dopamine agonist used in the management of Parkinson's disease itself. Delirium is a side effect.

Question 8 of 16

A 75-year-old lady with memory problems attended a specialist memory clinic with her daughter. Her daughter was very concerned and mentioned that her mother had been much more forgetful over the past year. She had left the gas cooker on and occasionally got lost when she walked to the shops. On one occasion she had been found by a neighbour wandering the streets in her dressing gown.

This lady had a family history of Alzheimers disease with both her mother and sister being diagnosed with the condition in their seventies.

On examination she had a Mini Mental State Examination Score of 19/30. Otherwise a full physical examination was unremarkable.

Magnetic resonance of imaging of the brain showed marked atrophy of the medial temporal lobes bilaterally with no evidence of a reversible cause of dementia.

You suspect that this lady has Alzheimers disease and wish to start her on donepezil.

Before starting her on this medication which of the following should you arrange?

A. Electrocardiogram (ECG)
B. Chest X-ray
C. Echocardiogram
D. Lung function tests
E. Ophthalmology review

A. Electrocardiogram (ECG)

Donepezil is generally avoided (relative contraindication) in patients with bradycardia and is used with caution in other cardiac abnormalities

Cholinesterase inhibitors such as donepezil, galantamine and rivastigimine are licensed for use in patients with mild to moderate Alzheimers disease. The National Institute for Health and Care Excellence (NICE) currently recommends treatment for all patients with moderate Alzheimers disease with a Mini Mental State Examination (MMSE) score of 10 20.

The British National Formulary lists sick sinus syndrome and supraventricular conduction problems (such as atrial flutter and atrial fibrillation) as relative contraindications in the prescribing of cholinesterase inhibitors. Although it is not specifically stated in NICE guidance there is a large evidence base to support the practice of performing a routine ECG prior to initiating treatment. There is insufficient evidence to support the use of routine echocardiograms prior to the initiation of cholinesterase inhibitor

Question 9 of 16

A 64 year old man presents with a 6 month history of abnormal behaviours which have been noticed by his wife. He has described seeing vivid visual hallucinations of clowns in his living room which sometimes talk to him and appear very real. He believes that he is the head of a circus and is about to go on a world tour although this is not true.

At times he is lucid and is fully independent but at other times he is disorientated in time and place and is unable to perform simple tasks such as preparing food and going to the shops. His wife thinks that his mood is also lower since the onset of symptoms. He presented in A+E today because of having a second fall in two weeks.

There is no history of infective symptoms. He went to see his GP two days ago who thought that he may have a UTI and prescribed trimethoprim.

He has a history of stroke 10 years ago and hypertension and takes warfarin, amlodipine and enalapril.

Physical examination is unremarkable except for slightly increased tone on the left side compared to the right.

Bloods:

Hb	14.9 g/dl
Platelets	387 * 10 /l9
WBC	12.8 * 10 /l9
Na +	142 mmol/l
K +	4.6 mmol/l
Urea	6.4 mmol/l
Creatinine	84 µmol/l
Bilirubin	6 µmol/l
ALP	64 u/l
ALT	15 u/l
Calcium	2.35 mmol/l

Albumin	41 g/l

MSU (from GP from 2 days ago): Heavy growth of E.coli Sensitive to trimethoprim, nitrofurantoin, amoxicillin and co-amoxiclav

CT Brain: some generalised atrophy and periventricular white matter changes normal for age. Changes in keeping with an old left sided lacunar infarct Mini Mental State Examination 17/30

Which medications would most appropriately treat the underlying diagnosis?

A. Olanzapine
B. Rivastigmine
C. Co-amoxiclav
D. Sinemet
E. Aspirin 300mg

B. Rivastigmine

The answer is Rivastigmine. The diagnosis here is Lewy Body dementia. Lewy body dementias core clinical features are fluctuating g cognition, visual hallucinations (present in 2/3rds of cases) and parkinsonism. Two out of three are needed for diagnosis. The visual hallucinations are often very vivid. This patient definitely has two out of the three. He also may have parkinsonism as he has bilaterally increased tone that is not in keeping with his old lacunar infarct.

He also has a few supportive features of Lewy Body Dementia hallucinations in other modalities, delusions, depression and repeated falls.

Currently, evidence best supports cholinesterase inhibitors in the treating of Lewy Body Dementia. It must be remembered that these patients have high sensitivity to neuroleptics so Olanzapine should not be used here. Schizophrenia is a less likely diagnosis as visual hallucinations are rare in late onset schizophrenia and late onset schizophrenia itself is rare. Also, fluctuating mental state is not usually seen in schizophrenia.

Whilst this patient has a UTI, it is sensitive to trimethoprim and therefore is already being appropriately treated and therefore further antibiotics are not required. As the symptoms have been present for 6 months, UTI is unlikely to be the underlying diagnosis.

Whilst the patient does have risk factors for stroke and focal neurology and a TIA is possible, it does not explain his other symptoms and therefore aspirin would not therefore represent treatment for the underlying diagnosis.

The patient does show features of parkinsonism but a diagnosis of Lewy Body is more suggested by the cognitive and psychiatric symptoms and therefore Sinemet would be not be considered before a cholinesterase inhibitor.

Question 10 of 16

A 67-year-old man is admitted to the hospital with community-acquired pneumonia. In the acute medical unit, he is becoming progressively more confused and has started having visual hallucinations of children running around the ward. These symptoms are worse at night. Otherwise, there is no evidence of any other neurological or psychiatric symptoms. There is no known past medical history of dementia.

What test would be most appropriate in to confirm the cause of this man's confusion?

A. Cambridge cognition examination
B. Confusion assessment method
C. Mental state examination
D. Mini-mental state examination
E. Six-item cognitive impairment test

B. Confusion assessment method

The Confusion Assessment Method (CAM) is the best way to differentiate between delirium and dementia

Current NICE guidance advises the use of the **Confusion Assessment Method** as the first-line diagnostic test for delirium rather than any of the other cognitive tests. This patient has symptoms consistent with delirium; acute onset, fluctuating course, and visual hallucinations with a preceding insult. It is therefore the most appropriate test in this acute setting. The diagnostic criteria for this test include an acute onset with a fluctuating course with inattention plus either disorganised thinking or an altered level of consciousness.

The **Cambridge cognition examination** is designed to diagnose and assess the severity of dementia. It is not specific for delirium which is the most likely diagnosis here and therefore is not the most appropriate test.

A **mental state examination** is a comprehensive way to assess mental disorders. Although this does include cognitive testing, it is not as useful or concise as the confusion assessment method to quickly differentiate between delirium and dementia. Therefore, as the psychiatric symptoms are limited to cognition, it would be more appropriate in this acute setting to use the confusion assessment method.

Although the **mini-mental state examination** can be used to assess cognition, it is not designed to distinguish between dementia and delirium. Although repeat tests may be useful in demonstrating deterioration or fluctuation in cognition, the confusion assessment method would be more appropriate in this instance of acute confusion to differentiate between delirium and dementia.

The **six-item cognitive impairment test** is again a test used to quantify the extent of dementia, rather than delirium. Therefore it would not be the most appropriate test here to distinguish between the two.

Question 11 of 16

A 65-year-old man is reviewed in the memory clinic. He has been diagnosed with Lewy-body dementia as well as Parkinson's disease. Whilst the Parkinson's disease is being managed with levodopa, he and his wife have found the dementia to be worsening. On objective testing, he is found to have mild dementia.

Which one of the following medications should be offered to alleviate his cognitive impairment?

A. Donepezil
B. Clopidogrel + atorvastatin
C. Haloperidol
D. Increase levodopa dose
E. No medical treatment is available

A. Donepezil

Acetylcholinesterase inhibitors (e.g. donepezil, rivastigmine) can help alleviate the features of Lewy body dementia

The correct answer is **Donepezil**. Donepezil is a cholinesterase inhibitor which works by increasing the levels of acetylcholine in the brain, improving cognitive function. According to the UK guidelines (NICE), donepezil can be prescribed for patients with mild to moderate dementia due to Alzheimer's disease or Lewy body dementia. In this case, as the patient has mild Lewy body dementia, donepezil would be an appropriate choice to alleviate his cognitive impairment.

Clopidogrel + atorvastatin are not indicated for treating cognitive impairment in dementia. Clopidogrel is an antiplatelet agent used for preventing thrombotic events, while atorvastatin is a statin used for lowering cholesterol levels and reducing cardiovascular risk. Although there have been studies suggesting that statins may have a protective effect against dementia, they are not recommended as a treatment for cognitive decline in current guidelines.

Haloperidol is an antipsychotic medication that might be used in managing behavioural and psychological symptoms of dementia (BPSD) such as agitation or hallucinations. However, it does not improve cognitive function and should be used with caution in patients with Lewy body dementia or Parkinson's disease due to the risk of worsening extrapyramidal symptoms.

Increase levodopa dose would not be an appropriate choice since levodopa mainly targets motor symptoms of Parkinson's disease rather than cognitive impairment associated with Lewy body dementia. Additionally, increasing the dose may lead to side effects such as dyskinesia or hallucinations without providing significant benefits for cognition.

No medical treatment is available is incorrect because there are medications like donepezil that can help alleviate cognitive impairment in patients with mild to moderate Lewy body dementia. Although there is no cure for dementia, medications like donepezil can help improve the quality of life for patients and their caregivers by managing symptoms.

Question 12 of 16

You are in General Practice when a 75-year-old lady is brought in by her concerned daughter. She reports that she has been acting strangely recently and calling her up in the middle of the night thinking that strangers are in the house. She also seems disorientated with time and frequently gets ready for bed in the middle of the day. Her daughter reports that some days she can seem 'back to her old self' and appears completely lucid.

On direct questioning the patient is orientated to time and space but is rambling and tangential, often speaking off topic. She admits to visual hallucinations including a cat that has been following her round very several months. She is otherwise well but says that she has become 'slowed down' in general over the last year.

Current medication includes rivaroxaban for atrial fibrillation and amitriptyline for fibromyalgia.

On review of the patient's medical records, you note she has been brought in several times over the past six months with similar complaints and each time has been given a course of antibiotics for a suspected UTI.

What is the most underlying cause of the patient's presentation?

A. Psychotic episode
B. Recurrent urinary tract infections
C. Lewy body dementia
D. Medication induced
E. Subdural haemorrhage

C. Lewy body dementia

Lewy body dementia typically presents with fluctuating cognition in contrast to other forms of dementia

The three cardinal features of Lewy body dementia are visual hallucinations, fluctuating confusion, and features of Parkinsonism. Presentations can be mistaken for delirium and are often treated for UTIs. A detailed history to uncover the longstanding and progressive nature of the disease is essential. Patients often report the sensation that others are present in their homes. Other features that could suggest Lewy body include a history of REM sleep behaviour disorder or 'confusional arousals' on waking from a nap. Careful examination should be taken to look for bradykinesia, rigidity, or rest tremor - this was suggested by the general 'slowing down' in this patient.

Medications such as amitriptyline have a high anticholinergic burden index and can contribute to cognitive impairment but wouldn't typically cause visual hallucinations. Equally subdural haemorrhage can cause a fluctuating confusion but would be more likely to have focal neurology rather than hallucinations. Nevertheless, brain imaging in this patient is essential.

Question 13 of 16

An 80-year-old man was brought to hospital after a collapse. A carer at his nursing home reports he became pale and slumped unresponsive in a chair but regained consciousness after a few minutes. His past medical history includes hypertension, hypothyroidism, mild dementia and a previous seizure 15 years ago.

Paramedics at the scene reported a heart rate of 34/min which has now resolved. Examination reveals normal heart sounds, capillary refill of 3 seconds and a regular pulse of 60/min.

Which of his medications may be responsible for the collapse?

A. Bendroflumethiazide
B. Triamterene
C. Levothyroxine
D. Sodium valproate
E. Donepezil

E. Donepezil

Donepezil is a cholinesterase inhibitor used in the treatment of mild to moderate dementia. Its vagotonic effect can lead to bradycardia and heart block. This can occur particularly when coadministered with other anticholinergics and drugs that inhibit the CYP450 enzyme 3A4 and 2D6 as plasma levels of donepezil will be increased.

Question 14 of 16

An 83-year-old woman attends the memory clinic along with her son with a 7-month history of memory loss. She reports feeling well today, but her son explains the previous week she was confused and disoriented. Her son also tells you that he fears his mother has been hallucinating since she has told him that she sees her husband sitting at the dinner table with her. However, he died 5 years ago. She has a background of osteoarthritis and borderline diabetes but has otherwise been well with no recent illnesses.

On examination, her heart rate is 86bpm and regular. Her sitting and standing blood pressures are 152/95mmHg and 138/86mmHg respectively. On auscultation, she has a clear chest with normal heart sounds. Her abdomen is soft with no palpable masses. There is a slight tremor in her left hand with increased rigidity. She walks well with a walking stick but there is a shuffling gait. Her MMSE is 20/30.

Assuming the likely diagnosis, what is the most appropriate treatment to initiate?

A. Haloperidol
B. Levodopa
C. Memantine
D. Rivastigmine
E. Ropinirole

D. Rivastigmine

Acetylcholinesterase inhibitors (e.g. donepezil, rivastigmine) can help alleviate the features of Lewy body dementia

This patient has Lewy body dementia (LBD). An MMSE of 20 suggests a diagnosis of mild dementia. Symptoms of LBD can vary from person to person and typically include fluctuating confusion, disturbances in sleep, mood changes and hallucinations. There is an overlap with Parkinson's disease, and motor symptoms may manifest as the disease progresses. This scenario describes a woman with increased rigidity, tremor and a shuffling gait which suggests the onset of Parkinsonian features. Unlike Parkinson's disease dementia, LBD is the development of cognitive impairment prior to the onset of motor symptoms. Whereas, the definition of Parkinson's disease dementia requires the duration of motor symptoms to be present for at least 1 year before the onset of cognitive impairment. NICE guidelines recommend the use of acetylcholinesterase inhibitors in the management of Alzheimer's disease and LBD, making rivastigmine the correct choice.

Haloperidol is an antipsychotic medication that works through the inhibition of dopaminergic receptors. It is sometimes given to treat acute agitation. However, it can worsen co-existing parkinsonian symptoms and should not be given in patients with Parkinson's disease or LBD.

Levodopa is the first-line treatment for Parkinson's disease. Whilst this patient does have symptoms of Parkinson's disease, she also has fluctuating confusion and cognitive impairment. From the clinical history, it seems that the onset of memory loss preceded motor symptoms, making LBD more likely.

Memantine is an NMDA receptor antagonist, blocking glutamate transmission in the brain. Memantine monotherapy is recommended as an option for managing severe LBD and Alzheimer's disease or moderate disease in patients who cannot tolerate AChE inhibition. An MMSE score of 12 or less suggests severe dementia. This patient has a much higher MMSE score and no contraindications to AChE therapy are given in the scenario, making rivastigmine the preferred initial treatment option.

Ropinirole is another treatment option for Parkinson's disease. It is not used in the treatment of cognitive impairment in LBD.

Question 15 of 16

A 58-year-old male is brought into your outpatient clinic by his wife. The patient does not understand why he needs to see a doctor and just wants to get back to work. However, she reports a rather vague history of increasing withdrawal from social interactions and odd repetition of 'catch phrases' over the past 9 months. In addition, she feels his behaviour has changed and is very inappropriate when meeting up with friends, once urinating at the table while having dinner. Last week, he gave her a grave headstone for her birthday, saying 'it is nice to be well-prepared!' While she was understandably upset, he was mystified as to why his well thought out gift might have caused distress. On examination, he continues to repeat the phrase 'Whats up doc?!' at a regular interval, disturbing your history taking. You finally complete a mini-mental test examination, scoring 27/30.

What is the most likely diagnosis?

A. No medical diagnosis
B. Borderline personality disorder
C. Bipolar disorder
D. Schizophrenia
E. Frontotemporal dementia

E. Frontotemporal dementia

The history described of behavioural change, lack of insight, mental rigidity and stereotyped behaviours. The prominent behavioural features with a lack of amnestic features are typical for the behavioural variant of frontotemporal dementia. These patients do not typically retain insight and cognitive functions may be normal in early disease. The main differential diagnoses consist of psychiatric disorders. The patient presents with first symptoms at late-fifties and while not impossible, it is rare for psychiatric disorders to be diagnosed so late in life. It is important to rule out other organic causes of behavioural changes with MRI neuroimaging, such as frontal lobe space occupying lesions.

Question 16 of 16

A 72-year-old man attends the memory clinic with his wife. He was referred by his GP after his wife noticed his short term memory had deteriorated, he was getting lost around the village he had lived in for the last 40 years and his mood had changed.

His past medical history includes hypothyroidism, hypertension and arthritis. He has recently been discharged from cardiology outpatients where they decided a pacemaker was not indicated as he is asymptomatic of the abnormalities found on his ECG. He takes levothyroxine, ramipril, amlodipine and paracetamol.

There was no abnormality found on physical examination. The patient scored 20/30 on MMSE (mild impairment 21-30, moderate impairment 11-10 and severe impairment 0-10). The results of his blood tests are listed below:

Hb	137 g/L	(135-180)

Platelets	279 * 10 /L 9	(150 - 400)
WBC	7.5 * 10 /L 9	(4.0 - 11.0)
Na +	138 mmol/L	(135 - 145)
K +	4.3 mmol/L	(3.5 - 5.0)
Urea	6.8 mmol/L	(2.0 - 7.0)
Creatinine	130 µmol/L	(55 - 120)
Calcium	2.4 mmol/L	(2.1-2.6)
Thyroid-stimulating hormone (TSH)	4.9 mU/L	(0.5-5.5)
Free thyroxine (T4)	13.5 pmol/L	(9.0 - 18)
Haematinics	normal	
HIV serology	negative	
Syphilis serology	negative	

His ECG showed sinus rhythm at 41bpm with a PR interval of 140ms.

An MRI head showed global cerebral atrophy, particularly in the temporal lobe (disproportionate hippocampal atrophy).

The patient and his wife are keen to discuss what treatments and support are available to them.

What would be the most suitable treatment for this patient?

A. Donepezil
B. Atorvastatin
C. Memantine
D. Galantamine
E. Sertraline

C. Memantine

Donepezil is generally avoided (relative contraindication) in patients with bradycardia and is used with caution in other cardiac abnormalities

Alzheimer's disease is the most common form of dementia. The short term memory loss and MRI findings in this patient are in keeping with this diagnosis.

Donepezil is advocated as the first-line treatment for Alzheimer's disease by NICE. However, this is contraindicated in this patient given his bradycardia on ECG and so is incorrect.

Atorvastatin can be used in vascular dementia as cardiovascular risk factor modification.

Galantamine is another acetylcholine esterase inhibitor like donepezil. Like donepezil, it can cause bradycardia but also atrioventricular block, and so is contraindicated in this patient who has resting bradycardia and 1st-degree heart block.

Sertraline is an antidepressant and is not recommended by NICE for the management of mood symptoms in Alzheimer's disease.

Memantine is the correct answer. This is used as second-line treatment (where 1st line treatment has failed or is contraindicated) or add on therapy. Given first-line treatment is contraindicated in this patient, memantine is the most suitable management option.